Introduction

The history of HIV/AIDS is a story of human suffering but also one of hope, human perseverance and scientific ingenuity. Many battles have been won, such as the identification of the virus, developing effective treatment and challenging societal prejudice. Another battle, which still rages in many parts of the world, is the reduction of stigma attached to what was thought of as the ‘gay plague’ by many for decades. HIV is indiscriminate in its ability to infect those of all ages, races, sexualities and nationalities. Unfortunately, as with many other viruses, it starkly reveals the usual fault lines of socio-economic inequality with a disproportionate burden falling on the most marginalised within society. Notable landmarks, such as the discovery of the virus itself and the development of effective ART, have been both life-saving and life-changing for millions who thought the diagnosis bought with it an inevitable death sentence.
The Origins of HIV

Much of our human DNA consists of viruses we have adopted, evolved and integrated into our genome since the first *Homo sapiens* emerged from Central Africa over 200,000 years ago. Indeed, it has been estimated that up to 8% of our genome consists of ancient retroviruses integrated into ours (Belshaw et al., 2004). It appears that we have long been exposed to numerous microbial assaults over the course of our evolution—with varying degrees of successful adaptation. It is quite possible that humans will eventually adapt to HIV, but this specific virus is far too young, ever-changing, virulent and deadly for this to be an acceptable method of controlling it.

Our immunological mechanisms safeguard us against many pathogens, especially those from animal species. These zoonoses are usually unable to take hold in human populations, but occasionally we see the infection cross species—the devastating COVID-19 pandemic is an example of this. Unfortunately, rapidly replicating and evolving viruses, such as HIV, can work quickly to exploit and overcome our protective mechanisms leading to a translocation of the infection from animals to humans, who have little or no innate immunity to fight off these infections. Thus, despite our relatively complex and sophisticated immune system, these pathogens can disable our immunological safeguards leading to the establishment of infection in humans.

An example of this is the circumnavigation of these protective mechanisms; we can use a protein called tetherin (also called CD317) as a demonstration of the many safeguards made ineffective by HIV. Tetherin is a transmembrane protein which ‘tethers’ newly formed virus to each other and to the cell membrane. HIV has a potent anti-tetherin protein which inactivates this protective mechanism and facilitates efficient viral budding releasing new virus to infect other cells. This circumnavigation of our sophisticated immune system may be one of the many reasons that cross-species infection occurred (Sauter et al., 2009). Research continues to investigate how else HIV is able to evade our sophisticated immune system and establish infections, which will hopefully provide more detailed insight into the functionality of the virus and possible ways to defeat it.
SIV

The origin of the Simian Immunodeficiency Virus (SIV), the precursor of HIV, dates back as far as 30,000 years. SIV is a collection of lentiviruses with a single phylogenetic lineage directly related to HIV. Lentiviruses are defined as viruses that have long, slow periods of incubation (‘lente’ is Latin for slow) and are known to affect a number of mammals including horses, sheep and cats as well as humans. The fact that they incubate for such long periods without symptoms allows for their propagation and spread within populations. It is believed that some lentiviruses became embedded in the mammal’s DNA many millions of years ago—in other words, the virus is incorporated into the host’s genome and can then be transmitted to its offspring.

Many lentiviruses affect a number of monkeys, gorillas and chimpanzees living in East and Central Africa, with SIV having been discovered in the late 1980s and early 1990s in chimpanzees (Huet, Cheynier, Meyerhans, Roelants, & Wain-Hobson, 1990) and sooty mangabeys (Hirsch et al., 1989). Most primate species are infected with a single type of SIV. However, it is now accepted that strains of SIV found in chimpanzees are a cross-species recombinant virus from different species of monkeys infected with SIV over many thousands of years, resulting in a virus similar to HIV. These recombinant viruses within primates have led to two distinct HIV populations; the more common HIV-1 and less virulent HIV-2. HIV-1 accounts for 90% of all infections with HIV-2 being found mainly in West Africa. The origins of both can be traced to chimpanzees (HIV-1) and sooty mangabey monkeys (HIV-2) with distinct viral sequences for each type.

SIV from red-capped mangabey and the greater spot-nosed monkey have been shown to be the origin of SIV in chimpanzees (Bonn, 2003), the precursor of HIV-1 and the most likely source of cross-species infection. In their natural environment, chimps are known to hunt and eat other species of monkeys, which may explain how SIV could have jumped into different species of primates. This SIV is now thought to have been transmitted from chimpanzees to humans from either the butchering or eating of chimp meat.
Fieldwork in Gombe National Park, Tanzania, studied different groups of chimpanzees and revealed that linked SIV infections were found to be passed on through sexual intercourse. Analysis of these linked infections demonstrated similar rates of transmission to heterosexual humans (transmission per coital act: 0.0008–0.0015 in chimps versus 0.0011 in heterosexual humans) (Rudicell et al., 2010). Other routes of infection include vertical transmission (i.e. mother to child) and during aggressive exchanges/blood to blood contact.

It was widely believed that SIV was non-pathogenic in many mammals and researchers attempted to understand why these species were not immunocompromised by the virus, hoping that it would lead to the elusive cure for HIV. However, a study in *Nature* (Keele et al., 2009) revealed AIDS-like illnesses in wild chimpanzees with those infected with SIV being 10–16 times more likely to die compared to non-infected chimps. Post-mortem results showed parasitic infections and significant depletions in the CD4 cells mirroring the pathology found in human AIDS.

**The First Human Infections**

HIV has four separate lineages divided into groups, namely, M, N, O and P. Group M is the main virus that has caused the global spread of HIV, while the others constitute only a fraction of total infections. Phylogenetic analysis has shown that group M originated in Kinshasa (or Leopoldville, as it was then called), now in the Democratic Republic of the Congo, in the early twentieth century (Castro-Nallar, Crandall, & Pérez-Losada, 2012).

The first humans to be infected with HIV are thought to have been part of the Bantu tribe, which comprises 30% of Africa’s population. There had been an unprecedented rise in lymphomas and Kaposi’s sarcoma in Africa in this specific population in the first half of the twentieth century without any clear indications as to the cause (Oettle, 1962). Given that the Bantu tribes were in close contact with wild chimpanzees, cross-species HIV infection may be the reason for this sudden unexpected
rise in these possible AIDS-defining illnesses. As the forces of colonialism and globalisation took hold, HIV was able to thrive and proliferate along transport routes and then be transported to Haiti, thought to be the main bridge of infection into the US. Many Haitians worked in Congo during this period as the Belgian colony was undergoing significant economic growth through the discovery and export of copper. The earliest retrospectively confirmed HIV case in humans was in 1959 in Zaire (now the Democratic Republic of the Congo), where SIV is believed to have crossed the species barrier (Nahmias et al., 1986). Using Western Blot analysis, scientists tested a blood sample taken from a Bantu male in Zaire in 1959 and found evidence of a HIV-like virus.

The first suspected case in the US was that of Robert Rayford in 1968, a 15-year-old African American who was admitted with disseminated chlamydia infection and subsequently died of pneumonia (Garry et al., 1988). Subsequent analysis of tissue samples revealed an earlier form of HIV—different from the form which predominates today. Rayford died three days after his 16th birthday with the source of his infection never having been identified. It is plausible that only a few cases of HIV were translocated into the US this early, with those affected never having been formally identified as having AIDS.

Throughout the 1970s, there were many cases of patients presenting with unusual symptoms suggesting immunosuppression with unusual opportunistic infections. Grethe Rask, a Danish surgeon who had been posted in Congo, returned to Denmark with a serious case of Pneumocystis carinii pneumonia (PCP), a fungal pneumonia, and subsequently died in 1977. The discovery of pneumocysts on her autopsy puzzled her colleagues and only later was it hypothesised that she may have contracted HIV whilst working near Kinshasa—an early reminder of how easy it is for HIV to cross international boundaries. In fact, it was impossible, at the time, to detect which part of the complex immune system was being destroyed by this mysterious condition as CD4 cells (a T-cell lymphocyte and the cell targeted by HIV) had yet to be discovered as part of the immune system.
Initial Medical Responses to HIV/AIDS

T-cell lymphocytes, the cellular host supplying the machinery for HIV replication, had only been discovered in the late 1970s, and when the AIDS crisis began, research into this new area of immunology was still in its infancy. The first person to discover the effect of HIV on CD4 cells (a subsection of T-cells) was Dr James Goedert of the National Cancer Institute in the US. He had developed a new diagnostic test called Fluorescent Activated Cell Sorting, a technique using fluorescent dyes to ‘label’ immune cells allowing CD4 counts to be calculated. A study from 1985 demonstrated severely depleted CD4 cells for the first time in gay men who had shown signs of immunosuppression (Goedert et al., 1985). In the early 1980s, this formed the only reliable diagnostic and reproducible test for HIV infection until the antibody test became available.

It was in the early 1980s that clinicians began to regard the mysterious illness as a distinct syndrome— for a few years they had been observing inexplicable cases of severe immunosuppression in otherwise young healthy men with homosexuality being the only common factor. Physicians in San Francisco saw increasing numbers of gay men attending their clinics with the pathognomonic purple skin lesions of Kaposi’s sarcoma. The presence of this new aggressive form in gay men remained a mystery as it usually presented as an indolent skin cancer seen in elderly Jewish men. Kaposi’s sarcoma lesions became one of the earliest herald lesions and visible stigmata of AIDS patients and became synonymous with inevitable death. Many US-based physicians used to treating gay men observed the lesions followed by a swift decline into immunosuppression and death. It was these physicians who alerted the medical community to a new pathogen circulating among gay men well before HIV was identified.

Kaposi’s sarcoma was often a precursor of other more serious and often fatal opportunistic infections. *Pneumocystis carinii* pneumonia (PCP) (or *Pneumocystis jirovecii* pneumonia [PJP] as it is now known) is a ubiquitous fungus that was generally seen only in malnourished children in Eastern Europe during the Second World War and in severely
immunosuppressed patients. Prior to AIDS, there were fewer than 100 cases of reported PCP in the US. The accepted treatment was, and remains, pentamidine, a potent antimicrobial.

Given that this was such an unusual treatment (and indeed because PCP itself was so rare), a technician at the Food and Drug Administration (FDA) was charged with the task of dispensing pentamidine and recording patient details when requests for the drug were received. It was the young FDA technician Sandra Ford who took the decision to alert her senior colleagues at the FDA after receiving several pentamidine requests for otherwise healthy young men (and multiple courses in some patients) who had no identifiable risk factors for immunosuppression. This was the first time that the authorities were made aware of this new disease observable mainly in young gay men. Sandra Ford was the first government official to take notice of this new syndrome and, thus, the catalyst for the start of the institutional response to the AIDS crisis.

Meanwhile, Michael Gottlieb, an Assistant Professor of Immunology at the University of California in Los Angeles Medical Centre, and Wayne Shandera, a public health doctor in Los Angeles, had encountered at least five cases of PCP in gay men and decided to submit a brief article describing their observations to the US Centre for Disease Control and Prevention (CDC) Morbidity and Mortality Weekly Report. This report, an alert system for medical professionals to share emerging information about new infections, was published on 5 June 1981 and is the first written scientific account of HIV/AIDS. The editors also erroneously attributed the cluster of this unusual disease to high levels of cytomegalovirus (CMV), a sexually transmissible infection that is highly prevalent in gay men. The working hypothesis at the time was that CMV had somehow overwhelmed the immune system, leading to profound immunosuppression.

This new syndrome attracted the attention of Dr Jim Curran, a public health physician who proceeded to establish the Kaposi’s sarcoma and Opportunistic Infection Task Force to ascertain the aetiology of the immunosuppression observed in growing numbers of gay men.

At the time, there were three key hypotheses. It was thought that the syndrome may be attributable to (1) an infectious agent, which was as yet unidentified; (2) amyl nitrate (or ‘poppers’), which is an inhaled form of
nitrates used by gay men during sex as a muscle relaxant; or (3) CMV, which could be overwhelming the immune system and, thus, causing the immunosuppression. All three hypotheses were treated as equally plausible given the lack of reliable empirical data. The second and third hypotheses were gradually rejected as more evidence came to light. More specifically, many samples of amyl nitrates were analysed and found to have no effect on immune status. For the CMV hypothesis, it was known that it is a virus that infects between 60 and 70% of adults in developed countries and close to 100% of those in developing countries (Cannon, Schmid, & Hyde, 2010), but the cases appeared, at the time, to be limited only to gay men. When scientists first began to examine the blood of those who were immunosuppressed, they found extremely high levels of CMV. In fact, CMV lies dormant in the body after infection and only when the body is immunosuppressed does it ‘reactivate’, leading to the high levels seen in the gay men exhibiting immunosuppression.

Much of the initial understanding of AIDS was from the work of Selma Dritz, the Public Health Assistant Director for San Francisco, who had been interviewing gay men affected by this new syndrome. She had previously completed work on ‘gay bowel syndrome’ (a common presentation in the 1980s and usually due to amoebiasis and giardia from faeco-oral contact) and was familiar with the sexual norms and behaviours of gay men in San Francisco. She attempted to trace who had developed Kaposi’s sarcoma and ascertained that many of the men affected were in fact connected—by sexual contact. This evidence constituted one of the missing pieces of the puzzle and was to be key to shaping the response to the AIDS crisis (Loewenberg, 2008).

Another significant development in the early epidemic was made by Arye Rubinstein, a paediatrician based in New York. He was alarmed when he began to observe cases of unexplained immunosuppression in newborn infants as early as December 1981. Many of these children were born to mothers from the Bronx, a deprived area of New York, who were also injecting drug users and were themselves showing the hallmarks of immunosuppression. He speculated that this might be linked to the syndrome observed in gay men, given the similarities. Transmission from mother to child was indicative of a bloodborne infectious agent as it
appeared to follow a very similar pattern to that of the recent outbreak of hepatitis B.

In times of uncertainty, scapegoats are often used as a way of helping people direct anger or frustration at otherwise unfathomable circumstances. AIDS was no different. In the early days of the research, many of those affected had had sexual contact with one person, Gaeten Dugas, a Canadian airline steward. As he was part of some sexual networks, many believed he was personally responsible for the propagation of HIV across the US—the cities he flew to were heavily affected by AIDS. Dugas continued to be sexually active after his diagnosis with Kaposi’s sarcoma, which shocked public health officials at the time. There was even talk of legally curtailing his sexual activities to reduce the spread of AIDS, setting a very dark precedent for many in the gay community. With hindsight, it is clear the ‘patient zero’ theory was inaccurate, and it has since been proven that HIV arrived in the US long before Gaetan Dugas was identified (Worobey et al., 2016).

A much more important catalyst for AIDS were the gay bathhouses in which many gay men at the time reported having condomless sex with multiple partners in a single visit. This undoubtedly led to an increase in the rate of new infections with HIV in the US and elsewhere. When it was understood that AIDS was associated with sexual behaviour, there were calls from public health officials and some within the gay community for the bathhouses to be closed. However, bathhouses represented a symbol of recently hard-won civil rights for gay men, and many refused to renounce the bathhouses despite the dangers that they clearly posed. Ultimately, the bathhouses were allowed to remain open provided they displayed posters about the risks associated with condomless sex. Attendance did fall in this period although the saunas and bathhouses continued to provide the perfect amplification apparatus for new transmissions in the gay community.

The search for the cause of AIDS was paramount not only to controlling its spread but also to generating possible treatment options for those affected. Given that T-cells were the potential target with a long period of infection before immunosuppression, a retrovirus was suspected. A known retrovirus, another human T-lymphocyte virus (HTLV) had already been identified and its clinical course seemed to fit this picture.
Two laboratories, one in the Pasteur Institute in Paris, France, and the other a government laboratory in Bethesda, US, worked with tissue and blood samples of known AIDS patients to ascertain the cause of what was initially called HTLV-III. Retroviral research at the time was painstakingly laborious and error-prone given the limited tools available at the time. A breakthrough came when the lymph node of a gay man was analysed in Paris and a new virus isolated, with both research groups publishing their findings in the same issue of *Science* in 1983 (Barre-Sinoussi et al., 1983; Gallo et al., 1983).

Once the pathogen had been identified, intense work began on how to identify whether someone had the infection. Often, clinicians use antibody tests to ascertain whether someone has been exposed to an infectious agent. Direct visualisation or isolation of the pathogen itself is time-consuming and technically difficult. The then recent test for hepatitis B had used a similar method for detecting previous infection, and this was translated into the first HIV antibody test in 1985—a huge step forward for patients to learn their HIV status in the early phase of infection (and prevent them from passing it onto sexual partners) and to help plan public health prevention strategies once the true prevalence had been ascertained in different communities.

**Early AIDS Treatments**

The early days of the HIV epidemic were characterised by fear, distress and uncertainty. Following the development of an antibody test, scientific discussions about HIV/AIDS turned to possible treatments, with the hope of enabling those diagnosed to survive. This optimism was sadly short-lived. Given the urgency of the situation, a large number of untested and largely ineffective antivirals and immune system ‘boosters’ appeared on the market. Many of these drugs (e.g. isoprinosine) had been used in the treatment of other viruses such as herpes and influenza, and it was hoped that would have a similar effect on HIV. Some other known drugs were able to stimulate the immune system to help control, or even resist, viral infections, such as interferon, a commonly used drug for hepatitis C. There were no data on the effectiveness of these drugs, but in the early
clamour for survival, many searched for a glimmer of hope. The terrible side effects of many of these under-researched drugs were silently tolerated in the hope that they would have some effect on extending the life expectancy of those infected with HIV.

Yet, the only reliable means of reaching any scientifically viable conclusions is to compare new treatments to established therapy in clinical trials. As there were no ‘gold standard’ treatments at the time, an effective agent against HIV proved elusive for many years. Drug development is a lengthy and complicated process; medicines take an average of 17 years to get from the experimental stage to becoming available to patients (Morris, Wooding, & Grant, 2011).

The history of zidovudine (AZT)—the first proven drug to work against HIV—starts in 1964, when Joseph Horwitz, a cancer researcher at Michigan Cancer Foundation, developed the drug to cure certain types of leukaemia. It was found to be ineffective and the compound was shelved. Twenty years later, it was revived by the Wellcome Foundation—a charity on the cutting-edge of antiviral treatments at the time. AZT is a base analogue, that is, it resembles one of the naturally occurring building blocks of DNA found in most human cells. When this imposter is ingested, it is incorporated into the viral DNA and inhibits further transcription, thereby halting viral replication. A number of other HIV drugs work in a similar way. (This is covered in detail in Chap. 5.) After demonstrating that AZT had direct antiretroviral effects, a sample was sent to the FDA in 1984 for further analysis.

Having confirmed its action against HIV, the FDA now set out to test the tolerability of AZT in human subjects. The first clinical trial published in 1987 showed impressive drug efficacy—during the 24-week trial, 19 of the 137 patients in the placebo arm had died, compared to just 1 of the 145 patients taking AZT (Fischl et al., 1987). The results were so dramatic that the trial was stopped early and all of the study patients were given AZT. It later emerged that patients from both arms of the clinical trial were sharing drugs to reduce the chances of taking placebo—an understandable act of desperation by those living with HIV at the time.

Sadly, the optimism surrounding AZT was short-lived. The first antiretroviral (ARV) clinical trial, the CONCORDE study which looked at
placebo versus AZT showed that, after 16 weeks, the mortality rate of those taking AZT was equal to placebo (‘Concorde’, 1994). AZT was found to be effective against HIV but only for a short period of time—when AZT was administered alone, HIV quickly developed resistance to the drug. Drug resistance was not properly understood at the time, but it was clear that this was a huge setback in the treatment of HIV. It was not until 1996 that effective combination therapy consisting of three drugs became a reality. The impact of highly effective ART cannot be underestimated and was a turning point in the fight against HIV.

**HIV/AIDS in the UK**

Although HIV incidence was far higher in the US than in the UK, gay communities in towns and cities throughout the UK were profoundly affected by the arrival of HIV, commonly referred to as the ‘silent killer’ on the gay scene. In 1982, Terrence Higgins, a 37-year-old gay man, was among the first individuals confirmed to die of an AIDS-related illness in the UK. In response to Terrence’s death, several of his friends set up the Terry Higgins Trust to help gay and bisexual men who were living with, at risk of, and affected by HIV. Following a public meeting organised by the Gay and Lesbian Switchboard (now called Switchboard LGBT+), which provided much of the sexual health information to sexual minorities at the time, volunteers came together to support the newly established HIV charity. Hundreds of volunteers manned the telephones, answering questions and signposting callers to appropriate services. In the early days of AIDS, this was the only coordinated response.

The gay community mobilised and recognised the severity and impact of HIV long before the authorities did—publicly at least. Mel Rosen, a well-known HIV activist in the US, attended a meeting organised by the Gay and Lesbian Switchboard and recounted disturbing accounts of young, otherwise healthy men dying in their hundreds. Rosen discussed the steps being taken in San Francisco to assist those affected, such as a buddy scheme for daily tasks and for supporting people living with HIV when others had rejected them.

Following its renaming as the ‘Terrence Higgins Trust’, volunteers from the charity produced the first ever educational leaflet with
practical advice on how to recognise symptoms and to reduce transmission. It also provided advice about how and where to seek treatment and, for gay men, stressed the importance of seeking a doctor aware of HIV and accepting of gay sexuality.

It is noteworthy that, at the time, HIV had not yet been discovered. AIDS was recognised only when individuals became symptomatic. Advice was at times contradictory and inaccurate. For instance, leaflets provided the following advice: ‘Have as much sex as you want, but with fewer and healthy people’. However, it was unclear what ‘fewer’ meant and how one might identify ‘healthy people’. It seems odd now that there was no mention of using condoms or trying to avoid the highest-risk sexual behaviours, such as condomless receptive anal sex. Talking openly about sex was not a socially acceptable method of health promotion in the early 1980s—a social impediment to HIV prevention that would continue for years and only increase the spread of HIV.

HIV entered the British public consciousness for the first time in 1983 when Horizon (a factual programme broadcast by the British Broadcasting Corporation [BBC]) first aired the documentary ‘Killer in the Village’. The programme provided a detailed account of HIV, the risk factors and the experience of gay men living with, or at risk of, HIV. The media played a fundamental role in shaping public understanding of HIV/AIDS. Much of the reporting was simply inaccurate due to the lack of information. It also drew upon the prevailing anti-gay attitudes in 1980s Britain, further fuelling the stigma surrounding gay men and their lifestyle.

The publication of a study in *The Lancet* in 1984 highlighted the growing challenge of HIV in the UK (Cheingsong-Popov et al., 1984). The study had examined 2000 serum samples across distinct populations. At the time HIV was called both HTLV III and LAV (lymphadenopathy associated virus) so they set out to detect both—before the commercially available tests we see today. Using immunofluorescence looking for antibodies, they revealed that 30 out of 31 gay men who had AIDS were HTLV III- and LAV-positive. They also correctly deduced these two viruses were the same. In their study, 79% of patients had lymphadenopathy and 17% showed no symptoms, and it became clear that even someone with advanced disease could still ‘look healthy’. However, it was
still unknown whether those with positive antibody results would go on to develop AIDS. Given the prevalence shown in this early study, this now represented a very real public health emergency.

Yet, the initial political response to AIDS in the UK was ambivalent at best. Initially, AIDS was presumed to affect gay men and injecting drug users only, two of the most marginalised groups in society at the time. Despite fervent campaigning by activists and clinicians in the UK, the political response to the epidemic was slow, indecisive and laced with anti-gay social attitudes.

Despite mounting evidence of increasing numbers of AIDS cases in the UK, there was initially limited political attention to the AIDS crisis—the then Prime Minister Margaret Thatcher and Secretary of State for Health and Social Care, Norman Fowler, said very little about AIDS during its early stages. In 1983, Margaret Thatcher had swept to power promising ‘back to family values’ which clearly resonated with the British electorate who returned a 144-seat majority for the Conservatives. The discourse of HIV prevention (including the overt focus on sex) seemed to contradict the family values which Thatcher’s government attempted to promote. Norman Fowler became a hugely important advocate for HIV prevention and, as a patron for the British HIV Association, remains a staunch supporter of HIV education and prevention to this day.

In 1985, Donald Acheson, the Chief Medical Officer at the time, had met with both clinicians on the front line of the AIDS crisis and volunteers from the newly formed Terrence Higgins Trust to understand how the condition was affecting people in the UK. Clinicians referred to an influx of patients with severe immunosuppression into hospitals which were largely unprepared—this was particularly acute in the London area where most of the first AIDS cases emerged. In the same year, the Expert Advisory Group on AIDS was set up consisting of 22 members of clinicians, researchers, politicians and voluntary sector workers. The principal aim of the Expert Advisory Group on AIDS was to advise the Chief Medical Officer on how to tackle the unfolding public health crisis.

The subsequent report ‘HTLV-III infection, the AIDS epidemic and control of its spread in the UK’ by Acheson (1986) clarified that AIDS posed a significant challenge to public health in the UK, affecting not
only gay men and injecting drug users but also the general population. Dr Acheson wrote:

heterosexual intercourse cannot be excluded as a possible means of transmission. Although the American data suggests that homosexual intercourse is the most important means of sexual spread of HTLV-III infection in our present state of knowledge, it would be wrong for policy to be based on the assumption that heterosexual intercourse will not in the long run assume a significant role.

Having fully understood the scale of the AIDS crisis, Norman Fowler advocated for a public health campaign with clear information about risky sexual practices. This was initially construed as distasteful and potentially harmful to the public and thus resisted by Margaret Thatcher. Though cautious about the language used in public health messaging, the government now realised that it was necessary to talk about sex openly in order to prevent HIV. The government allocated £2.5 million for a public health campaign to provide accurate information about HIV transmission.

There were no data on public awareness of AIDS in 1985 which would inform the public health campaign. In other words, it was unclear what the British public knew or thought about AIDS or how accurate their knowledge was. One US study in 1985 (Price, Desmond, & Kukulka, 1985) revealed high levels of AIDS awareness in adolescents who reportedly had acquired information from the mainstream media. The results showed that, despite high levels of awareness, only 27% of young people believed themselves to be at risk of infection. It seemed that AIDS was perceived to be a ‘gay disease’—a representation also promoted by the mass media—and that anyone outside of this population was not at risk.

Another US survey (DiClemente, Zorn, & Temoshok, 1986) showed that only 66% of respondents knew that AIDS could not be transmitted through kissing or close non-sexual contact. In their London survey (with comparator groups in San Francisco and New York), Temoshok, Sweet, and Zich (1987) found that Londoners were the least worried about
AIDS, but also that those with the least AIDS knowledge thought that they were at much lower risk.

The First Public Health Campaign in the UK

The UK government opted for two separate national press advertisements—each full page for maximum impact—in all Sunday papers. The wording of the adverts was to become contentious as Margaret Thatcher resisted references to risky sex, noting in a Number 10 memo that ‘Do we have to have the section on risky sex? I should have thought it could do immense harm if young teenagers were to read it’.¹

The fact that the AIDS public health campaign was conducted from neither the Cabinet Office nor Number 10 directly suggests that the Prime Minister wished to distance herself from a public backlash which could be politically damaging. At one point, Margaret Thatcher questioned the legitimacy of using the term ‘anal sex’ and enquired about its consistency with the Obscene Publications Act—the term ‘rectal sex’ was finally agreed upon.

This highlighted one of the biggest barriers to the initial public health campaign, because of its association, in most cases, with sexual behaviour. This meant that HIV prevention in turn necessitated open discussions about sex and gay sexual behaviour, which was not openly discussed before. Due to pervasive conservative attitudes at the time and indeed a government that itself espoused a socially conservative ideology, this was challenging—political hesitation about engaging with HIV and sexual behaviour undoubtedly fuelled the epidemic.

The advertisements were finally published on 16 March 1986 and consisted of poorly presented dense text with no mention of high-risk sex, condoms or how to reduce the risk of infection. Both the Expert Advisory Group on AIDS and the Terrence Higgins Trust emphasised that a clearer message about AIDS and its risk factors was necessary for it to resonate among all members of the public and not only among those who were believed to be at risk. The government faced pressure from

¹ http://discovery.nationalarchives.gov.uk/details/r/C15189597.
those on the right of its party but, to its credit, proceeded with its HIV prevention campaign. £20 million was allocated to include a leaflet on HIV which was delivered to every household in the UK, a television advertisement, billboard posters and another newspaper campaign. The iconic ‘tombstone’ television advert, ‘Don’t Die of Ignorance’, was deliberately unsettling and alarmist and drew on imagery of death for maximum impact.

The TV advert appeared to utilise fear as a catalyst for awareness, engagement and behaviour change. The aim of the campaign was to ensure that everyone had heard of AIDS, knew how it was transmitted and, crucially, knew how to reduce their own risk of infection. It is doubtful that all of these objectives were achieved due to the pressure to avoid discussions about sexual behaviour.

There is much debate about the value of fear in increasing awareness, engagement and behaviour change (Ruiter, Abraham, & Kok, 2001). Although some believed that the campaign marginalised certain groups and instilled unnecessary fear in those who were in fact at low risk of HIV infection, the campaign did have a profound effect on the British public and is still remembered by many today who witnessed it first-hand. The impact of the campaign on sexual health was nothing short of astonishing. The incidence of gonorrhoea, a useful proxy for condomless sex, decreased from 50,000 cases per year to 10,000 following the airing of the television advertisements (Mohammed et al., 2018). National interest was maintained when both the Independent Television (ITV) and BBC began a week-long series of programmes called ‘AIDS Help!’ in which politicians, activists and alternative comedians discussed HIV on television. The use of television, in addition to the print media, made a significant contribution to HIV education and myth-busting in the British public.

Gay men were the group most affected by HIV and at highest risk of infection. They were, however, a stigmatised group whose identities, lifestyles and health the government generally avoided acknowledging. Thus, there was no government-endorsed campaign to prevent HIV among gay men until 1989, which generated confusion, misinformation and, undoubtedly, further engagement in high-risk sexual behaviour. In the
absence of an institutional campaign, community groups, such as the Terrence Higgins Trust and the National AIDS Trust (formed in 1987 with an emphasis on HIV policy development), provided grassroots education to ensure accurate information, dispel HIV myths and ultimately curb the spread of HIV in the gay community. The government understood that community organisations were best positioned to provide HIV education and that they would have the greatest impact on their respective communities. In 1989, David Mellor, the Minister for Health at the time, pledged increases in funding for HIV. £25 million was provided in 1988, which rose to £62 million and then to £132 million in 1989 and 1990, respectively. Of this, £62 million was set aside for HIV treatment and care with the rest allocated to education and support for both those at risk and patients already living with HIV.

The involvement of the UK government and the pledge of funding to support HIV education, advocacy and prevention undoubtedly saved many thousands of lives in the pre-ART era when HIV still constituted a life-limiting condition. It is important to note that this early political intervention was by no means common across Europe and, thus, in many other countries the virus devastated high-risk groups.

The late 1980s saw a gradual change in tone towards those living with HIV as understanding improved and the general public realised that it affected all groups indiscriminately. In 1987, Princess Diana, a trailblazing advocate of HIV education, was one of the first celebrities to be photographed touching and hugging people living with HIV. It must be remembered that many at the time thought that one could contract HIV from touching. These iconic images helped dispel some of the myths concerning HIV transmission and sought to normalise the condition for many across the world. Princess Diana continued her advocacy work until her death, which has subsequently been taken forward by her son Prince Harry.

In 1988, the first ‘World AIDS Day’ was created with an emphasis on testing and educating those who might not perceive themselves to be at risk. At this time, HIV was still largely thought of as the ‘gay plague’ and the result of lifestyle choices made by gay men.
The Slow March to Controlling the Virus

A huge shift in British social attitudes towards HIV occurred in 1990 when *Eastenders*, one of the most popular BBC soap operas in the UK, introduced its first HIV-positive character. Mark Fowler, an affable market trader, was a heterosexual man who had acquired HIV from his previous girlfriend. The show sought to challenge stereotypes about those living with HIV and demonstrated that everyone was at risk, not just gay men. With a viewership of up to 30 million, over half the country was able to witness the issues facing those living with HIV. The importance of this storyline cannot be underestimated. It dealt with relevant topics, such as the difficulties around HIV disclosure, use of third-sector organisations (he attended therapy at the Terrence Higgins Trust) and how people react to HIV based on their own lack of knowledge—which many gay men had experienced. This was followed by the death of Freddy Mercury, lead singer of *Queen*, in 1991 which, given the band’s huge following worldwide, sought to highlight the need for testing and early diagnosis. At that time, more open discussions around sexual practices were taking place slowly, which began to challenge stigma and deeply held prejudices against gay men and HIV.

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**Clinical Snapshot 1: Antiretroviral Therapy**

The most significant turning point in the treatment of HIV came in July 1996 at the 11th Conference for AIDS in Vancouver, where it was shown that giving patients 3 drugs (i.e. combination therapy) with the use of the newly released protease inhibitors, led to significant reductions in AIDS deaths by 60–80%. These landmark findings revolutionised the treatment of HIV with patients no longer facing imminent death and led to the now accepted model of treating the majority of HIV patients in an outpatient setting, with early testing and treatment being the norm. Early HIV drugs were often toxic and required perfect adherence to be effective, but offered hope to thousands of people who were infected. The evolution of combination therapy has been rapid and now the emphasis is on quality of life, principally by reducing the side effects of antiretroviral therapy. As treatments continue to evolve, longer acting injectable drugs and implants will become the standard of care in the future, negating the need for daily oral therapy and improving patient satisfaction and adherence to medication.
As HIV testing became standardised and more commonplace, more infections were detected. There was an increase in new HIV diagnoses in 1985, most likely reflecting improved testing rather than actual prevalence. After the public health campaigns and HIV education packages, the number of new HIV diagnoses in the late 1980s and early- to mid-1990s remained relatively stable at just under 2000 new diagnoses per year. With the advent of effective treatment in 1996, the number of people dying from AIDS- and HIV-related complications reduced dramatically. However, a side effect of this reduction in AIDS-related mortality was a creeping complacency within the gay community in relation to the disease. It was no longer a death sentence. In the UK, the drugs were effective, free and easily available. Younger gay men had not lived through the trauma of the AIDS crisis with many having a misplaced sense of security about the longer-term effects of living with HIV. As such, at the turn of the century HIV rates began to increase year on year for the next 15 years with 3480 new diagnosis in 2015, nearly double the figure from the 1990s (see Fig. 2.1).

During these years of a steady increase in new diagnoses, public health campaigns became more nuanced with the ‘use a condom’ message
staying front and centre but also utilising more third-sector organisations to provide HIV education. Many of these organisations had the knowledge and prior experience of engaging certain communities at risk of HIV and took an approach which they knew would resonate with their target audience—and using colloquial language to demystify some of the technical terms that were used in other campaigns.

In 2004, the Blair Labour government published a white paper ‘Choosing Health’ which, for the first time, prioritised sexual health. Much of the focus was on the rising rates of chlamydia in young people, but the paper was instrumental in setting standards for sexual health and improving access to testing, raising awareness of HIV and increasing funding for overstretched sexual health services. The New Labour model of introducing targets in healthcare were instrumental in improving HIV testing amongst all groups, not just gay men.

From 2010, there was an acceleration of new HIV infections in gay men due in part to the proliferation of smart phone location-based social networking applications (e.g. Grindr, Scruff) whereby gay men could find a sexual partner within minutes in their local area if desired (Jaspal, 2017). Prior to this, only computer-based applications, such as Gaydar, were available and lacked the speed and convenience of the newer smart phone applications. During this time, there was also an explosion of ‘chemsex’, that is, the use of drugs in sexualised settings. This is discussed further in Chap. 3.

Clinicians in the UK have struggled to fully understand the complexities of this new era of HIV risk behaviour. It is not uncommon to hear of someone taking crystal methamphetamine, being awake for many days and, with the use of Grindr, having dozens of sexual partners within a relatively short period of time. The identification and treatment of this subset of the gay population has been difficult and is contingent on the ability of clinicians and services to respond effectively to this challenge. As many gay men are now also injecting drugs (or ‘slamming’), the standard injecting drug services have struggled to cope with the complex health issues in this population. As the landscape has shifted, so have the

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2 https://webarchive.nationalarchives.gov.uk/+/http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4094550.
public health campaigns, and much work has been undertaken to understand the emerging risks associated with young gay men today. Some of these risks were summarised in Chap. 1.

In addition to the increasingly nuanced public health campaigns to improve HIV awareness, three significant developments since 2015 have turned the tide in HIV incidence in the UK. First, PrEP has enabled gay men to take control of their HIV risk by taking a pill on either a daily or intermittent basis to prevent their acquisition of HIV. As PrEP is only dispensed for three to six months at a time, users must attend sexual health services where testing can be performed and high-risk behaviours identified and addressed. PrEP is covered in more detail in Chap. 4 on HIV prevention. Second, there has been an increase in HIV testing, which is central to early HIV diagnosis. On the one hand, this facilitates the identification of those with undiagnosed HIV who may be transmitting the virus to others, and, on the other hand, it provides an opportunity for increasing patients’ awareness and understanding of HIV and appropriate prevention approaches. Third, those who are living with HIV and on effective ART cannot transmit HIV to their sexual partners. This powerful U = U message (undetectable = untransmittable) has had a seismic impact on the lives and wellbeing of gay men and all those living with HIV. They no longer need to be in fear of onward transmission. Moreover, U = U has the potential to challenge social stigma surrounding HIV given that fear of infection constitutes a key component of stigma.

In view of these significant advances, between 2012 and 2018, HIV incidence in gay, bisexual and other men who have sex with men (MSM) in the UK decreased by 71% (O’Halloran et al., 2020). This sharp decline should be cause for celebration as gay men begin to utilise the various different forms of HIV prevention and adapt them to their own lifestyles and attitudes. Moreover, sexual health charities have developed innovative ways of reaching subgroups within the gay community which have historically been viewed as ‘hard-to-reach’. However, there remains much work to be done.
HIV Today: An Epidemiological Snapshot

HIV prevalence in the UK is approximately 0.18% of the population aged between 15 and 59. Gay men constitute a relatively small minority group and are estimated to represent approximately 2% of the London population. Yet, Public Health England (2018) data show that, of the 103,800 people currently living with HIV, approximately 49,800 are gay, bisexual or other MSM. Moreover, it is estimated that 1 in 11 gay men in London is HIV-positive. HIV incidence in gay and bisexual men is reducing year on year due to a mixture of increased testing, availability of PrEP and treatment as prevention for those living with HIV.

There have been significant increases in most STIs in gay men from 2014 to 2018. A 61% increase in both chlamydia (from 11,760 to 18,892) and syphilis (from 3527 to 5681), and a 43% increase in gonorrhoea (from 18,568 to 26,574). The increase in STIs is multifactorial with many attributing this to reduced condom use due to PrEP and those living with HIV being unable to transmit the virus when on effective treatment. The fact remains that many gay men have never used condoms for a variety of reasons. If anything, this increase in STIs allows sexual health clinics to intervene in those who may need support around drug or alcohol use and may never have visited a sexual health clinic previously.

Overview

In this chapter, the socio-historical, scientific and epidemiological aspects of HIV were considered. Since the initial clinical observations of HIV, there have been significant scientific developments, which have resulted in a much improved disease prognosis for those diagnosed and treated early. Moreover, biomedical tools for preventing HIV are highly effective. These developments have not occurred in a social vacuum but rather they have been shaped by activism, society and politics. As demonstrated in
this chapter, the stigma associated with both sex and the groups disproportionately affected by HIV at the start of the epidemic caused some political trepidation about discussing the disease openly. Moreover, there were challenges in addressing the possible drivers of infection in key populations, such as gay men. History demonstrates that, while silence may have facilitated short-term political victories for some, its implications for the progression of HIV/AIDS have been devastating. Where there is silence, there is decreased awareness, understanding and action against the virus. The fight against this invisible enemy was, and still is, a feat of human endurance against all odds. The human cost of HIV/AIDS would have been far higher unless the numerous community groups, activists, scientists, clinicians and politicians had stepped up and tackled not only the effects of the virus but also the insidious stigma of HIV. Their efforts have enabled the general population to acknowledge and understand the health of marginalised groups within our societies. Often, the groups most affected by HIV are unable to advocate in order to challenge the thinking of politicians and the general public. HIV would be an even more dangerous global threat if these people had not stepped in to help. To achieve the eradication of HIV, public health campaigns should be based on evidence and education to help those at risk understand how to have a healthy sex life without fear or prejudice. These campaigns must not be marred or impeded by politics. This chapter elucidates the centrality of the socio-historical dimension of HIV in enabling us to control, curb and, ultimately, eradicate the virus.

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