Approximately 39.5 million people are infected with the human immunodeficiency virus (HIV) or acquired immunodeficiency syndrome (AIDS) throughout the world (Viana et al., 2011). AIDS was first discovered in 1981 when a group of patients were found to exhibit unusual opportunistic infections, including pneumonia and Koposi sarcoma, a rare skin tumor (Kindt et al., 2007). Scientists discovered that these patients had lowered cellular immune responses and significantly decreased numbers of T-helper cells, which are a type of white blood cells that activates and directs other immune cells to destroy pathogens (Hutchinson, 2001). In 1983, human immunodeficiency virus 1 (HIV-1) was found to be responsible for the development of AIDS (Kates, 2006). HIV-1 is a retrovirus, which means they carry their genetic information in the form of RNA instead of DNA (NAID, 2004). When the retrovirus enters a cell, the RNA is transcribed into copy DNA (cDNA) (NAID, 2004). The virus, called a provirus at this point, can now replicate along with the host cell DNA, which would lead to cell death, or it can remain latent (NAID, 2004). HIV-1 specifically belongs to a group of retroviruses called lentiviruses that are characterized by a long lag time between the initial infection and the beginning of symptoms (NIAID, 2004). HIV-1 can be spread by sexual contact via seminal or vaginal fluid, infected blood via shared drug use equipment or transfusions, and from mother to child via breast milk (Des Jarlais et al., 1988; Demartino et al., 1992; Hutchinson, 2001).

In 1986, a virus that is morphologically similar to HIV-1, but causes the production of different antigens, was found in patients in western Africa (Sharp and Hahn, 2011). This virus is termed human immunodeficiency virus 2 (HIV-2). Oddly, this virus is more closely related to a simian virus (or simian immunodeficiency viruses (SIV)) then it is to HIV-1 (Sharp and Hahn, 2011). HIV-2 infected patients tend to have a lower amount of viruses compared to HIV-1 infected patients; and most cases of HIV-2 infected patients do not progress to AIDS, although, when HIV-2 progresses to AIDS in patients symptoms are almost identical to HIV-1 to AIDS progression (Sharp and Hahn, 2011).

After the first case of AIDS was reported in 1981 and the discovery of HIV in 1984, extensive research has been conducted to characterize, test for, prevent, treat and potentially find a cure for HIV and AIDS (Kates 2006). Because HIV is a rapidly evolving virus, it is important to be able to accurately test for and determine if one possesses the virus. Furthermore, early
detection of HIV may lead to more effective treatment(s), thus potentially reducing mortality (Kates 2006). Since the discovery of HIV/AIDS, many tests have been developed. The developments of successful tests require their ability to: detect HIV-specific antibodies, circulating viral antigens, RNA, isolate the virus, or detect the genetic material from HIV in cells or blood plasma (Phair and Wolinsky 1992). Currently, there are several means to obtain samples for testing that include blood, plasma, oral fluid, and urine (Kates 2006). The most commonly used tests are the Western Blot test, which is used to detect specific proteins in cells or tissue lysates; Enzyme-linked immunosorbent assay (ELISA), which detects the presence of specific antigens; line immunoassay, which detect recombinanat HIV-1 and HIV-2 antigens; and indirect immunofluoroscence assay, which is used to detect specific antibodies via microscope (Branson and Mermin, 2011, Zhao et al. 2011).

ELISA was the first HIV-1 antibody test approved by the FDA in 1985 (Kates, 2006). ELISA was first described in 1971 by Engvall and Perlman, and has been used to test for the presence of antigens (the foreign molecules) or antibodies (immune system proteins that detect antigens) for various diseases. There are three different types of ELISA: indirect, sandwich, and competitive. The indirect ELISA test is the preferred test for HIV-1 because the methodology is simple, inexpensive, and accurate (Van der Groen et al., 1991). Indirect ELISA tests for the presence of antibodies, whereas sandwich and competitive ELISA test for the presence of antigens (Kindt et al., 2006). For indirect ELISA, blood serum from the patient is added to a well that is coated with antigen in question, HIV-1 in this case. If the patient is infected with HIV-1 (i.e. the patient has HIV-1 antibodies), the antibodies will bind to the antigen (Kindt et al., 2006). Then, another set of antibodies that are linked with a specific enzyme are added to the well to bind with the original antibodies (Kindt et al. 2006). A material is then added to bind with the enzyme. Once bound, a reaction producing color takes place; if the color is observed, the patient is presumed to be infected with HIV-1(Kindt et al., 2006).

Results from this type of testing are classified into two categories: HIV-1 seropositive and HIV-1 seronegative and as such, two types of errors may occur (type I and type II) (Gentleman et al. 1992). A type I error occurs when HIV-1 seronegative results are classified as HIV-1 seropositive and a type II error occurs when HIV-1 seropositive results are classified as HIV-1 seronegative (Gentleman et al. 1992). Obviously type II errors are most detrimental.
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