HIV-2 Infection

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Module 6: Key Populations
Lesson 8: HIV-2 Infection

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Background

Human immunodeficiency virus (HIV) is classified into two types: HIV-1 and HIV-2. Although HIV-1 and HIV-2 share the same routes of transmission, and both can cause acquired immunodeficiency syndrome (AIDS), important differences exist between the viruses in epidemiology, natural history, diagnosis, and management.[1,2,3] Compared with individuals with HIV-1, persons with HIV-2 typically have attenuated clinical progression and lower rates of sexual and perinatal HIV transmission.[4,5,6,7] Significant differences exist in the antiretroviral management of HIV-1 and HIV-2.[2] Clinicians should become familiar with the differences between HIV-1 and HIV-2 and maintain a high index of suspicion of HIV-2 in persons from regions where HIV-2 circulates. Acquisition of HIV-2 does not protect against infection with HIV-1, and acquisition of HIV-1 does not protect against acquisition of HIV-2.[3,8] Thus, it is important to understand that same individual can acquire both HIV-1 and HIV-2.[8,9]

HIV-2 Epidemiology

HIV-2 Globally

The World Health Organization (WHO) estimated that 41 million people were living with HIV worldwide in 2025, but similar HIV-2 specific prevalence estimates are lacking.[10] Since the beginning of the HIV pandemic, approximately 1 to 2 million people have been infected with HIV-2, including those with both HIV-1 and HIV-2.[1,11,12,13] Most persons infected with HIV-2 reside in West Africa or in countries with strong colonial or socioeconomic ties with West Africa, most notably France, Spain, and Portugal. Significant numbers of HIV-2 cases have been reported in significant numbers in several former Portuguese colonies, including Angola, Mozambique, Brazil, and India (mainly in the states of Goa and Maharashtra and to a lesser degree in some southern regions).[1] During the last two decades, HIV-2 prevalence has declined in several West African and European countries, but the reasons for this are unclear.[14,15,16]

HIV-2 in the United States

In the United States, it is estimated that fewer than 1% of persons with HIV have HIV-2. The first case of HIV-2 in the United States was reported in 1987.[17] Most individuals diagnosed with HIV-2 in the United States have emigrated from a region where HIV-2 is endemic region or have had exposure to a person from an HIV-2 endemic region.[18] In addition, most of the earlier reported small number of cases of HIV-2 in the United States have been clustered in the Northeast.[18,19] A Centers for Disease Control and Prevention (CDC) analysis of National HIV Surveillance System (NHSS) 2010–2017 data concluded that an HIV-2 diagnosis was rare in the United States during that time period.[20] The image series below summarizes the major characteristics of persons in the report who were diagnosed with HIV-2 (Figure 1).[20]

Pathogenesis, Transmission, and Natural History of HIV-2

Pathogenesis of HIV-2

Compared with HIV-1, HIV-2 is less virulent and is characterized by lower plasma HIV RNA levels, a slower decline in CD4 cell counts, and a longer time to progress to AIDS.[2,8,14] In a prospective cohort of 133 persons with HIV-2 who were not receiving antiretroviral therapy from 1991 through 2009, most participants had persistently low HIV-2 RNA levels remained consistently low, with 36 to 42% of the HIV-2 RNA levels below 100 copies/mL.[21] This same study showed the rate of disease progression and mortality rates correlated with the baseline plasma HIV-2 RNA levels obtained in 1991: individuals with baseline HIV-2 RNA levels less than 100 copies/mL had very low mortality.[21] In another longitudinal follow-up study in West Africa, investigators found that HIV-2 is more pathogenic than previously thought and that most people with untreated HIV-2 will eventually develop AIDS within 15 years of HIV acquisition (Figure 2).[22] The exact reasons for the differences in pathogenicity between HIV-1 and HIV-2 remain incompletely defined, but both intrinsic viral factors and innate and adaptive immunity are likely to play important roles.[8] Persons with HIV-2 who suffer immunologic decline develop opportunistic infections similar to those in individuals with HIV-1.[8]

Transmission of HIV-2

Studies on the natural history of perinatal transmission conducted in West Africa (in the pre-antiretroviral era) found that the rate of perinatal transmission of HIV-2 was significantly lower than for HIV-1, typically less than 5% versus approximately 25%.[7,23] Sexual transmission and genital shedding are also less efficient in persons with HIV-2.[6,24,25,26] Acquisition of HIV-2 does not provide protection against infection with HIV-1.[27]

Effect of HIV-2 Coinfection on HIV-1 Progression

Limited data exist on the natural disease progression in persons with concomitant HIV-1 and HIV-2 infection; in West Africa, up to 15% of individuals have dual infection, though the proportion of persons with HIV-2 monoinfection has decreased in recent years.[8,28] One study suggested that persons with dual HIV-1 and HIV-2 infection have slower disease progression and delayed death compared with individuals who have HIV-1 monoinfection, with the greatest benefit occurring when HIV-2 infection precedes HIV-1 infection.[28] Other studies, including a robust meta-analysis, found no survival benefit in persons dually infected with HIV-1 and HIV-2 compared to persons with HIV-1 alone.[29,30]

Surveillance Case Definition for HIV-2 Infection

In 2014, the Centers for Disease Control and Prevention released a Revised Surveillance Case Definition for HIV Infection that added specific criteria for defining a case of HIV-2, which were not part of the 2008 case definition.[31] To classify an adult as having HIV-2, one or more of the following laboratory criteria are necessary:

- FDA-approved HIV-1/HIV-2 type-differentiating antibody test result positive for HIV-2 and negative for HIV-1,
- Positive HIV-2 Western blot result and negative or indeterminate HIV-1 Western blot result,
- Positive qualitative HIV-2 nucleic acid test (NAT),
- Detectable quantitative HIV-2 NAT (viral load), *or*
- Laboratory results are interpreted as consistent with HIV-2 infection by a laboratory expert experienced in differentiating HIV-2 from HIV-1 (if laboratory evidence for HIV-2 is ambiguous).

In addition, the 2014 Revised Surveillance Case Definition for HIV Infection classifies an individual as having dual infection (with HIV-1 and HIV-2) if both the HIV-1 nucleic acid test (NAT) and the HIV-1 NAT are positive.[31]

Diagnostic Testing for HIV-2

Approach to Diagnostic Testing for HIV-2

Using the tests as recommended in the CDC and Association of Public Health Laboratories (APHL) HIV diagnostic algorithm can reliably detect HIV-2; the HIV-1/HIV-2 antigen-antibody test is the recommended initial test, followed by an HIV-1/HIV-2 differentiation immunoassay if the initial test is positive (Figure 3).[32, 33] In some circumstances, such as an indeterminate HIV-2 result on the HIV-1/HIV-2 differentiation immunoassay, HIV-2 NAT diagnostic testing is recommended.[34] Diagnostic HIV-2 testing (qualitative HIV-2 RNA) is available through multiple commercial and clinical laboratories, including some tests that provide both qualitative detection and differentiation of HIV-2 and HIV-1. It is important to recognize that about 30 to 40% of persons with HIV-2 who are not receiving antiretroviral therapy have undetectable plasma HIV-2 RNA levels; for this reason, HIV-2 RNA testing alone is not a reliable screening test.[35,36,37]

Interpretation of Diagnostic Tests in HIV-2

- **Enzyme Immunoassay (EIA)**: A person with HIV-2 will likely have a positive HIV enzyme immunoassay (EIA) regardless of which test is used because most, but not all, EIA tests detect both HIV-1 and HIV-2.[38] Commercially available EIAs do not generally differentiate between HIV-1 and HIV-2, although there is a specific HIV-2 EIA that is FDA-approved (Genetic Systems HIV-2 EIA). Several point-of-care EIA HIV tests are FDA-approved for the detection of both HIV-1 and HIV-2.
- **Differentiation Assays for HIV-1/HIV-2**: Two assays are available in the United States that can differentiate HIV-1 from HIV-2.[39,40,41] The CDC-recommended algorithm for HIV testing utilizes the HIV-1/HIV-2 differentiation assay as the second step in the algorithm.[32]
- **HIV Western Blot**: The HIV Western blot is no longer routinely used for HIV diagnosis and is not recommended in the CDC HIV testing algorithm. Infection with HIV-2 may cause a negative, indeterminate, or positive HIV-1 Western blot due to cross-reacting antibodies. Persons with HIV-2 often have an indeterminate HIV-1 Western blot pattern, with the presence of Gag bands (p55, p24, or p17) and Pol bands (p66, p51, or p31), but the absence of Env bands (gp160, gp120, or gp41) (Figure 4).[1,19] The Western blot pattern in persons with HIV-2 occurs because HIV-1 and HIV-2 have 60% similarity in the regions encoding *gag* and *pol*, compared with only 30 to 40% similarity in the region encoding *env*.[42] In the United States, HIV-2 Western blot tests are not FDA-approved, but some HIV-2 supplemental HIV-2 antibody tests are commercially available through reference laboratories.[1]
- **Qualitative Plasma HIV-2 RNA and HIV-2 DNA**: An HIV-2 nucleic acid test is typically utilized to confirm a positive HIV-2 differentiation assay or to provide additional information when an HIV-1/HIV-2 differentiation assay is indeterminate. A positive qualitative HIV-2 RNA confirms a diagnosis of HIV-2. A negative HIV-2 plasma RNA test, however, does not rule out HIV-2, since approximately 30 to 40% of persons with untreated chronic HIV-2 have undetectable plasma HIV-2 RNA levels.[35,36,37] To address this issue, several laboratories have developed a qualitative assay that detects HIV-2 DNA in individuals with HIV-2 who have undetectable plasma HIV-2 RNA levels.[43]

Antiretroviral Activity and Resistance

The following summarizes what is known related to the activity and resistance of antiretroviral medications and HIV-2. At this time, HIV-2 drug resistance genotypic testing is not commercially available in the United States, but may be available in some centers as a research use-only test.

Nucleoside Reverse Transcriptase Inhibitors (NRTIs)

In general, nucleoside reverse transcriptase inhibitors (NRTIs) are active against HIV-2, but due to naturally occurring polymorphisms in HIV-2, they may have less activity and a lower genetic barrier to resistance with HIV-2 than HIV-1.[44] Although HIV-1 and HIV-2 share some classic NRTI resistance mutations, such as the M184V mutation, which causes high-level resistance to lamivudine and emtricitabine, HIV-2 often follows different resistance pathways than HIV-1. For example, HIV-2 resistance to zidovudine occurs through the Q151M mutation rather than through the common thymidine analog mutation (TAM) pathways typically observed with HIV-1.[45] Further, studies have produced conflicting results regarding the frequency of the K65R mutation in HIV-2 and its impact on susceptibility to tenofovir and abacavir.[44,46,47] Available data suggest the development of the Q151M mutation in combination with a K65R or M184V mutation results in resistance to zidovudine, lamivudine, and emtricitabine; the presence of all three mutations together causes broad NRTI class resistance.[48] Islatravir has potent antiviral activity against HIV-2, but there are no clinical trials to support the use of islatravir to treat HIV-2.[49]

Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs)

Multiple studies have shown that HIV-2 has intrinsic resistance to all non-nucleoside reverse transcriptase inhibitor (NNRTI) drugs.[44] This intrinsic resistance occurs because the Y181I and Y188L substitutions are natural polymorphisms present in HIV-2 strains.[51] These mutations alter the NNRTI binding pocket in the reverse transcriptase enzyme, rendering it less receptive to NNRTI medications; this includes the newest NNRTI, doravirine, as well as long-acting injectable rilpivirine as part of the injectable cabotegravir and rilpivirine combination.[51]

Integrase Strand Transfer Inhibitors (INSTIs)

All the INSTIs, including bictegravir, cabotegravir, dolutegravir, elvitegravir, and raltegravir, have potent activity against HIV-2.[2,48,52,53,54,55,56] A small, single-arm, clinical trial from Portugal showed that dolutegravir plus 2 NRTIs was effective for treatment-naïve persons with HIV-2, and another small study showed modest efficacy with dolutegravir in persons with HIV-2 who had resistance to first-generation INSTIs.[57,58] As with HIV-1, raltegravir and elvitegravir have a relatively low barrier to resistance to HIV-2, due to intrinsic polymorphisms at secondary integrase sites.[57,59] Resistance that affects the susceptibility of HIV-2 to INSTIs shares some similarities with that of HIV-1, though some key differences exist.[55,56,60] For example, the integrase mutations N155H or Q148R confer resistance to raltegravir in both HIV-2 and HIV-1, but in HIV-2, the Y143 pathway requires secondary mutations to cause significant resistance (contrary to what is observed with HIV-1).[55] A study revealed a new resistance pathway of HIV-2 to INSTIs that involves a 5-amino acid insertion at codon 231 of the HIV-2 integrase, a region in the integrase C-terminal domain.[61,62,63] This insertion mutation results in high-level resistance to elvitegravir and raltegravir and moderate-level resistance to dolutegravir.[61] Resistance to bictegravir was observed in several isolates, but overall, it retained the most potent INSTI in isolates with the codon 231 insertion mutation (231INS).[61]

Protease Inhibitors

Several studies have reported that HIV-2 has inherent partial or full resistance to some protease inhibitors, with only lopinavir-ritonavir, darunavir, and saquinavir having clinically useful activity against HIV-2.[44,64,65,66,67] Investigators have identified four residues at amino acid positions 32, 47, 76, and 82 in the HIV-2 protease binding cleft that confer class-wide sensitivity of HIV-2 to PIs.[68] Studies have shown

that fewer HIV protease mutations are necessary for resistance to develop in HIV-2 compared with HIV-1. Due to innate polymorphisms, a lower number of mutations is required for HIV-2 to become resistant to PIs as compared to HIV-1.[69] Similar to treatment for HIV-1, if a protease inhibitor is used to treat HIV-2, boosting with ritonavir or cobicistat is recommended. Based on available data, the recommended protease inhibitor-based regimens for HIV-2 consist of boosted darunavir or lopinavir, in combination with two NRTIs.[2] Notably, atazanavir is not recommended for the treatment of HIV-2.

Entry Inhibitors

- **Attachment Inhibitors**: Limited data suggest that temsavir, the active moiety of fostemsavir, is not active against HIV-2 in vitro.[70,71]
- **Post-Attachment Inhibitor**s: Limited in vitro data show that ibalizumab is active against HIV-2.[72]
- **CCR5 Inhibitors**: The efficacy of the CCR5 inhibitor, maraviroc, is uncertain since HIV-2 can use several different co-receptors to enter cells, and there is no commercial HIV-2 co-receptor tropism screening assay that would determine whether the patient has pure CCR5-tropic HIV-2.[1,2,73,74]
- **Fusion Inhibitors**: In vitro data have shown HIV-2 has intrinsic resistance to the fusion inhibitor, enfuvirtide. Resistance to enfuvirtide correlates with genetic diversity at the target regions for the drug, namely the HR1 domain of the viral gp41 region.[75]

Capsid Inhibitors

Limited in vitro data show that lenacapavir, currently the only available HIV capsid inhibitor, has activity against HIV-1 and HIV-2, but this activity is 11- to 25-fold less than against HIV-1.[71,76] More data are needed on lenacapavir and HIV-2.

Foscarnet

Limited data suggest foscarnet, a pyrophosphate analog polymerase inhibitor, can be considered as part of a salvage regimen in people with multidrug-resistant HIV-2.[77,78,79]

HIV-2 Treatment Studies

Data to guide the optimal timing or regimen choice for initial antiretroviral therapy in persons with HIV-2 are limited, consisting of three small, single-arm trials of INSTI-based (raltegravir, elvitegravir, and dolutegravir) treatment for initial antiretroviral therapy.[5,58,80] In addition, a randomized controlled trial (FIT-2) compared three regimens for the treatment of HIV-2: (1) raltegravir plus tenofovir DF plus emtricitabine (or lamivudine), (2) lopinavir-ritonavir plus tenofovir DF plus emtricitabine (or lamivudine), and (3) zidovudine plus tenofovir DF plus emtricitabine (or lamivudine).[81] The regimens that contained raltegravir or lopinavir-ritonavir were safe and effective, but the arm with the triple NRTI regimen was stopped due to inferiority.[81] A retrospective study of 24 individuals with HIV-2 (5 treatment-naïve and 19 treatment-experienced) found that bictegravir-tenofovir alafenamide-emtricitabine was effective in treating HIV-2.[82] The limited data on HIV-2 treatment are due to a combination of factors, including the low prevalence of HIV-2 (especially in the United States and Europe) and the lower virulence of HIV-2 compared with HIV-1, which has made investigation of antiretroviral therapy for HIV-2 a lower priority than for HIV-1.[1]

HIV-2 Treatment Recommendations

Timing of Initiating Antiretroviral Therapy with HIV-2

Although individuals with HIV-2 generally have slower disease progression than persons with HIV-1, they generally have a less robust CD4 count increase in response to antiretroviral treatment.[83,84,85,86] This poor CD4 cell count response to antiretroviral therapy in persons with HIV-2 suggests that persons with HIV-2 should start antiretroviral therapy without delay. In addition, early treatment of HIV-2, in theory, would reduce transmission of HIV-2 to others.

- **Recommendation**: The Adult and Adolescent ARV Guidelines recommend starting antiretroviral therapy at or soon after HIV-2 diagnosis to prevent disease progression and transmission of HIV-2 to others.[2]

Recommended Antiretroviral Regimens for Treatment of HIV-2

The following summarizes key recommendations in the Adult and Adolescent ARV Guidelines for the treatment of HIV-2 (without HIV-1 infection).[2]

- Based on experience with the treatment of HIV-1, a three-drug antiretroviral regimen should be used to treat HIV-2 to maintain viral suppression and to avoid the development of resistance from suboptimal therapy.
- Since resistance testing is not commercially available for HIV-2, baseline resistance testing is not an option to guide initial therapy. Transmitted HIV-2 drug resistance has been reported, but to date, appears to be rare.[87,88]
- The preferred treatment regimen for persons with HIV-2 should consist of 2 NRTIs in combination with an INSTI (bictegravir, dolutegravir, elvitegravir, or raltegravir).
- The alternative regimen is two NRTIs plus a boosted protease inhibitor (darunavir or lopinavir) active against HIV-2. If a protease inhibitor is used, boosted darunavir may be preferred over boosted lopinavir because it is better tolerated.
- The following medications should not be used to treat HIV-2: any medication in the NNRTI class, fostemsavir, enfuvirtide, and long-acting injectable cabotegravir and rilpivirine.
- Persons with HIV-2 and hepatitis B virus (HBV) coinfection require an antiretroviral regimen that contains drugs with activity against both HIV-2 and HBV. They should also be tested for hepatitis D (delta) virus as coinfection with HBV is not uncommon in West Africa.[89]
- For persons with multidrug-resistant HIV-2, ibalizumab and lenacapavir could be considered as part of a salvage regimen, based on in vitro activity.

Treatment of Persons with HIV-1 and HIV-2 Dual Infection

The following summarizes key recommendations in theAdult and Adolescent ARV Guidelines for the treatment of HIV-1 and HIV-2 dual infection.[2]

- Individuals with HIV-1 and HIV-2 dual infection should undergo baseline genotypic resistance testing for HIV-1; resistance testing for HIV-2 is not commercially available in the United States.[2,90] In this setting, if possible, monitoring of both HIV-1 and HIV-2 plasma RNA levels should be performed.[91,92]
- The preferred treatment of persons with HIV-1 and HIV-2 dual infection is 2 NRTIs in combination with an INSTI, even if the baseline plasma HIV-2 RNA level is low or undetectable.
- All the preferred regimens for the treatment of HIV-1 have good activity against both HIV-1 and HIV-2.
- The following medications should not be used to treat persons with dual HIV-1 and HIV-2 infection: any medication in the NNRTI class, fostemsavir, enfuvirtide, and long-acting injectable cabotegravir and rilpivirine.

- Persons with HIV-1 and HIV-2 dual infection and HBV coinfection require an antiretroviral regimen that contains drugs with activity against HIV-1, HIV-2, and HBV.
- For persons with multidrug-resistant HIV-1 and HIV-2, ibalizumab and lenacapavir could be considered as part of a salvage regimen, based on in vitro activity.

Clinical Follow-up and Laboratory Monitoring for HIV-2

Until further HIV-2 treatment data are available, clinicians should follow recommendations for HIV-1 clinical management and HIV primary care, including opportunistic infection prophylaxis and laboratory monitoring on antiretroviral therapy.[2] The HIV-1 nucleic acid tests do not reliably detect or quantitate HIV-2. Quantitative plasma HIV-2 RNA viral load assays for monitoring response to therapy are available through the University of Washington Laboratory Medicine (HIV-2 RNA Quantitation) and the New York State Department of Health (HIV-2 Quantification). Therefore, the management of persons with HIV-2 on antiretroviral therapy should include routine monitoring of quantitative plasma HIV-2 RNA levels, similar to what is done for persons with HIV-1. Ongoing CD4 count monitoring is also recommended in persons with HIV-2, even if their viral load is undetectable or suppressed.

Summary Points

- Compared with HIV-1, HIV-2 is a less virulent and less transmissible virus. More recent data have shown that HIV-2 is more pathogenic than previously thought, and most persons with HIV-2 will develop AIDS within 15 years.
- The CDC/APHL HIV diagnostic testing algorithm (1) detects HIV-1 and HIV-2 infection in the initial screening test and (2) distinguishes HIV-1 and HIV-2 using the HIV differentiation assay used in the second step.
- Persons with HIV-2 have poorer CD4 cell count responses to antiretroviral treatment relative to persons with HIV-1, so early diagnosis and early initiation of therapy for HIV-2 should be emphasized.
- HIV-2 has intrinsic resistance to all NNRTI drugs, some PIs, fostemsavir, and enfuvirtide.
- HIV-2 is generally susceptible to NRTIs, INSTIs, and certain PIs, although naturally occurring polymorphisms may result in a lower genetic barrier to resistance to HIV-2 than to HIV-1. Among the available PIs, lopinavir and darunavir have the best activity against HIV-2.
- The preferred antiretroviral treatment for HIV-2 infection is 2 NRTIs plus an INSTI. The alternative regimen is 2 NRTIs plus a boosted PI (darunavir or lopinavir).
- Individuals with HIV-1 and HIV-2 coinfection should receive an antiretroviral regimen that can effectively treat both viruses, ideally two NRTIs plus an INSTI.
- In the event of clinical or virologic failure on antiretroviral therapy, consultation with an expert in HIV-2 management is recommended.

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Figures

Figure 1 Characteristics of Persons Diagnosed with HIV-2, United States, 2010–2017

Source: Peruski AH, Wesolowski LG, Delaney KP, et al. Trends in HIV-2 Diagnoses and Use of the HIV-1/HIV-2 Differentiation Test - United States, 2010-2017. MMWR Morb Mortal Wkly Rep. 2020;69:63-6.

This is a dynamic visualization. Please visit our website to experience this dynamic content.

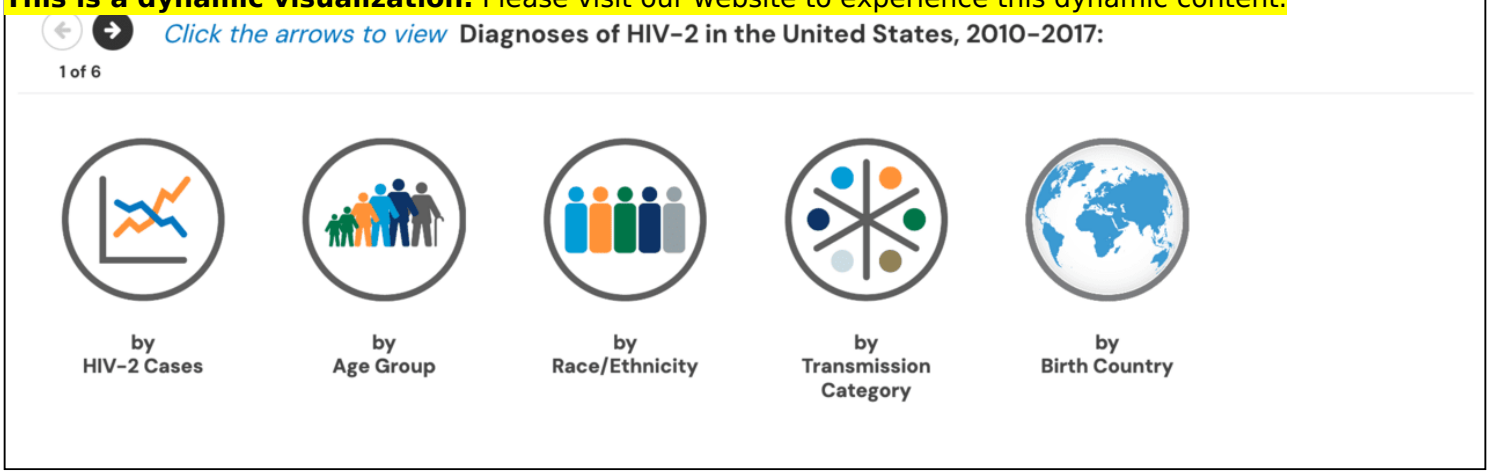


Figure 2 Median Time to AIDS and Death in Persons with HIV-1 or HIV-2 in West Africa

This graphic shows follow-up of 225 persons with HIV-1 and 87 with HIV-2. Median time to development of AIDS was slower in persons with HIV-2, but median survival was brief after AIDS in both groups.

Source: Esbjörnsson J, Månsson F, Kvist A, et al. Long-term follow-up of HIV-2-related AIDS and mortality in Guinea-Bissau: a prospective open cohort study. Lancet HIV. 2018;S2352-3018(18)30254-6.

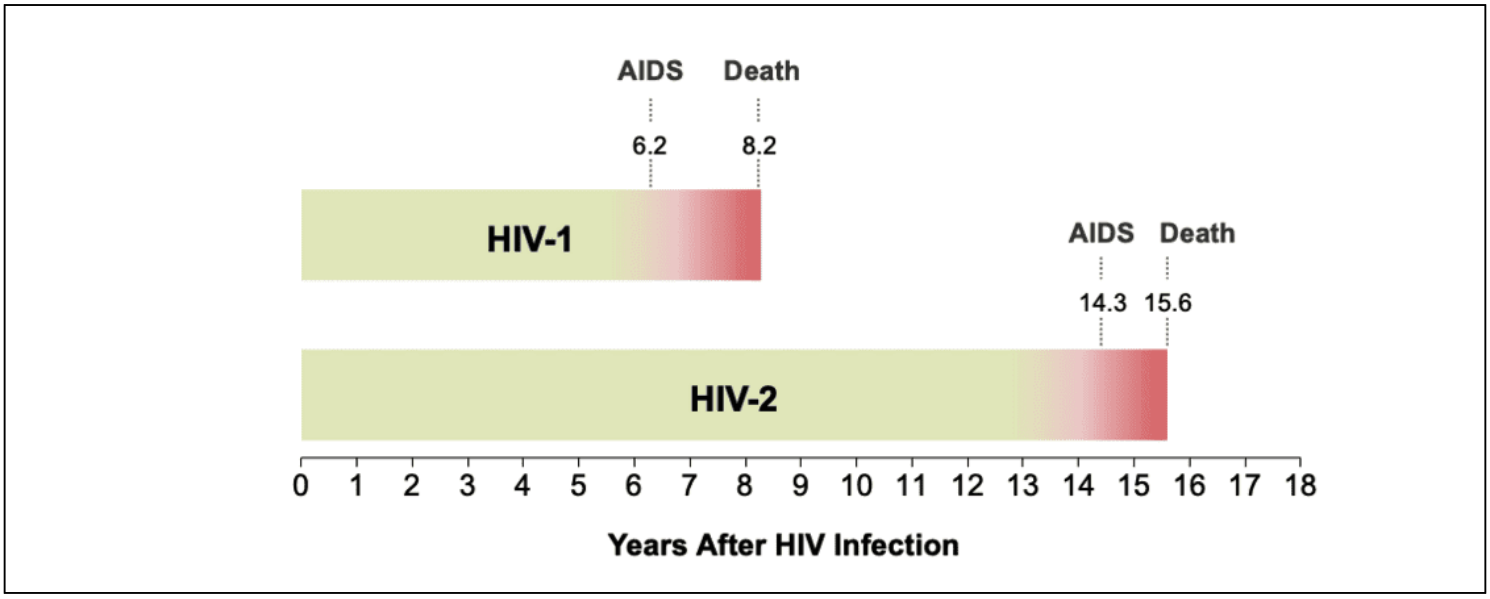


Figure 3 2018 CDC AHPL Recommended Laboratory HIV Testing Algorithm for Serum or Plasma Specimens

Abbreviation: APHL = Association of Public Health Laboratories; NAT = nucleic acid test

Source: Centers for Disease Control and Prevention and Association of Public Health Laboratories. 2018 Quick reference guide: Recommended laboratory HIV testing algorithm for serum or plasma specimens. Published January 27, 2018.

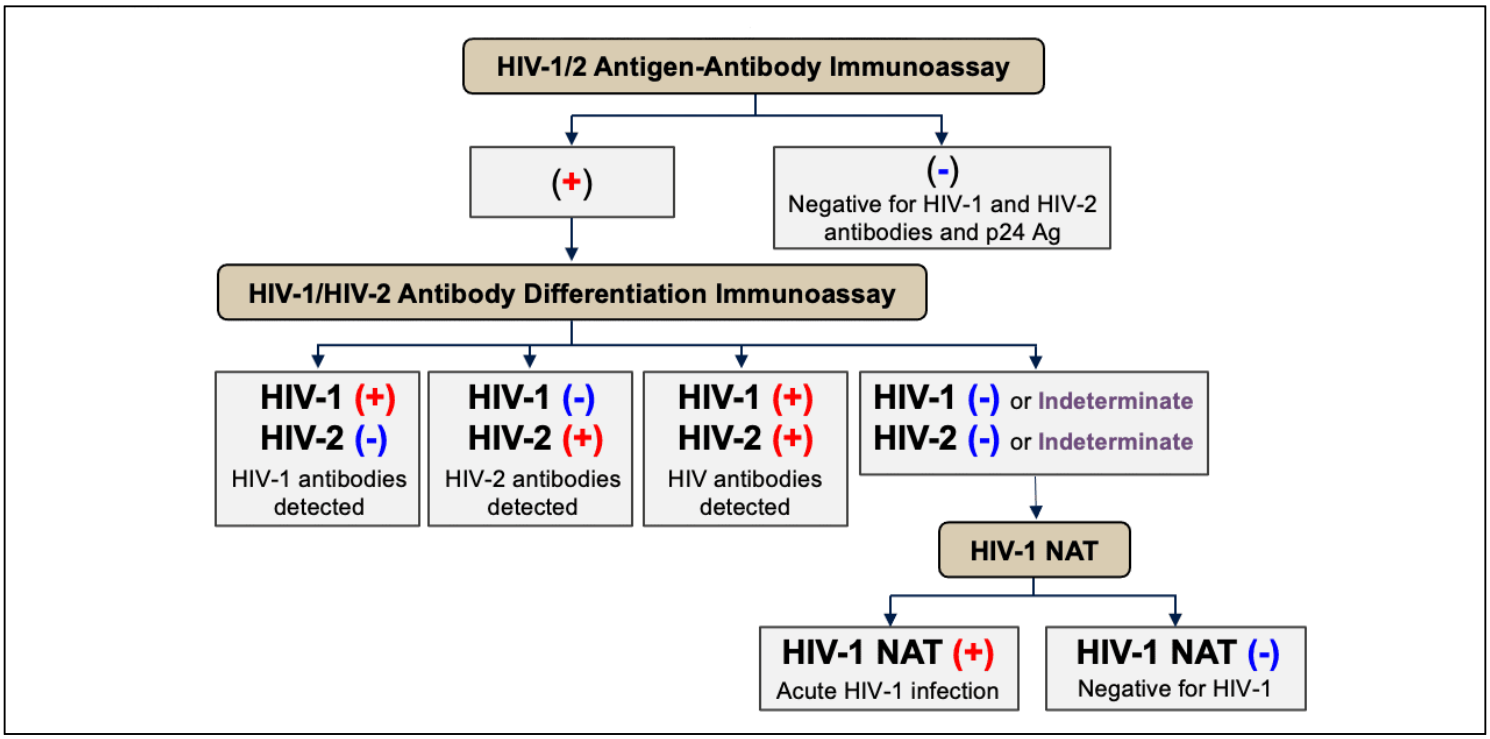


Figure 4 HIV-1 and HIV-2 Gene Products, Proteins, and Glycoproteins

Note the differences between some of the HIV-1 and HIV-2 proteins; these differences explains why HIV-1 Western blot tests fail to detect HIV-2 infection or give an indeterminate result.

Gene and Product	HIV-1	HIV-2
env		
Env Precursor	**gp160**	**gp140**
External Glycoprotein	gp120	gp105/125
Transmembrane Protein	gp41	gp36/41
pol		
Reverse Transcriptase	p66	p68
Reverse Transcriptase	p51	p53
Endonuclease	p31	p31/34
gag		
Gag Precursor	**p55**	**p57**
Matrix	p17	p17
Capsid	p24	p26
Nucleocapsid Precursor	p15	p15