Evolution, Distribution, and Diversity of Immunodeficiency Viruses

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Abstract

Immunodeficiency viruses infect the host and primarily affect the immune system of an organism, i.e., host. Till date Human Immunodeficiency Virus (HIV), Simian Immunodeficiency Virus (SIV), Feline Immunodeficiency Virus (FIV), Bovine Immunodeficiency Virus (BIV), and Dog Immunodeficiency Virus (DIV) were reported in the literature. These viruses belong to phylum—Incertae sedis, family—Retroviridae, genus—*Lentivirus*, and order—Ortervirales. The review discusses about evolution, distribution, and diversity of immunodeficiency viruses, which helps in understanding the biology of HIV and how to develop a vaccine to the most harmful and dreadful diseases.

Keywords

Bovine immunodeficiency virus (BIV) · Dog immunodeficiency virus (DIV) · Feline immunodeficiency virus (FIV) · Human immunodeficiency virus (HIV) · Immunodeficiency virus · Simian immunodeficiency virus (SIV)

13.1 Immunodeficiency Viruses

Viruses that infect the host and affect the immune system of the host upon infection are generally known as immunodeficiency viruses (IV). The host then acquires a disease known as Acquired Immunodeficiency Disease Syndrome (AIDS). These immunodeficiency viruses belong to phylum—Incertae sedis, family—Retroviridae, genus—*Lentivirus*, and order—Ortervirales. The different types of

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immunodeficiency viruses are Human Immunodeficiency Virus (HIV) (Barré-Sinoussi et al. 1983; Clavel et al. 1986), Simian Immunodeficiency Virus (SIV) (Daniel et al. 1985), Feline Immunodeficiency Virus (FIV) (Pedersen et al. 1987), Bovine Immunodeficiency Virus (BIV) (Van Der Maaten et al. 1972a), and Dog Immunodeficiency Virus (DIV) (Safran et al. 1992). Among these viruses, HIV and SIV are widely studied and there are also reports on other immunodeficiency viruses like FIV, BIV, and DIV.

13.2 Human Immunodeficiency Virus

HIV is a retrovirus that infects human and affects the immune system of the human upon infection (Fig. 13.1). The host then acquires a disease known as AIDS. Worldwide statistics as of 2017 for HIV and AIDS are mentioned in Table 13.1. In 1983, HIV called HIV1 was isolated by "… researchers at the Pasteur Institute in France …" which was known to cause AIDS (Barré-Sinoussi et al. 1983). In 1986 "… HIV-2, was isolated from AIDS patients in West Africa …" (Clavel et al. 1986).

13.2.1 Evolution, Distribution, and Diversity of HIV

Two types of HIVs exist, HIV1 and HIV2; among them, HIV1 was discovered first and HIV2 was identified later (Fig. 13.2). HIV1 is distributed worldwide (Fig. 13.3), whereas HIV2 is observed mainly in western Africa (Vidal et al. 2000). The reason for widespread HIV1 can due to the ancestor of HIV1, which might have mutated at a much faster rate and traveled along with *Homo sapiens* population generating diversity in HIV1 (Vidal et al. 2000; Sharp and Hahn 2008). HIV1 group might have stemmed from strain SIVcpz of SIV (Keele et al. 2006), whereas HIV2 group might have stemmed from strain SIVsmm of SIV (Gao et al. 1992, 1994). HIV1 is further classified into group M (main), group O (outlier), group N (non-M/non-O), and group P. Group M is further subdivided into ten subtypes A–K, CRF's (Vidal et al. 2000; Sharp and Hahn 2008). Lemey et al. (2004) classified HIV2 into groups A and B. Further, HIV2 was classified

Table 13.1 Worldwide statistics as of 2017 for HIV and AIDS (Global Statistics 2017)

S. no.	Parameter	Statistics
1	People living with HIV	36.9 million (31.1–43.9 million)
2	People on antiretroviral therapy	21.7 million (19.1–22.6 million)
3	People newly infected with HIV	1.8 million (1.4–2.4 million)
4	People died with HIV infection	940,000 (670,000–1.3 million)
5	People living with HIV since epidemic	77.3 million (59.9–100 million)
6	People died with HIV infection since epidemic	35.4 million (25.0–49.9 million)

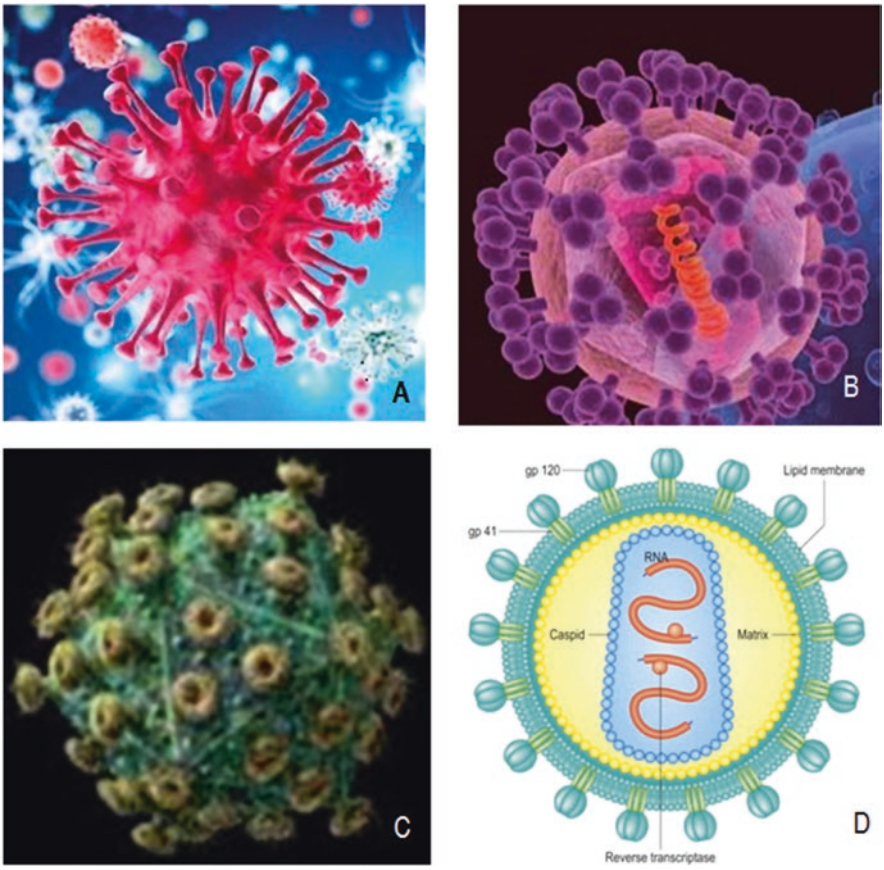

Fig. 13.1 (**a**) HIV infects humans and causes disease known as AIDS, (**b**) SIV infects nonhuman primates and causes disease known as SAIDS, (**c**) FIV infects cats and causes disease known as FAIDS, and (**d**) BIV infects cattle and causes disease known as BAIDS

phylogenetically into hypothetical groups such as group C–G, and 96FR12034 (Damond et al. 2004). Epidemiological, physiological, clinical, and phylogenetic evidence favored that HIV1 and HIV2 are due to "… several cross-species transmission of HIV from chimpanzee to humans …" (Castro-Nallar et al. 2012; Huet et al. 1990; Hahn et al. 2000; Plantier et al. 2009; Van Heuverswyn and Peeters 2007). Intensive studies were carried out on the evolution and divergence of HIV1 and HIV2 using phylogeny. The divergence time of HIV1, subtype A of HIV2 and subtype B of HIV2 dated to 1920s (Worobey et al. 2008), 1940 ± 16 (Lemey et al. 2003) and 1945 ± 14 (Gilbert et al. 2007), respectively. HIV1 was introduced into North America in 1968 (1966–1970) (Pérez-Losada et al. 2010; Surekha and Neelapu 2019).

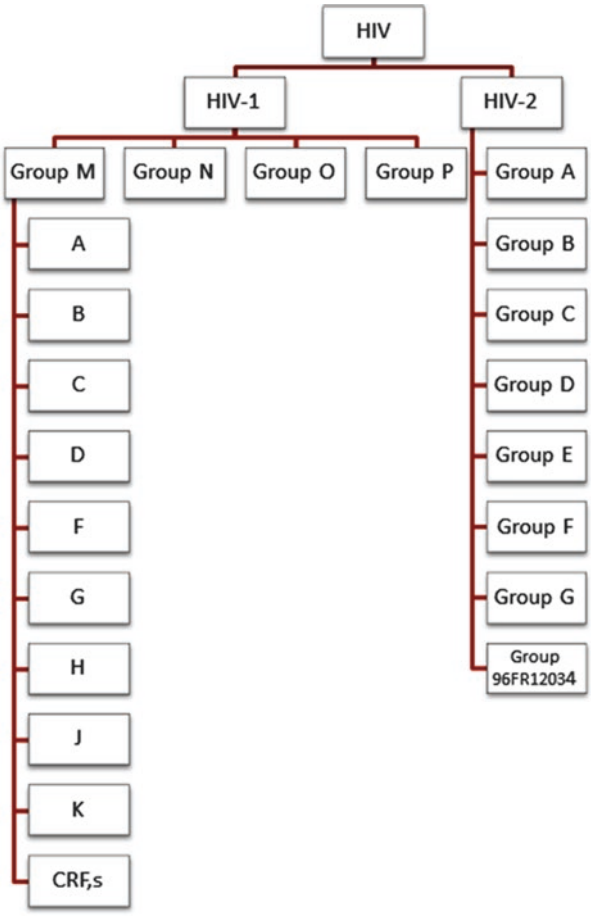

Fig. 13.2 Classification of HIV into two major types HIV1 and HIV2

13.2.2 Pathogenesis of HIV

HIV1 advances into the host cells using two molecules of HIV envelope (glycoprotein—gp120 and gp41). This ingress of glycoproteins stimulates intracellular signal cascades and facilitates replication of virus. The glycoproteins gp120 and gp41 form spikes on the surface of virion's. The protein gp120 binds to the $CD4^+$ receptor and attaches to the host cell membrane. A marked decrease in $CD4^+$ T cells (both activated and memory) is the characteristic feature of infection and disease. The virus then interacts with receptors (CCR5, CXCR4) leading to structural changes of the protein. The viral and the cellular membranes of the host are fused and form a viral pore. This allows the transfer of the viral core into the cytoplasm of the host. After the core disassembles, the RNA is reverse transcribed into DNA by the virus' with the help of enzyme reverse transcriptase. The DNA is then integrated into the

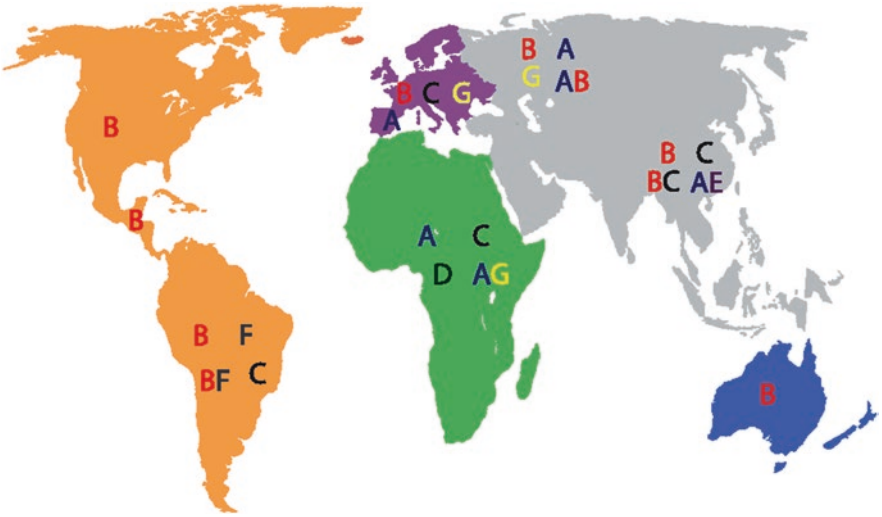

Fig. 13.3 Worldwide representation of HIV1 and its subtypes across the continents (Source: Castro-Nallar et al. (2012))

gene rich and transcriptionally active domains of the host's genome with the help of the viral protein integrase, DNA repair enzymes of the host and LEDGF/p75 (lens epithelium-derived growth factor—integrase binding host factor). Once the host cell is transformed into a potential virus producer, transcription of viral genetic material occurs. Later viral proteins are transported and assembled at the cell membrane with the help of vesicular sorting pathway (ESCRT-I, -II, and -III). TSG101 domain, a short sequence motif in p6 of Gag I is used for protein sorting. Cleavage of the Gag-Pol polyprotein by the viral protease produces mature infectious virions …" (Naif 2013).

13.3 Simian Immunodeficiency Virus

Simian Immunodeficiency Virus (SIV) infects "non-human primates" and affects the immune system of the "non-human primates" upon infection (Fig. 13.1). The host then acquires a disease known as Simian Acquired Immunodeficiency Disease Syndrome (SAIDS). SIV was isolated in 1985 from captive rhesus macaques suffering from SAIDS (Daniel et al. 1985).

13.3.1 Evolution, Distribution, and Diversity of SIV

SIVs infect nearly 45 different species of "non-human primates." Different types of SIV infecting "non-human primates" are mentioned in Table 13.2 (Gordon et al. 2005). SIV may be present at least 32,000 years ago in monkeys and apes (Worobey

Table 13.2 Major types of SIVs and their corresponding nonhuman primate hosts infected with SIV

S. no.	SIV type	Nonhuman primate infected with SIV	S. no.	SIV type	Nonhuman primate infected with SIV
1	SIVcpz	Chimpanzee	19	SIVsun	Sun-tailed monkey
2	SIVgor	Gorillas	20	SIVprg	Preuss's guenon
3	SIVagm	African green monkeys	21	SIVwrc	Western red colobus
4	SIVsmm	Sooty mangabeys	22	SIVolc	Olive colobus
5	SIVrcm	Red-capped mangabeys	23	SIVkrc	Kibale red colobus
6	SIVgsn	Greater spot-nosed monkeys	24	SIVtrc	Tshuapa red colobus
7	SIVmus	Mustached guenons	25	SIVsyk	Sykes' monkey
8	SIVmon	Mona monkeys	26	SIVdeb	De Brazza's monkey
9	SIVagi	Agile mangabey	27	SIVden	Dent's mona monkey
10	SIVmnd 2	Mandrill	28	SIVwol	Wolf's mona monkey
11	SIVdrl	Drill monkey	29	SIVgsn/SIVmon/ SIVmus 1/SIVmus 2 clade, SIVtal	Northern talapoin
12	SIVmac	Rhesus macaque	30	SIVasc	Red-tailed guenon
13	SIVmne	Pig-tailed macaque	31	SIVbkm	Black mangabey
14	SIVstm	Stump-tailed macaque	32	SIVreg [formerly SIVery]	Red-eared guenon
15	SIVsab, SIVver, SIVgri	Grivet monkey	33	SIVblu	Blue monkey
16	SIVtan	Tantalus monkey	34	SIVcol	Colobus guereza
17	SIVmnd 1	Mandrill	35	SIVkcol 1	Black-and-white colobus
18	SIVlho	L'hoest's monkey	36	SIVkcol 2, SIVblc (formerly SIVbcm)	Black colobus

et al. 2010). Primate's infection with SIV dated back to 14 Ma, if the virus and host were coevolved then it dates to 85 Ma (Compton et al. 2013). The "non-human primates" include species of gorilla, chimpanzees, and monkeys. The species of gorilla are *Gorilla gorilla* (western lowland gorillas), and *Gorilla beringei* (eastern Grauer's gorillas). The species of western gorilla are subdivided into *Gorilla gorilla diehli* (Cross River gorilla) and *Gorilla gorilla gorilla* (western lowland gorilla). The eastern species are classified into *Gorilla beringei graueri* (Grauer's gorilla), *Gorilla beringei beringei* (mountain gorilla), and *Gorilla beringei* subspecies (Bwindi gorilla). The chimpanzee species include *Pan troglodytes verus* (western Africa), *Pan troglodytes* ellioti (Nigerian), *Pan troglodytes troglodytes* (central), and *Pan troglodytes schweinfurthii* (eastern). The monkey species include African green monkeys, sooty mangabeys, *Cercocebus torquatus* (red-capped mangabeys),

Cercopithecus nictitans (greater spot-nosed monkeys), *Cercopithecus cephus* (mustached guenons), and *Cercopithecus mona* (mona monkeys). The association of SIV with apes, gorillas, and monkeys can be approximately 32,000 years or even much longer (Worobey et al. 2010). It is hypothesized that "… several cross-species transmission events …" of SIVcpz and SIVsmm might have resulted in evolution of HIV1 and HIV2 (Huet et al. 1990; Hahn et al. 2000; Plantier et al. 2009; Van Heuverswyn and Peeters 2007). SIVcpz from chimpanzees "… crossed the species barrier …" and migrated to humans giving rise to HIV1. Similarity, SIVsmm from sooty mangabeys "… crossed the species barrier …" and migrated to humans giving rise to HIV-2.

13.3.2 Pathogenesis of SIV

SIV infects $CD4^+$ T cells and the SIV-infected cells undergo apoptosis within 1 day of infection. The immune system of simians is not able to replace the cells at the same pace leading to the deterioration of immune function. Subsequently, the host acquires immunodeficiency syndrome leaving the animal susceptible to fatigue and life-threatening coinfections (Fig. 13.4a). "… The interaction of SIV with the host's immune system triggers innate immune responses followed by virus-specific adaptive cellular and humoral immune responses. Rapidly occurring mutations lead to immune evasion. Chronic immune activation contributes to the functional impairment of the immune system. As the disease progresses, adaptive immune responses are unsuccessful in containing the virus replication, and overt signs of a chronic immune suppression become evident …" (Schmitz and Korioth-Schmitz 2013). SIV infection leads to changes in mucosal tissues (cervicovaginal, gastrointestinal, and penile tissues) and all organ systems (brain, lung, and heart) (Haase 2011). The severity of the infection has an impact and functionally impairs the organs (Alammar et al. 2011; Kelly et al. 2012). Successful infection of SIV also results in loss of $CD4^+$ T cells. SIV uses glycoproteins to bind $CD4^+$ receptors of T cells and interacts with co-receptor CXCR4 leading to conformational changes of the protein. "… The viral and the cellular membranes of the host are fused allowing the transfer of the SIV genome into the cytoplasm of the host. The RNA is reverse transcribed into DNA by the virus with the help of enzyme reverse transcriptase. Then this DNA is integrated into the host cell's genome, transforming the host cell into a potential virus producer. The virus then uses host cell's replication machinery to transcribe its DNA back into RNA. The copies of RNA are packed into virus particles of about 80–100 nm in diameter and bud off or free from the host cell infecting more cells. SIV infections are non-pathogenic in their natural African simian primates. However, if the virus infects an Asian or Indian rhesus macaque, these non-African simian primates will develop simian AIDS (SAIDS). SIVs nef gene down-regulates expression of CD3, CD4, and MHC class I and therefore do not induce immunodeficiency. Whereas, nef gene in HIV-1 lost its ability to down-regulate CD3, which results in the immune activation and apoptosis …" (Swigut et al. 2004).

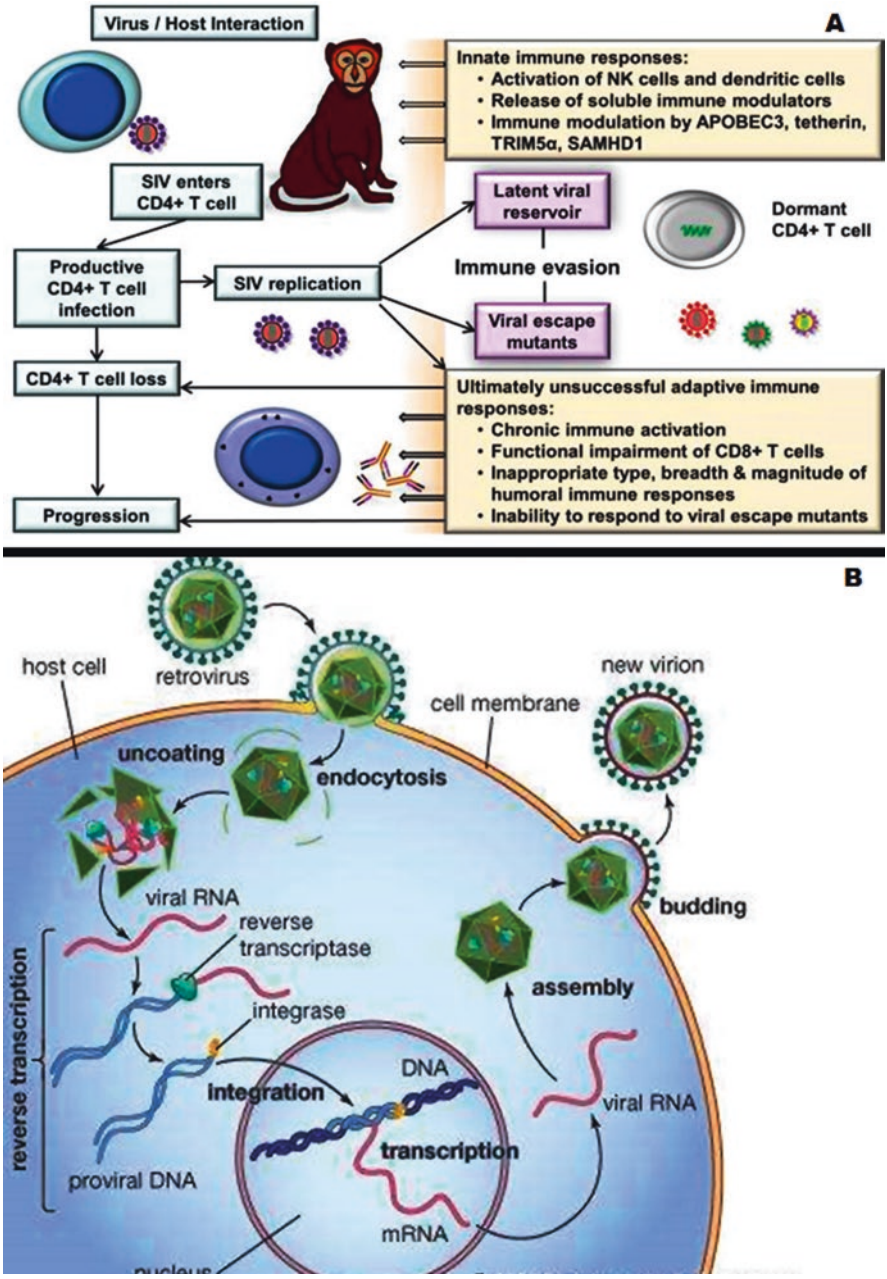

Fig. 13.4 (**a**) Consequences and immunopathogenesis in simian when infected with a simian immunodeficiency virus (SIV) (Source: Schmitz and Korioth-Schmitz (2013)). (**b**) Replication of SIV in the host simian (Source: Encyclopaedia Britannica Inc. https://www.britannica.com/science/SIV)

13.4 Feline Immunodeficiency Virus

Feline Immunodeficiency Virus (FIV) infects cats and affects the immune system of the cats upon infection (Fig. 13.1). The cat then acquires a disease known as Feline Acquired Immunodeficiency Disease Syndrome (FAIDS). FIV was first isolated from a cat exhibiting an immunodeficiency-like syndrome (Pedersen et al. 1987).

13.4.1 Evolution, Distribution, and Diversity of FIV

FIV is endemic in Felidae species (Biek et al. 2003, 2006; Brown et al. 1994; Carpenter et al. 1996, 1998; Carpenter and O'Brien 1995; Driciru et al. 2006; Hofmann-Lehmann et al. 1996; Munson et al. 2004; Olmsted et al. 1992; Troyer et al. 2004; Troyer et al., 2005), from free-ranging lions (Pecon-Slattery et al. 2004) to domestic cats (Olmsted et al. 1989). FIV occurs in *Felis catus* (domestic cats), *Puma concolor* (pumas) (Langley et al. 1994), *Panthera leo* (lions), *Otocolobus manul* (pallas cat) (Barr et al. 1997), *Panthera pardus* (puma leopard), *Acinonyx jubatus* (cheetah), *Leopardus pardalis* (ocelot), *Panthera onca* (jaguar), *Leopardus weidii* (margay), *Leopardus tigrina* (tigrina), large African lions, and *Crocuta crocuta* (spotted hyaena) (Troyer et al. 2005). The different types of FIVs like FIV_{Ple}, FIV_{Fca}, FIV_{Pco}, FIV_{Oma}, FIV_{Lpa}, FIV_{Aju}, FIV_{Ccr}, FIV_{Pon}, FIV_{Lwe}, and FIV_{Lti} were reported in literature (Fig. 13.5). The major types of FIVs and their corresponding felines infected with FIV are listed in Table 13.3.

Currently, isolates of FIV are classified into six subtypes (A, B, C, D, E, and F) (Weaver 2010). Weaver (2010) performed a detailed phylogenetic analysis of FIV and classified FIV into six subtypes (A, B, C, D, E, and F) (Fig. 13.4). Sodora et al. (1994) initially classified FIV into three subtypes (A, B, and C). Sodora et al. (1994) used phylogenetic analysis to classify FIV isolates based on FIV *env* (envelope sequence). The geographical location of the subtype A is California and Europe; subtype B is Japan and the central and eastern USA; and Subtype C is southwestern Canada. Nishimura et al. (1998), later classified FIV into five subtypes (A, B, C, D,

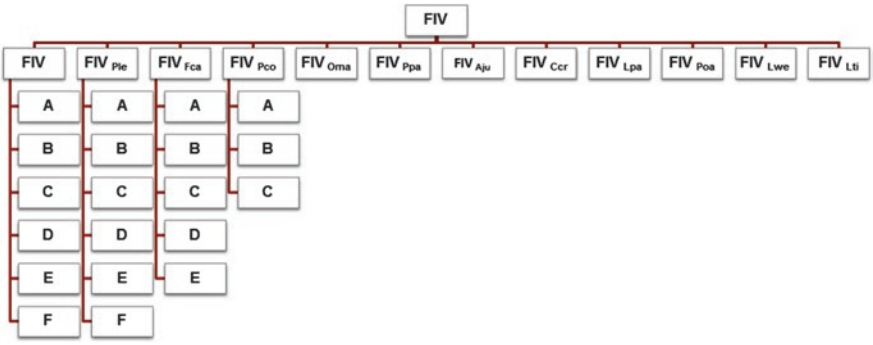

Fig. 13.5 Classification of FIV and their subtypes

Table 13.3 Major types of FIVs and their corresponding felines infected with FIV

S. no.	FIV type	Felines infected with FIV
1	FIV_{Ple}	*Panthera leo* (lions)
2	FIV_{Fca}	*Felis catus* (domestic cats)
3	FIV_{Pco}	*Puma concolor* (pumas)
4	FIV_{Oma}	*Otocolobus manul* (pallas cat)
5	FIV_{Ppa}	*Panthera pardus* (puma leopard)
6	FIV_{Aju}	*Acinonyx jubatus* (cheetah)
7	FIV_{Ccr}	*Crocuta crocuta* (spotted hyaena)
8	FIV_{Lpa}	*Leopardus pardalis* (ocelot)
9	FIV_{Pon}	*Panthera onca* (jaguar)
10	FIV_{Lwe}	*Leopardus weidii* (margay)
11	FIV_{Lti}	*Leopardus tigrina* (tigrina)

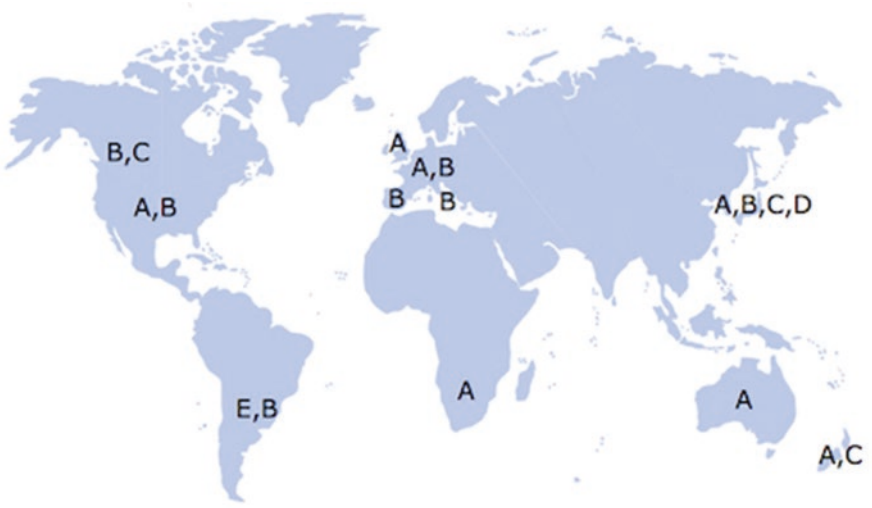

Fig. 13.6 Worldwide distribution of FIV and its subtypes across the continents (Source: Teixeira et al. (2012))

and E) based on FIV *env* (envelope sequence). The geographical location of the subtype A is California and Northern Europe; subtype B is central and eastern USA and southern European countries; subtype C is California and British Columbia; subtype D is Japan; and subtype E is Argentina. FIV_{Ple} has diverged into six subtypes A–F, each with distinct geographic areas of endemicity (Brown et al. 1994; Troyer et al. 2004; O'Brien et al. 2006). FIV_{Fca} has diverged into five subtypes, A–E (Sodora et al. 1994; Kakinuma et al. 1995; Pecoraro et al. 1996), where subtypes A, B, and C are widespread worldwide. Subtype D is found in Japan and Vietnam (Kakinuma et al. 1995; Nakamura et al. 2003), whereas subtype E is only found in Argentina (Pecon-Slattery et al. 2008). The four subtypes A–D are found in cat populations from Japan (Nishimura et al. 1998; Kakinuma et al. 1995). FIV_{Pco} has diverged into three subtypes A, B, and C (Pecon-Slattery et al. 2008) (Fig. 13.6).

13.4.2 Pathogenesis of FIV

FIV uses receptor CD9 (Poeschla and Looney 1998) for entry and then infects $CD4^+$ T (Brown et al. 1991), FIV can also infect astroglial cells, $CD8^+$ T lymphocytes, macrophages, and kidney cells (Pedersen et al. 1989; Brunner and Pedersen 1989; Phillips et al. 1990; Zenger et al. 1995). "… This leads to a significant drop in cells which have critical roles in the immune system. Low levels of $CD4^+$ and other affected immune system cells cause the cat to be susceptible to opportunistic diseases once the disease progresses to feline acquired immune deficiency syndrome (FAIDS) …" (Bendinelli et al. 1995). Symptoms of immunodeficiency associated with FIV are chronic lesions, opportunistic infections, neurological abnormalities, and weight loss (Yamamoto et al. 2010).

CD134 is mostly present on T cells which are activated and binds to OX40 ligand, causing T cell activation, stimulation, proliferation, and apoptosis. The virus enters the host's cells using the glycoprotein env and interacts with surface receptors. The glycoprotein SU binds to receptor CD134 of the host cell leading to conformational changes of protein SU. These changes facilitate interaction between SU and the chemokine receptor CXCR4; and fuses viral membrane and cellular membrane of the host (Hu 2012). "… This allows the transfer of the viral RNA into the cytoplasm of the host. Then RNA is reverse transcribed and integrated into the genome of the host through non-homologous recombination. Once viral RNA is integrated into the host genome, the virus can be dormant in the asymptomatic stage without being detected by the immune system of the host or can cause lysis of the host cell …" (Lecollinet and Richardson 2008; Hartmann 2011).

13.5 Bovine Immunodeficiency Virus

Bovine Immunodeficiency Virus (BIV) infects cattle and affects the immune system of the cattle upon infection (Fig. 13.1). The cattle then acquires a disease known as Bovine Acquired Immunodeficiency Disease Syndrome (BAIDS). BIV strain "… R-29 was originally isolated from an 8-year-old dairy cow suspected of having bovine lymphosarcoma …" (Van Der Maaten et al. 1972a).

13.5.1 Evolution, Distribution, and Diversity of BIV

BIV is widespread in dairy and beef cattle in Australia (Forman et al. 1992), Canada (McNab et al. 1994), France (Polack et al. 1996), Germany (Muluneh 1994), Italy (Cavirani et al. 1998), Japan (Hirai et al. 1996; Meas et al. 1998), Korea (Cho et al. 1999), Netherlands (Horzinek et al. 1991a), New Zealand (Horner 1991), the UK (Clayton 1994), the USA (Amborski et al. 1989; Cockerell et al. 1992; St. Cyr Coats et al. 1994), and buffaloes in Pakistan (Meas et al. 2000). The prevalence of BIV in dairy cattle is higher than beef cattle (Amborski et al. 1989; Cho et al. 1999). The different BIV strains reported in literature are BIV R-29 (isolated from cow) (Van

Der Maaten et al. 1972b), BIV-106, and BIV-127 (variants of BIV R-29) (Braun et al. 1988; Garvey et al. 1990), BIV CR1 (BIV strain from Costa Rica (Hidalgo et al. 1995), BIV FL112 (Hirari et al. 1996), and FL491 (Suarez et al. 1993) (strain from Florida, USA).

13.5.2 Pathogenesis of BIV

BIV infects nondividing host cells and uses two phases—entry phase (first phase) and replication phase (second phase) to replicate itself (Berkowitz et al. 2001). In the first phase, glycoprotein of the virus envelope interacts and binds with the specific receptor of the cell. "… Then virus uses either of the ways, receptor mediated endocytosis or viral envelope-cell membrane fusion to enter into the host cell. The virus then dissembles in the cell and reverse-transcribes the RNA genome into DNA with the help of enzyme reverse transcriptase (Gonda, 1992). The activity of reverse transcriptase is higher at lower concentrations of Mn^{2+} ions when compared to Mg^{2+} ions (Horzinek et al. 1991b). The DNA is then transported to nucleus and is integrated into the genome of the host cell. In the second phase, the integrated viral DNA is transcribed and the transcript (viral mRNA) is transported and translated in the cytoplasm …." The translated viral structural proteins are then assembled into virus particles and form a viral complex (virus buds) along with the viral RNA at the plasma membrane. Then viral proteases process the viral buds and are released from the cell in the mature stages, which are ready to infect another cell (Gonda 1992).

13.6 Dog Immunodeficiency Viruses

Dog Immunodeficiency Virus (DIV) is a retrovirus which infects dog and affects the immune system of the dogs upon infection. The dog then acquires a disease known as DAIDS. Safran et al. (1992) isolated canine immunodeficiency virus, (CIV)—(canine lentivirus) from a leukemic dog. The ultrastructure and morphogenesis of CIV is strikingly similar to that displayed by other lentiviruses, while the immunological relatedness close to any other lentivirus or oncovirus was not established. These findings suggest that this canine retrovirus, fits in the subfamily lentivirus and not related to other known members. Additional, investigations on DIV (CIV) are essential to establish the biology, immunopathogenesis of virus; and how immunodeficiency is acquired by dogs.

13.7 Conclusions and Future Perspectives

Immunodeficiency viruses infect the host and primarily affect the immune system of an organism i.e. host. HIV, SIV, FIV, BIV, and DIV were known to infect humans, simians, cats, cattle, and dogs respectively. The ultrastructure and morphogenesis of IVs like HIV, SIV, FIV, BIV, and DIV are strikingly similar. At the same time

immunological relatedness of the lentiviruses like HIV, SIV, FIV and BIV are close with exception in DIV. Therefore, there is a need to establish the immunological relatedness of the DIV. The similarities in the ultrastructure, morphogenesis and immunological relatedness of lentivirus provide us with an opportunity for better understanding of the immunodeficiency in different hosts. This information further can be used to develop a vaccine to the most harmful and dreadful disease.

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Authors Contribution HSV, CS and NNRR initiated the review, participated in writing and revised the manuscript.

Conflict of Interest Statement The authors declare that there is no potential conflict of interest.

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