HIV-2 Infection

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

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Background

Comparison of HIV-1 and HIV-2

Human immunodeficiency virus (HIV) is categorized into two main types: HIV-1 and HIV-2. Although HIV-1 and HIV-2 have the same routes of transmission and both can cause acquired immunodeficiency syndrome (AIDS), important differences exist between the viruses in terms of epidemiology, natural history, diagnosis, and management.\[1,2\] Compared with individuals with HIV-1, persons with HIV-2 typically have attenuated clinical progression and lower rates of sexual and perinatal HIV transmission.\[3,4,5,6\] Significant differences exist in the antiretroviral management of HIV-1 and HIV-2.\[2\] Clinicians should become familiar with the differences between these HIV infections and maintain a high index of suspicion of HIV-2 in persons from regions where HIV-2 circulates, individuals can acquire HIV-1, HIV-2, or both HIV-1 and HIV-2.\[7\]
HIV-2 Epidemiology

HIV-2 Globally

The World Health Organization (WHO) estimates that 39 million people are living with HIV worldwide at the end of 2022, but current HIV-2 specific prevalence estimates are lacking. [8] 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, 9, 10, 11] Most persons infected with HIV-2 reside in West Africa, or in countries that have strong colonial or socioeconomic ties with West Africa, most notably France, Spain, and Portugal; HIV-2 has 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 approximately the last two decades, HIV-2 prevalence has declined in several West African countries, but reasons for this are unclear. [12]

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. [13] The vast majority of persons diagnosed with HIV-2 in the United States have emigrated from an HIV-2 endemic region, or had exposure to a person from an HIV-2 endemic region. [14] In addition, most of the earlier reported small number of cases of HIV-2 in the United States have been clustered in the northeast. [14, 15] More recently, the Centers for Disease Control and Prevention (CDC) analyzed data from the National HIV Surveillance System (NHSS) that included data on persons diagnosed with HIV-1, HIV-2, or both during 2010–2017 in the United States and 6 dependent areas. [16] Overall, this report concluded the diagnosis of HIV-2 was rare during this time period. [16] The image series below summarizes the major characteristics for persons in the report who were diagnosed with HIV-2 (Figure 1). [16]
Pathogenesis, Transmission, and Natural History of HIV-2

Pathogenesis of HIV-2

Relative to HIV-1, HIV-2 is less virulent and is characterized by lower plasma HIV RNA levels and slower decline in CD4 cell counts, longer time to the development of AIDS.\[2,12,17\] For example, in West Africa, investigators prospectively followed 133 persons with HIV-2 who were not receiving antiretroviral therapy from 1991 through 2009 and found HIV-2 RNA levels remained consistently low, with 36 to 42% of the HIV-2 RNA levels falling in the range of less than 100 copies/mL.\[18\] 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.\[18\]

In another longitudinal follow-up study in West Africa, investigators have shown 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).\[19\] The exact reasons for the differences in pathogenicity of HIV-1 and HIV-2 remain incompletely defined, but both intrinsic viral factors and innate and adaptive immunity are likely to play important roles.\[17\]

Transmission of HIV-2

In natural history perinatal studies conducted in West Africa (in the pre-antiretroviral era), the rate of perinatal transmission of HIV-2 was significantly lower than for HIV-1, typically less than 5% versus approximately 25%.\[6,20\] Sexual transmission and genital shedding is also less efficient in persons with HIV-2.\[5,21,22,23\]

Acquisition of HIV-2 does not provide protection against infection with HIV-1.\[24\]

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% (approximately) of individuals have dual infection, though the proportion of persons with HIV-2 monoinfection has decreased as the prevalence of HIV-2 has decreased.\[17,25\] Unfortunately, initial infection with HIV-2 does not appear to protect against subsequent HIV-1 acquisition, as was initially reported in a cohort of female sex workers in Dakar, Senegal.\[24,26,27\] One study suggested that persons with both HIV-1 and HIV-2 have slower disease progression and delayed death when compared with those who have HIV-1 alone, with the greatest benefit occurring when infection with HIV-2 precedes HIV-1 infection.\[25\]

Other studies, including a robust meta-analysis, found no survival benefit in persons dually infected with HIV-1 and HIV-2 when compared with persons who have HIV-1 alone.\[26,28\]
2014 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 was not part of the 2008 case definition.[29] 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)
- Laboratory results 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 an HIV-1 NAT and an HIV-2 NAT are positive.[29]
Diagnostic Testing for HIV-2

Approach to Diagnostic Testing for HIV-2

In CDC screening guidelines for HIV issued prior to 2014, specific HIV-2 testing was recommended only for persons with known HIV-2 risk factors.\textsuperscript{30} With this older HIV testing algorithm, which utilized HIV enzyme immunoassay (EIA) as the screening test and HIV-1 Western blot as the confirmatory test, the diagnosis of HIV-2 was often missed because HIV EIA testing detects both HIV-1 and HIV-2, but does not distinguish between them; in addition, the traditional HIV-1 Western blot fails to detect HIV-2 (or it indicates an indeterminate or false positive result for HIV-1).\textsuperscript{14, 30, 31} It is important to note that HIV-1 RNA and DNA assays do not reliably detect HIV-2.\textsuperscript{1, 31} Use of 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 (\textit{Figure 3}).\textsuperscript{31, 32} In some circumstances, such as an indeterminate HIV-2 test on the HIV-1/HIV-2 differentiation immunoassay, HIV-2 NAT diagnostic testing is recommended.\textsuperscript{33} Diagnostic HIV-2 qualitative testing is now available for diagnostic purposes through two laboratories: University of Washington Laboratory Medicine (HIV-2 DNA/RNA Qualitative) and the New York State Department of Health (HIV-2 Qualitative RNA Detection). 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 diagnostic test.\textsuperscript{34, 35, 36}

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.\textsuperscript{30} 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).
- **Point-of-Care Tests:** Several point-of-care HIV tests are FDA-approved for the detection of HIV-2, including the OraQuick Advance Rapid HIV-1/2 Antibody Test, Clearview HIV 1/2 STAT-PAK, Clearview COMPLETE HIV 1/2, INSTI HIV-1/HIV-2 Rapid Antibody Test, Architect HIV Ag/Ab Combo Assay, and Alere Determine HIV-1/2 Ag/Ab. These point-of-care tests do not distinguish HIV-1 from HIV-2 infection.
- **Differentiation Assays for HIV-1/HIV-2:** In the United States, the Geenius HIV 1/2 Supplemental Assay, and the BioPlex 2200 HIV Ag-Ab are FDA-approved for differentiating HIV-1 from HIV-2 infection.\textsuperscript{37, 38, 39} The Geenius test can detect four antibodies to HIV-1 (p31, gp160, p24, and gp41) and two antibodies to HIV-2 (gp36 and gp140) (\textit{Figure 4}).\textsuperscript{37, 40} The CDC recommended algorithm for HIV testing utilizes the HIV-1/HIV-2 differentiation assay as the second step in the algorithm.\textsuperscript{31} The CDC has issued a technical update on HIV-1/2 differentiation assays that provide guidance for three results that may occur in the Geenius assay that were not previously seen with the Multipost HIV differentiation assay: HIV-2 positive with HIV-1 cross reactivity, HIV-2 indeterminate, and HIV indeterminate.\textsuperscript{33} If the result is HIV-2 positive with HIV-1 cross reactivity, the CDC recommends considering this result as positive for HIV-2 infection. For specimens with a result of HIV-2 indeterminate or HIV indeterminate results, additional testing is required (often including HIV-2 nucleic acid testing) and guidance in the technical update should be followed carefully; ideally, expert consultation is obtained in this setting.
- **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 absence of Env bands (gp160, gp120, or gp41) (\textit{Figure 5}).\textsuperscript{1, 15} The Western blot pattern in persons with HIV-2 occurs because HIV-1 and HIV-2 have 60% similarity in the regions encoding \textit{gag} and \textit{pol}, compared with only 30 to 40% similarity in the region encoding \textit{env}.\textsuperscript{41} 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.\textsuperscript{1}
• **Qualitative Plasma HIV-2 RNA**: An HIV-2 nucleic acid test is typically utilized to confirm a positive HIV-2 differentiation assay or to provide additional information in the setting of an indeterminate result on the HIV Geenius differentiation assay. A positive qualitative HIV-2 RNA will confirm 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.[34, 35, 36] To address this issue, investigators have developed a qualitative assay that detects HIV-2 total nucleic acid in patient peripheral blood mononuclear cells; this assay can detect HIV-2 DNA and RNA in persons with HIV-2 who have undetectable plasma HIV-2 RNA levels.[42]

**Quantitative HIV-2 RNA Testing**

The HIV-1 nucleic acid amplification tests (NAAT) do not reliably detect or quantitate HIV-2. Quantitative 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).[2]
Antiretroviral Susceptibility and Resistance

The following summarizes what is known related to HIV-2 susceptibility and resistance to medications in specific antiretroviral medication classes.

**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, may have less activity and a lower genetic barrier of resistance to HIV-2 than to HIV-1. 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. Studies have produced conflicting results regarding the frequency of the K65R mutation in HIV-2 and its impact on susceptibility to tenofovir and abacavir. 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.

**Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs)**

Multiple studies have shown that HIV-2 has intrinsic resistance to non-nucleoside reverse transcriptase inhibitor (NNRTI) drugs. This intrinsic resistance occurs because the Y181I and Y188L substitutions are natural polymorphisms present in HIV-2 strains and 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 the long-acting injectable rilpivirine as part of the injectable cabotegravir and rilpivirine combination.

**Integrase Strand Transfer Inhibitors (INSTIs)**

Accumulating evidence suggests INSTIs usually have potent activity against HIV-2. Dolutegravir, bictegravir, and cabotegravir have in vitro activity against HIV-2. A recent, small, single arm, clinical trial from Portugal showed that dolutegravir when paired with 2 NRTIs was effective treatment for treatment-naive persons with HIV-2 and another small study showed modest efficacy with dolutegravir in persons with HIV-2 who had resistance to first-generation INSTI. Elvitegravir has activity against HIV-2 and, as in HIV-1, there is a high degree of cross-resistance between raltegravir and elvitegravir. As seen with HIV-1, raltegravir and elvitegravir have a relatively low barrier to resistance to HIV-2, perhaps even lower with HIV-2 than with HIV-1 due to intrinsic polymorphisms at secondary integrase sites. The resistance that affects susceptibility of HIV-2 to INSTIs shares some similarities with those of HIV-1, though some key differences exist. For example, the integrase mutations N155H or Q148R confer resistance to raltegravir in both HIV-2 and HIV-1, but for HIV-2 the Y143 pathway requires secondary mutations to cause significant resistance (contrary to what is observed with HIV-1). 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, which is a region in the integrase C-terminal domain. This insertion mutation results in high-level resistance to elvitegravir and raltegravir and moderate-level resistance to dolutegravir. 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).

**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. Investigators have identified four residues at amino acid positions—32, 47, 76, and
82—in the protease binding cleft that differ between HIV-2 and HIV-1 and predict protease inhibitor sensitivity; changes at these four amino acids can confer class-wide resistance to PIs.[66] 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 are required for HIV-2 to become resistant to PIs as compared to HIV-1. 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, lopinavir, or saquinavir, 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.[67,68]
- **Post-Attachment Inhibitors**: Limited in vitro data shows that ibalizumab is active against HIV-2.[69]
- **CCR5 Inhibitor**: 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.[1,2,70,71]
- **Fusion Inhibitors**: In vitro data have shown HIV-2 has intrinsic resistance to the fusion inhibitor, enfuvirtide.[48] Resistance to enfuvirtide correlates with genetic diversity at the target regions for the drug, namely the HR1 domain of the viral gp41 region.[72]

Capsid Inhibitors

Limited in vitro data shows 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.[68,73] More data are needed on lenacapavir and HIV-2.
HIV-2 Treatment Studies

Clinical Trials for the Treatment of HIV-2

There are limited data from clinical trials that inform guidance on the optimal timing or regimen for initial antiretroviral therapy of persons with HIV-2, which have included three small, single arm trials of INSTI-based (raltegravir, elvitegravir, and dolutegravir) treatment for initial antiretroviral therapy.[4, 57, 74] In addition, a randomized controlled trial comparing tenofovir DF-emtricitabine plus raltegravir versus tenofovir DF-emtricitabine plus lopinavir- ritonavir has been completed but the results have not yet been reported. There are no clinical data on the use of bictegravir-tenofovir alafenamide-emtricitabine for the treatment of HIV-2, but in vitro data suggest that HIV-2 is highly sensitive to bictegravir.[53] The lack of data on HIV-2 treatment is 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 less of a priority than for those with HIV-1.[1]
HIV-2 Treatment Recommendations

Timing of Initiating Antiretroviral Therapy with HIV-2

Although individuals with HIV-2 generally have a slower disease progression than persons with HIV-1, they generally have a less robust CD4 count increase in response to antiretroviral treatment.\[^{75,76,77,78}\] 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 ART 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

Based on experience with the treatment of HIV-1, a three-drug antiretroviral regimen should be used to treat HIV-2 in order to maintain viral suppression and to avoid 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.\[^{79,80}\]

- **Recommendation**: The Adult and Adolescent ART Guidelines recommend initial treatment of persons with HIV-2 with two NRTIs in combination with an INSTI (bictegravir, dolutegravir, elvitegravir, or raltegravir).\[^{2}\] The alternative regimen is two NRTIs plus a boosted protease inhibitor (darunavir or lopinavir) active against HIV-2.\[^{2,78,81}\] 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.\[^{2}\]

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

Individuals with HIV-1 and HIV-2 coinfection should undergo baseline genotypic resistance testing for HIV-1; resistance testing for HIV-2 is not commercially available in the United States.\[^{2,81}\] In this setting, if possible, monitoring of both HIV-1 and HIV-2 plasma RNA levels should be performed.\[^{82,83}\]

- **Recommendation**: The Adult and Adolescent ART Guidelines recommend treatment of persons with HIV-1 and HIV-2 coinfection with 2 NRTIs in combination with an INSTI, even if the baseline HIV-2 plasma viral load is low or undetectable.\[^{2}\] All of the preferred regimens in the Adult and Adolescent ART Guidelines for the treatment of HIV-1 have good activity against both HIV-1 and HIV-2.\[^{2}\] The following medication 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.\[^{2}\]

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 amplification tests (NAAT) do not reliably detect or quantitate HIV-2. Traditionally, because of limited availability of HIV-2 RNA assays, response to antiretroviral therapy in most individuals with HIV-2 has been gauged only by regular clinical monitoring and repeated CD4 cell count monitoring. Quantitative HIV-2 RNA viral load assays for monitoring response to therapy are now available through the University of Washington Laboratory Medicine (HIV-2 RNA Quantitation) and the New York State Department of Health (HIV-2 Quantification).\[^{2}\] Since several laboratories are now capable of...
performing quantitative HIV-2 RNA levels, the management of persons with HIV-2 on antiretroviral therapy should include routine monitoring of quantitative HIV-2 levels, similar to what is done for persons with HIV-1. Ongoing CD4 count monitoring is 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 has shown that HIV-2 is more pathogenic than previously thought and most persons with HIV-2 will develop AIDS within 15 years.
- Infection with HIV-2 should be considered in persons with risk factors for HIV-2 acquisition and in persons with a clinical illness (such as an AIDS-associated opportunistic infection) that suggests HIV infection but in whom testing for HIV-1 is negative.
- Use of the CDC and 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 with the HIV differentiation assay used in the second step. A positive HIV-2 qualitative RNA or DNA confirms infection, but HIV-2 RNA alone is not reliable to rule out infection since approximately 30 to 40% of persons with HIV-2 have undetectable HIV-2 RNA levels.
- 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 of HIV-2 infection is two NRTIs plus an INSTI. The alternative regimen is two 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.
- Laboratory diagnostics for HIV-2 are available through two laboratories: University of Washington Laboratory Medicine (HIV-2 DNA/RNA Qualitative and RNA Quantitative) and the New York State Department of Health (HIV-2 RNA Qualitative and Quantitative).


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Figures

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


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.

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

The Geenius HIV-1/HIV-2 Supplemental Assay is a single-use immunochromatographic test that utilizes multiple recombinant or synthetic peptides to detect HIV-1 and HIV-2. Note the HIV-2 antibodies detected include gp36 and gp140 (marked by yellow color).

**Figure 5 HIV-1 and HIV-2 Gene Products, Proteins, and Glycoproteins**

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

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