

# HIV/AIDS

## AN UPDATE FOR HOME HEALTHCARE CLINICIANS

Human immunodeficiency virus (HIV) infection, once fatal, has become a chronic disease that can be treated and well-managed. Antiretroviral treatment (ART) can result in undetectable levels of HIV in the bloodstream. The risk factors, transmission, diagnosis, treatment, acute HIV infection, potential opportunistic infections, and malignancies should be understood by all those caring for persons living with HIV. Preventive treatment is possible with preexposure and postexposure prophylaxis regimens. Home healthcare providers can assist individuals to adhere to medication regimens, monitor efficacy of treatment, recognize complications of HIV and side effects of ART. Most importantly, they can support, educate, and counsel persons living with HIV and their families.

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**I**t has been almost 40 years since the first cases of human immunodeficiency virus and acquired immune deficiency syndrome (HIV/AIDS) were reported in the United States. Once a fatal disease, HIV infection has become a chronic infection that can be treated and well-managed. There are three stages of the disease: acute HIV infection, chronic HIV, and AIDS. Before the advent of antiretroviral therapy (ART), HIV infection almost always resulted in opportunistic infections and early death from AIDS. Now for persons living with HIV infection, one pill per day can suppress the virus and allow maximum quality of life (May et al., 2014).

### Epidemiology

An estimated 1.1 million people in the United States were living with HIV at the end of 2015, and 522,283 of those people were living with diagnosed HIV infections that had been, or were currently, classified as stage 3 (AIDS). In 2014, the number of new HIV infections in the United States was 37,600, with an estimated 10% annual decline in HIV infections from 2010 to 2014 (Singh et al., 2017). The highest rate of transmission of HIV infection in the United States occurs in male-to-male sexual contact, accounting for 70% of new HIV infections. People who inject drugs account for 9% of HIV diagnoses, and 8,600 cases are due to heterosexual contact with someone who had or was at high risk for HIV. African Americans are disproportionally diagnosed with HIV and have the highest rate of new-onset HIV/AIDS (Centers for Disease Control and Prevention [CDC], 2018a).

Because ART has been such a highly successful intervention, persons with HIV live longer and the percentage of older individuals with HIV has increased. It is estimated that by 2020, more than 50% of persons living with HIV infection will be aged 50 years or older (Brooks et al., 2012). This will bring new challenges due to comorbidities that commonly arise with age.

### Etiology and Pathophysiology

HIV is a retrovirus that is within the genus *Lentivirus*. Lentiviruses have a long latency period and a slowly progressing disease course. There are two major strains of HIV; HIV-1, which is the type that

infects most individuals in the United States, and HIV-2 is the strain most common in West Africa. A retrovirus has RNA as its genetic material and comes equipped with its own enzyme, called reverse transcriptase, that can convert its RNA into DNA. Reverse transcriptase allows the virus to integrate into DNA-based host cells and manufacture more viruses (Naif, 2013).

HIV targets cells that express the CD4 receptor and the chemokine receptors CCR5 or CXCR4. These include T lymphocytes (called T helper cells or CD4+ T cells); monocytes and macrophages in the lymph nodes, spleen, bone marrow, lung and brain; and dendritic cells in lymphoepithelial tissue in the vagina, rectum, and tonsils (Lucas & Nelson, 2015).

The hallmark of HIV is a progressive depletion of CD4+ T cells, which are integral to both cell-mediated and antibody-mediated immune mechanisms. Attacking these cells allows the virus to annihilate both immune mechanisms of the body. Macrophages act as reservoirs of HIV allowing for viral persistence. Macrophages are believed to serve as vehicles for dissemination of HIV between different tissues of the body (Ciborowski & Gendelman, 2006). Mucous membranes throughout the body are rich in macrophages. HIV sequentially attaches to the CD4 surface receptor and CCR5 or CXCR4 (or fusin) chemokine coreceptors on host cells (Chun & Fauci, 2012). The virus then uses reverse transcriptase to change its RNA into DNA and inserts its DNA into the host cell DNA using the enzyme integrase. Integrase is the HIV enzyme that incorporates the genetic directions for synthesis of viruses into the host cell genome (Craigie, 2012). Protease is an HIV enzyme that assists in the assembly of protein components to construct new viruses (Yang et al., 2012).

As the number of viruses increases in the bloodstream, more host cells are attacked, and the number of CD4 cells in the blood decreases. The viral load increases as the CD4+ cell count gradually drops, weakening the body's immune response usually over a number of years. The CD4 cell count is used to monitor the course of the disease. If the CD4+ cell count drops below 200 cells/mm<sup>3</sup>, severe immune dysfunction occurs and

The viral load increases as the CD4+ cell count gradually drops, weakening the body's immune response usually over a number of years.

a diagnosis of AIDS is made. If left untreated, people at this stage of the disease usually succumb to opportunistic infections (Naif, 2013).

### Most Common Risk Factors/Transmission

Globally, unprotected heterosexual activity is the most common mode of transmission among persons of reproductive age. However, there are a number of behaviors that place an individual at high risk for HIV infection. Anal sex is the riskiest type of sex for transmitting HIV, particularly if it occurs without a condom or use of medicines to prevent or treat HIV. During anal sex, the receptive partner is at 13 times greater risk than the insertive partner. However, it is possible for either partner to contract HIV through anal sex from body fluids (blood, semen, preseminal fluid, or rectal fluids) of a person who has HIV. Vaginal sex, insertion of the penis into the vagina, can transmit the virus if either partner has HIV. The receptive individual is at higher risk than the insertive partner; however, both partners are at risk (CDC, 2018b).

Though the risk of HIV transmission through oral sex is low, several factors may increase that risk, including: open sores in the mouth or vagina or on the penis, bleeding gums, oral contact with menstrual blood, and the presence of other sexually transmitted diseases (CDC, 2018b). Injection drug use is another high-risk behavior if people share needles or syringes. Occupational exposure of healthcare workers who sustain needle sticks involving HIV-infected blood at work have a 0.23% risk of becoming infected. Risk of exposure due to splashes with body fluids is thought to be near zero regardless of blood in the fluid (CDC, 2018b). Mother-to-fetal transmission of HIV is another route of infection, as well as mother-to-infant during breastfeeding. Another possible source of HIV is contaminated blood or blood product transfusions, and contaminated organ or tissue transplants, where screening for bloodborne viruses is inadequate or unavailable (CDC, 2018b).

### Clinical Manifestations and Diagnostic Testing

Acute HIV infection causes a mononucleosis-like syndrome that consists of fever, headache, pharyngitis, lymphadenopathy, and myalgia within 28 days of contraction of the virus. The acute HIV syndrome lasts a couple of weeks, and then resolves. During the acute infection, many individuals do not seek healthcare due to the nonspecific

nature of the symptoms (Burgess & Kasten, 2013). After resolution of the acute syndrome, the infection enters a time of clinical latency when there are no particular symptoms of HIV infection. This stage is referred to as chronic HIV infection. If the infection is untreated, increasing levels of viremia and decreasing numbers of CD4 cells occur usually over a number of years (Naif, 2013).

Screening for HIV is recommended for patients in all healthcare settings after they have been notified, unless they chose to “opt out” (CDC, 2018a). Separate written consent is not necessary. Persons at high risk for HIV should be tested at least annually (CDC, 2018). Diagnostic laboratory testing includes CD4 count, HIV viral load, and HIV drug-resistance testing. The CD4 count is the most accurate measurement of the degree of immune system impairment. Normal CD4 counts vary from approximately 800 to 1,200 cells/mm<sup>3</sup>. Severe immunologic impairment occurs at a CD4 cell count of 200 cells or less (U.S. Department of Health and Human Services [DHHS], 2017). The HIV RNA blood test (also called viral load) is the most accurate measure of the number of viruses in the bloodstream. The results of a viral load test are described as the number of copies of HIV RNA in a milliliter of blood. The virus is usually detectable in the bloodstream within 4 to 11 days of contraction (Sell, 2013).

The HIV RNA blood test should be initiated prior to ART and again after ART is initiated 2 to 8 weeks later. If the viral load remains detectable, repeat every 4 to 8 weeks until the viral load <200 copies/mL. Viral load testing can be repeated every 3 to 6 months thereafter. Viral load indicates the severity of the disease and susceptibility to AIDS. If the viral load falls to undetectable levels, the patient can still transmit the virus to others. The virus is still present in the body, however, it is not apparent using current routine laboratory testing procedures (DHHS, 2017).

The HIV drug-resistance test is also done to check if the virus has a mutation that endows it with resistance to specific types of ART. This test can guide choice of treatment regimen. The HIV drug-resistance test can be done if the viral load is above 500 copies/mL. (DHHS, 2016). Clinicians also test for HLA B\*5701 that detects a gene that predisposes to an allergic reaction to the specific ART medication abacavir. This test also guides the choice of treatment (DHHS, 2011). The HIV enters cells by attachment to the CD4 receptor followed by binding to either the CCR5 or CXCR4 receptors.

**TABLE 1. HIV Antiretroviral Drugs**

NRTIs	NNRTIs	Fusion Inhibitors	CCR5 Antagonists	Protease Inhibitors	INSTIs
Emtriva® (emtricitabine)	Edurant® (rilpivirine)	Fuzeon® (enfuvirtide)	Selzentry® (maraviroc)	Aptivus® (tipranavir)	Isentress® (raltegravir)
Epivir® (3TC, lamivudine)	Intelence® (etravirine)			Crixivan® (indinavir)	Tivicay® (dolutegravir)
Retrovir® (AZT, zidovudine)	Rescriptor® (delavirdine)			Invirase® (saquinavir)	Vitekta® (elvitegravir) (Note: Vitekta must be “boosted” with a pharmacokinetic enhancer, either Tybost® or Norvir®.)
Videx-EC® (ddI, didanosine)	Sustiva® (efavirenz)			Kaletra® (lopinavir + ritonavir combined in one tablet)	
Viread® (tenofovir DF)	Viramune® (nevirapine)			Lexiva® (fosamprenavir)	
Zerit® (d4T, stavudine)				Norvir® (ritonavir)	
Ziagen® (abacavir)					
				Prezista® (darunavir)	
				Reyataz® (atazanavir)	
				Viracept® (nelfinavir)	

*Note.* Several of the NRTI drugs may be combined into one tablet to make it easier to take the medications. These drugs are known as fixed-dose combinations:

- Combivir® (Retrovir + Epivir)
- Descovy® (Tenofovir alafenamide + Emtriva)
- Epzicom® (Epivir + Ziagen)
- Trizivir® (Retrovir + Epivir + Ziagen)
- Truvada® (Viread + Emtriva)

Adapted from:

U.S. Department of Health and Human Services. (2018, January 24). *HIV treatment*. What to start: Choosing an HIV regimen. Retrieved from <https://aidsinfo.nih.gov/understanding-hiv-aids/fact-sheets/21/53/what-to-start--choosing-an-hiv-regimen>

U.S. Department of Health and Human Services. (2018, February 20). *HIV treatment*. FDA-approved HIV medicines. Retrieved from <https://aidsinfo.nih.gov/understanding-hiv-aids/fact-sheets/21/58/fda-approved-hiv-medicines>

U.S. Department of Health and Human Services. (2017, October 17). *Guidelines for the use of anti-retroviral agents in adults and adolescents living with HIV*. Retrieved from <https://aidsinfo.nih.gov/guidelines/html/1/adult-and-adolescent-arv-guidelines/11/what-to-start>

A blood test can determine the specific coreceptor affinity (i.e., CCR5, CXCR4, or both) of the patient's virus (DHHS, 2017). To detect HIV antibodies, the enzyme-linked immunosorbent assay/Western Blot test is used. The HIV antibody takes between 2 weeks and 6 months to appear in the bloodstream (U.S. Department of Veterans Affairs [VA], 2018). Therefore, an individual can have virus in the bloodstream before the HIV antibody appears in the bloodstream. The time between contraction of the virus and antibody appearance is called the window period. When the antibody appears in the bloodstream (called seroconversion), the individual changes from seronegative to seropositive status.

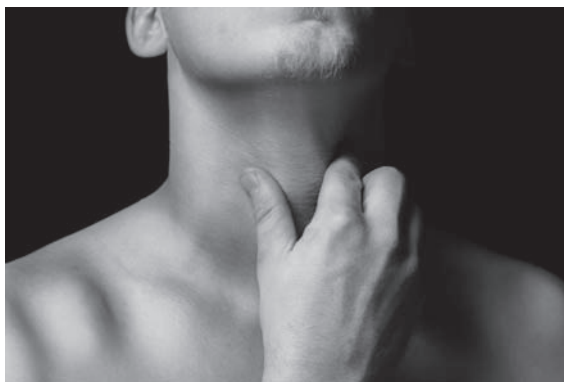
Because HIV often occurs due to unprotected sexual activity, clinicians also test for STDs,

mainly syphilis, gonorrhea, and chlamydia. Hepatitis B and hepatitis C blood testing are also recommended. The individual with HIV is highly susceptible to tuberculosis, so testing in the form of a PPD is done or a blood test called interferon-gamma release assay (HIV.gov, 2017).

## Antiretroviral Treatment

Early initiation of ART is recommended. Recent studies have shown that very early initiation of ART can preserve immune function and reduce complications of HIV-1 infection (The TEMPRANO ANRS 12136 Study Group, 2015). Medicines for HIV are grouped into six drug classes according to where they act in the HIV life cycle (Table 1):

- Nonnucleoside reverse transcriptase inhibitors (NNRTIs)



Acute HIV infection causes a mononucleosis-like syndrome that consists of fever, headache, pharyngitis, lymphadenopathy, and myalgia within 28 days of contraction of the virus.

- Nucleoside reverse transcriptase inhibitors (NRTIs)
- Protease inhibitors (PIs)
- Fusion inhibitors
- CCR5 antagonists (CCR5s) (also called entry inhibitors)
- Integrase strand transfer inhibitors (INSTIs)

Fusion inhibitors act by blocking HIV from entering the host cell. Chemokine coreceptor 5 (CCR5) antagonists are also entry inhibitors that block HIV from latching on to the CCR5 receptor and gaining entry into the host cell (VA, 2018). Both NRTIs and NNRTIs act by blocking the HIV enzyme reverse transcriptase that prevents HIV from changing its RNA into DNA. Therefore, these drugs block HIV from replicating (DHHS 2017). INSTIs block HIV from inserting its viral DNA into the host cell DNA. This in turn blocks HIV from replicating itself inside the host cell. PIs block HIV from synthesizing the pieces that are needed to synthesize more HIV particles in the host cell. Many PIs need another drug to boost their effect. A commonly used booster drug is cobicistat (Tybost) (VA). INSTI-based regimens are recommended as initial therapy for most people with HIV. In large clinical trials and in clinical practice, INSTI-based regimens have achieved high rates of virologic suppression and often have greater tolerability than PI- or NNRTI-based regimens. Many of the regimens below are available as a coformulated single pill (DHHS, 2018) (see Supplemental

Digital Content 1, available at <http://links.lww.com/HHN/A55>).

### Monitoring Efficacy of ART

The patient's response to ART is monitored by sequential measurements of viral load and CD4 cell counts. Optimal viral suppression is indicated by a viral load below the level of detection (usually less than 20 to 75 copies of the virus per mL, depending on the assay). Undetectable levels are typically achieved in 8 to 24 weeks (DHHS, 2017). Although levels of virus in the bloodstream can become undetectable, the patient can still transmit the virus to others. HIV can survive within reservoirs, such as macrophages, for long periods of time and not be detected (Naif, 2013).

### Common Side Effects of ART

The overall benefits of viral suppression and improved immune function as a result of effective ART far outweigh the risks associated with the adverse effects of some ART agents. Newer ART agents/regimens are associated with fewer adverse effects than in the past (DHHS, 2017). Serious life-threatening events (e.g., hypersensitivity reaction) require immediate discontinuation and reinitiation of an alternative regimen. Toxicities that are not life-threatening can usually be managed by substituting another ART agent or by managing the adverse event with additional pharmacological or nonpharmacological interventions (DHHS, 2017).

Nausea and diarrhea are common side effects of ART agents, particularly the PIs. In some cases, these gastrointestinal effects dissipate with continued use (DHHS, 2017). Decreased bone mineral density has been observed with many ART regimens, particularly tenofovir disoproxil fumarate (Güerri-Fernández et al., 2018). Dyslipidemia and insulin resistance that increase cardiovascular disease risk are also common. Hypertriglyceridemia and high LDL levels are common effects of PIs; particularly lopinavir/ritonavir and fosamprenavir/ritonavir (Maggi et al., 2017). Lipoatrophy, wasting of subcutaneous fat, has been most common with NRTIs, such as stavudine and zidovudine (de Waal et al., 2013). Renal side effects that cause elevated creatinine and proteinuria have been frequently reported with use of tenofovir disoproxil fumarate (Kamkuemah et al., 2015). Hepatotoxicity has been found particularly with nevirapine and

jaundice is commonly seen with atazanavir (Price & Thio, 2010). NNRTIs are associated with skin rash and abacavir is notable for causing hypersensitivity in persons with class I MHC allele, HLA-B\*5701 (Chaponda & Pirmohamed, 2011). Finally, ART, particularly NRTIs and NNRTIs, have also been associated with neuropsychiatric problems, including depression, cognitive impairment, and sleep disturbances (Treisman & Soudry, 2016).

#### **Paradoxical Response to ART: Immune Reconstitution Inflammatory Syndrome**

After initiation of ART, immune restoration is not always a smooth transition to health for all patients. Starting ART may be initially accompanied by an aberrant inflammatory response termed immune reconstitution inflammatory syndrome (IRIS), wherein patients experience deterioration in response to ART, despite efficient control of HIV viral replication and no apparent drug toxicity. The hallmark of the syndrome is paradoxical worsening of an existing infection or disease process or appearance of a new infection/disease process soon after initiation of therapy (Sharma & Soneja, 2011). The most common forms of IRIS are associated with mycobacterial infections, fungi, cytomegalovirus, and herpes viruses. IRIS has been reported as occurring in 10% to 25% of persons living with HIV who initiate ART at very low CD4 counts (French, 2012). The majority of patients with IRIS have a self-limiting disease course, and ART is usually continued and treatment for the associated condition optimized. The overall mortality associated with IRIS is low; however, patients with central nervous system involvement with raised intracranial pressures in cryptococcal and tubercular meningitis, and respiratory failure due to acute respiratory distress syndrome have poor prognosis and require aggressive management including corticosteroids (Sharma & Soneja).

### **Complications of HIV**

#### **Opportunistic Infections Associated With HIV**

Prophylaxis against specific opportunistic infections is indicated for patients with substantial immunosuppression. With effective ART, immune function can be restored and prophylaxis can be discontinued. Prophylaxis against the opportunistic infections; *Pneumocystis jiroveci*, *Toxoplasma gondii*, and *Mycobacterium avium* should begin depending on CD4 counts (DHHS,

2018). Other common opportunistic infections associated with HIV are presented in Supplemental Digital Content 2 (available at <http://links.lww.com/HHN/A56>).

#### **Malignancies Associated With HIV**

AIDS-defining malignancies include Kaposi sarcoma, non-Hodgkin lymphoma, and invasive cervical cancer (National Cancer Institute, 2017). These types of cancer are decreasing in patients with HIV infection, whereas non-AIDS-defining cancers that include anal cancer, Hodgkin lymphoma, hepatocellular carcinoma, skin cancer, head and neck cancer, and lung cancer are increasing. Reasons for this change are unclear (Wang et al., 2014). Clinicians should perform routine malignancy screening and should be alert for symptoms in people living with HIV infection (see Supplemental Digital Content 3, available at <http://links.lww.com/HHN/A57>). A baseline anal Papanicolaou (Pap) test should be done in all patients with HIV. Women living with HIV should have a baseline Pap test, and if normal, a second one 6 months later. If the second is normal, annual Pap smears are recommended.

### **Prevention of HIV Infection**

#### **Preexposure and Postexposure**

##### **Prophylaxis of HIV**

HIV preexposure prophylaxis (PrEP) is the provision of antiretroviral medications to noninfected individuals at high risk for exposure to HIV (World Health Organization, 2015). In the largest study to date of PrEP use in clinical practice, 850 individuals did not contract HIV and demonstrated 92% medication adherence (Marcus et al., 2016). PrEP is a combination of tenofovir and emtricitabine in one pill (brand name Truvada). Persons who use PrEP must take the drug every day, use condoms, and follow up with their healthcare provider every 3 months.

Postexposure prophylaxis (PEP) is the use of antiretroviral drugs after a single high-risk event to prevent contraction of HIV. After the exposure to HIV, PEP must be started within 72 hours to be effective. It includes a 28-day course of triple ART that includes tenofovir and emtricitabine (Truvada) with raltegravir or dolutegravir (DHHS, 2016).

#### **AIDS**

The most advanced stage of HIV infection is AIDS, when the CD4 cell count is less than 200 cells/mL

and the individual commonly has an AIDS-defining condition. There are some specific opportunistic infections and malignancies that are AIDS-defining conditions (see Supplemental Digital Content 2 and 3, available at <http://links.lww.com/HHN/A56> and <http://links.lww.com/HHN/A57>, respectively). Without treatment, AIDS

Prophylaxis against specific opportunistic infections is indicated for patients with substantial immunosuppression.

is usually fatal; however, many people living with HIV never develop AIDS. With aggressive treatment, persons diagnosed with AIDS can regain their immune strength. According to a recent meta-analysis, most patients who receive ART will survive for >10 years after the onset of AIDS, whereas the majority of the patients who do not receive ART die within 2 years of the onset of AIDS (Poorolajal et al., 2016).

## Summary/Conclusion

HIV infection has become a manageable chronic disease that can allow those affected to have a maximum quality of life. Home healthcare clinicians will have patients living with HIV infection and should understand the disease in a comprehensive manner. HIV infection has an interesting history in this country, and remarkable stride have been made in treating the disease. The virus can be treated so that there are undetectable levels in the bloodstream. The risk factors, transmission, acute HIV infection, diagnosis, treatment, and potential opportunistic infections and malignancies should be understood by all those treating persons living with HIV and their families. Preventive treatment is possible with PrEP and PEP regimens. Antiretroviral treatment that involves combinations of drugs of different categories can be administered in a single pill. Home healthcare providers can assist individuals to adhere to medication regimens, monitor efficacy of treatment, recognize complications of HIV and side effects of ART. Most importantly, they can support, educate, and counsel persons living with HIV and their family. ■

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The author declares no conflicts of interest.

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## REFERENCES

- Brooks, J. T., Buchacz, K., Gebo, K. A., & Mermin, J. (2012). HIV infection and older Americans: The public health perspective. *American Journal of Public Health, 102*(8), 1516-1526.
- Burgess, M. J., & Kasten, M. J. (2013). Human immunodeficiency virus: What primary care clinicians need to know. *Mayo Clinic Proceedings, 88*(12), 1468-1474.
- Centers for Disease Control and Prevention. (2018a). *HIV/AIDS*. Retrieved from <https://www.cdc.gov/hiv/default.html>
- Centers for Disease Control and Prevention. (2018b). *HIV transmission*. Retrieved from <https://www.cdc.gov/hiv/basics/transmission.html>
- Chaponda, M., & Pirmohamed, M. (2011). Hypersensitivity reactions to HIV therapy. *British Journal of Clinical Pharmacology, 71*(5), 659-671.
- Chun, T. W., & Fauci, A. S. (2012). HIV reservoirs: Pathogenesis and obstacles to viral eradication and cure. *AIDS, 26*(10), 1261-1268.
- Ciborowski, P., & Gendelman, H. E. (2006). Human immunodeficiency virus-mononuclear phagocyte interactions: Emerging avenues of biomarker discovery, modes of viral persistence and disease pathogenesis. *Current HIV Research, 4*(3), 279-291.
- Craigie, R. (2012). The molecular biology of HIV integrase. *Future Virology, 7*(7), 679-686.
- de Waal, R., Cohen, K., & Maartens, G. (2013). Systematic review of antiretroviral-associated lipodystrophy: Lipodystrophy, but not central fat gain, is an antiretroviral adverse drug reaction. *PLoS One, 8*(5), e63623.
- French, M. A. (2012). Immune reconstitution inflammatory syndrome: Immune restoration disease 20 years on. *The Medical Journal of Australia, 196*(5), 18-21.
- Güerri-Fernández, R., Lerma-Chippirraz, E., Fernandez Marron, A., García-Giralt, N., Villar-García, J., Soldado-Folgado, J., ..., Knobel, H. (2018). Bone density, microarchitecture, and tissue quality after 1 year of treatment with tenofovir disoproxil fumarate. *AIDS, 32*(7), 913-920. doi:10.1097/QAD.0000000000001780
- HIV.gov. (2017). *Lab tests and why they are important*. Retrieved from <https://www.hiv.gov/hiv-basics/staying-in-hiv-care/provider-visits-and-lab-test/lab-tests-and-results>
- Kamukemah, M., Kaplan, R., Bekker, L. G., Little, F., & Myer, L. (2015). Renal impairment in HIV-infected patients initiating tenofovir-containing antiretroviral therapy regimens in a primary healthcare setting in South Africa. *Tropical Medicine & International Health, 20*(4), 518-526.
- Lucas, S., & Nelson, A. M. (2015). HIV and the spectrum of human disease. *The Journal of Pathology, 235*(2), 229-241.
- Maggi, P., Di Biagio, A., Rusconi, S., Cicalini, S., D'Abbraccio, M., d'Ettore, G., ..., Squillace, N. (2017). Cardiovascular risk and dyslipidemia among persons living with HIV: A review. *BioMed Central Infectious Disease, 17*, 551.
- Marcus, J. L., Volk, J. E., Pinder, J., Liu, A. Y., Bacon, O., Hare, C. B., & Cohen, S. E. (2016). Successful implementation of HIV pre-exposure prophylaxis: Lessons learned from three clinical settings. *Current HIV/AIDS Reports, 13*(2), 116-124.
- May, M. T., Gompels, M., Delpech, V., Porter, K., Orkin, C., Kegg, S., ..., Sabin, C.; UK Collaborative HIV Cohort (UK CHIC) Study. (2014). Impact on life expectancy of HIV-1 positive individuals of CD4+ cell count and viral load response to antiretroviral therapy. *AIDS, 28*(8), 1193-1202.
- Naif, H. M. (2013). Pathogenesis of HIV infection. *Infectious Disease Reports, 5*(Suppl. 1), e6. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3892619/>
- National Cancer Institute. (2017). *HIV infection and cancer risk*. Retrieved from <https://www.cancer.gov/about-cancer/causes-prevention/risk/infectious-agents/hiv-fact-sheet>

- Poorolajal, J., Hooshmand, E., Mahjub, H., Esmailnasab, N., & Jenabi, E. (2016). Survival rate of AIDS disease and mortality in HIV-infected patients: A meta-analysis. *Public Health*, 139, 3-12.
- Price, J. C., & Thio, C. L. (2010). Liver disease in the HIV-infected individual. *Clinical Gastroenterology and Hepatology*, 8(12), 1002-1012.
- Sell, J. K. (2013). Management of human immunodeficiency virus in primary care. *Primary Care*, 40(3), 589-617.
- Sharma, S. K., & Soneja, M. (2011). HIV & immune reconstitution inflammatory syndrome (IRIS). *The Indian Journal of Medical Research*, 134(6), 866-877.
- Singh, S., Song, R., Johnson, A., McCray, E., & Hall, I. (2017). *HIV incidence, prevalence, and undiagnosed infections in men who have sex with men*. Retrieved from [http://www.viraled.com/modules/info/files/files\\_58b45f1a3e343.pdf](http://www.viraled.com/modules/info/files/files_58b45f1a3e343.pdf)
- The TEMPRANO ANRS 12136 Study Group. (2015). A trial of early antiretrovirals and isoniazid preventive therapy in Africa. *The New England Journal of Medicine*, 373(9), 808-822.
- Treisman, G. J., & Soudry, O. (2016). Neuropsychiatric effects of HIV antiviral medications. *Drug Safety*, 39(10), 945-957.
- U.S. Department of Health and Human Services. (2011). *Guidelines for the use of antiretroviral agents in adults and adolescents living with HIV laboratory testing: HLA-B\* 5701 screening*. Retrieved from <https://aidsinfo.nih.gov/guidelines/html/1/adult-and-adolescent-arv/6/drug-resistance-testing>
- U.S. Department of Health and Human Services. (2016). *Guidelines for the use of antiretroviral agents in adults and adolescents living with HIV laboratory testing: HIV drug resistance testing*. Retrieved from <https://aidsinfo.nih.gov/guidelines/html/1/adult-and-adolescent-arv/6/drug-resistance-testing>
- U.S. Department of Health and Human Services. (2017). *Guidelines for the use of antiretroviral agents in adults and adolescents living with HIV*. Laboratory testing for initial assessment and monitoring of patients with HIV receiving antiretroviral therapy. Retrieved from <https://aidsinfo.nih.gov/guidelines/html/1/adult-and-adolescent-arv/3/tests-for-initial-assessment-and-follow-up>
- U.S. Department of Health and Human Services. (2018). *HIV treatment*. FDA-approved HIV medicines. Retrieved from <https://aidsinfo.nih.gov/understanding-hiv-aids/fact-sheets/21/58/fda-approved-hiv-medicines>
- U.S. Department of Veterans Affairs. (2018). *HIV/AIDS*. Understanding laboratory tests. Retrieved from <https://www.hiv.va.gov/patient/diagnosis/labs-index.asp>
- Wang, C. C., Silverberg, M. J., & Abrams, D. I. (2014). Non-AIDS-defining malignancies in the HIV-infected population. *Current Infectious Disease Reports*, 16(6), 406.
- World Health Organization. (2015). *Guideline on when to start antiretroviral therapy and on pre-exposure prophylaxis for HIV*. Retrieved from [http://apps.who.int/iris/bitstream/10665/186275/1/9789241509565\\_eng.pdf](http://apps.who.int/iris/bitstream/10665/186275/1/9789241509565_eng.pdf)
- Yang, H., Nkeze, J., & Zhao, R. Y. (2012). Effects of HIV-1 protease on cellular functions and their potential applications in antiretroviral therapy. *Cell & Bioscience*, 2(1), 32. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3490751/>

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## Instructions for Taking the **CE Test Online** HIV/AIDS: An Update for Home Healthcare Clinicians

- Read the article. The test for this CE activity can be taken online at [www.nursingcenter.com/ce/HHN](http://www.nursingcenter.com/ce/HHN). Tests can no longer be mailed or faxed.
- You will need to create a free login to your personal CE Planner account before taking online tests. Your planner will keep track of all your Lippincott Professional Development online CE activities for you.
- There is only one correct answer for each question. A passing score for this test is 14 correct answers. If you pass, you can print your certificate of earned contact hours and the answer key. If you fail, you have the option of taking the test again at no additional cost.
- For questions, contact Lippincott Professional Development: 1-800-787-8985.

Registration Deadline: December 4, 2020

Disclosure Statement:

The authors and planners have disclosed no potential conflicts of interest, financial or otherwise.

Provider Accreditation:

Lippincott Professional Development, will award 1.5 contact hours for this continuing nursing education activity.

Lippincott Professional Development is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 1.5 contact hours. Lippincott Professional Development is also an approved provider of continuing nursing education by the District of Columbia, Georgia, and Florida CE Broker #50-1223.

Payment:

- The registration fee for this test is \$17.95.