

**Table 2 Notes on selected full-length nucleotide sequences that appear in the alignments.**

Sequence	Accession	Origin	Reference
B-WEAU	U21135	USA	Ghosh, S. K., Unpublished (1995)
<p>WEAU is the reference strain for this alignment as well as for the Los Alamos Molecular Immunology Compendium, the companion volume for this HIV sequence compendium. The sequence is from a cytopathic HIV-1 virus clone from an isolate from an acutely infected patient, from Birmingham, Alabama. The lambda phage clone was obtained from a co-culture of this patient's PBMCs, first with normal donor PHA-stimulated lymphocytes for 14 days, and then with the H9 T-cell line for another 14 days. The blood specimen was obtained 15 days after the onset of clinical symptoms of acute (primary) infection, and 35 days after a single sexual encounter (receptive anal intercourse) with a partner whose virus was proven phylogenetically to be responsible for the transmission event. The clone that was sequenced has an SI phenotype, and a frameshift in nef. The single nucleotide deletion in nef found in the full length WEAU 1.60 clone is not present in 10/10 PCR amplified sequences from the patient's uncultured PBMCs (instead there is a "T".) The full-length WEAU 1.60 provirus was sequenced in its entirety by two different laboratories (George Shaw and Leroy Hood) with 100% concordance. George Shaw provided detailed information concerning this sequence. The patient WEAU is "Patient #1" in Clark, S. J. et al., <i>N Engl J Med</i> 324:954-960 (1991); "WEAU 0575" in Piatak, M., et al., <i>Science</i> 259:1749-1754 (1993) and is also discussed in Borrow, P., et al., <i>Nat Med</i>, 3:205-11 (1997).</p>			
A-U455	M62320	Uganda	Oram, J. et al., <i>ARHR</i> 6:1073-1078 (1990)
<p>This sequence is from the 1985 Ugandan isolate U455. It was cloned in phage, and is defective env, vpr and vpu. The env ORF in this sequence is interrupted by an in-frame stop codon beyond the COOH end of the V5 region. This sequence clusters with subtype A HIV-1.</p>			
A-92UG037.1	U51190	Uganda	Gao, F. et al., submitted (1997)
<p>Sample 92UG037 is part of a set of isolates obtained through the WHO Global Programme on AIDS (WHO Network, <i>ARHR</i> 10:1327-1344 (1994)) and comes from an asymptomatic 31-year-old female from Entebbe, Uganda; she had not taken any anti-retroviral therapy prior to sampling. The risk factor for infection was heterosexual contact. The isolate 92UG037 was established and propagated by short term cocultivation with normal donor lymphocytes, and then the near full length genome was PCR amplified and sequenced. 92UG037 is subtype A. An LTR sequence is available under accession numbers U51287, and an additional env/nef sequence with accession number U09127. There is an inframe stop codon in pol at position 3144 in this clone. The isolate from which this sequence was derived is NSI and uses CCR5 or CCR8 (Bjorndal, A. et al., <i>J Virol</i> 71:7478 (1997) and Rucker, J., et al., <i>J Virol</i> 71:8999-9007 (1997). See also: Gao, F. et al., <i>J Virol</i> 70:7013-7029 (1996) This sequence was kindly made available prior to publication, and the manuscript is currently submitted (Gao F., et al., (1997)).</p>			
A-Q23.17	AF004885	Kenya	Poss, M. Unpublished (1997)
<p>This subtype A sequence was from a woman from Mombasa, Kenya, who had been recently infected with HIV-1. The blood sample was drawn in 1993. An env gene fragment from a PCR amplification from this same blood sample was published in Poss M., et al. <i>ARHR</i> 13:493-499 (1997). The full length sequence was kindly released prior to publication by M. Poss and colleagues, U. Washington.</p>			

## Introduction

**Table 2 (cont.)**

Sequence	Accession	Origin	Reference
B-MN	M17449	USA	Gurgo C., et al., <i>Virology</i> 164:531–536 (1988)
			MN is from one of the earliest available isolates, and is a commonly used reference and vaccine strain. The MN isolate was taken from a 6 year old male pediatric AIDS patient in 1984. The MN sequence was cloned from the isolate in lambda phage. The coding sequences for pol, nef and vpu are prematurely truncated. A set of V3 sequences from this isolate are available L48364-L48379, Lukashov, V. and Goudsmit J., <i>AIDS</i> 9:1307–1311 (1995)
B-AD8	AF004394	USA	Theodore, T. S., et al., <i>ARHR</i> 12:191–194 (1996)
			AD8 is a molecular clone that was replication competent after reconstruction. It has a defective vpu due to the loss of the start codon. It was derived from the macrophage tropic isolate AD87. AD87 was derived from isolate ADA, described in Westervelt, P., et al., 88:3097–3101 <i>PNAS</i> (1991)
B-WR27	U26546	USA	Salminen, M. O., et al., <i>Virology</i> 213:80–86 (1995)
			WR27 was a patient with clinical progression to Walter Reed Stage 5 when blood was drawn for viral isolation in 1988. This sequence is from a PCR clone from a primary isolate that was expanded in donor PBMC. The virus sequence had a V3 loop predictive of an SI phenotype. Rev has an inframe stop codon in both exons.
B-RF	M17451	Haiti	Starcich, B. R., et al., <i>Cell</i> 45:637–648 (1986)
			RF is among the first isolates, and is commonly used reference and vaccine study strain. The sequence is from the full-length lambda phage clone HAT-3, from isolate RF, cultured in HUT-78 cells. RF is from a 28 year old symptomatic Haitian male, who moved to the U.S. in 1980 and was sampled in 1983, shortly before his death. RF has defective gag and vpu genes. Several env genes are available from this isolate, U30778-U30781. See also: Reitz M., et al., <i>ARHR</i> 8:1950 (1992)
B-89.6	U39362	USA	Collman, R., et al., <i>J Virol</i> 66:7517–7521 (1992)
			89.6 is cloned from a highly cytopathic primary macrophage-tropic isolate from an AIDS patient living in Philadelphia, although the patient was originally from Jamaica. 89.6 was cloned in phage and was replication competent after reconstruction. Also see Kim, F., et al., <i>J Virol</i> 69:1755–1761 (1995)
B-BCSG3	L02317	USA	Ghosh, S. K., et al., <i>Virology</i> 194:858–864 (1993)
			The isolate BC was obtained in 1987, from an individual from the U.S. with terminal AIDS. The clone was obtained after short term co-culture of HUT-78 cells with a PHA-stimulated PBMCs. BCSG3 is the sequence from the provirus SG3; the full length provirus was cloned intact in lambda phage. It is very cytopathic in human T-cell lines, and did not replicate in monocyte-macrophages. BC and BCSG3 replicate more efficiently in chimpanzee than in human lymphocytes. All typical HIV-1 genes are intact except vpu, which is normally 243 bp in length; vpu was disrupted after 70 nucleotides by a 23 bp deletion that results in a frameshift and truncation of the open reading frame.
B-JRFL	U63632	USA	O'Brien, W. A., et al., <i>Nature</i> 348:69–73 (1990)
			This sequence is from an infectious lambda phage clone of the 1986 isolate JRFL, derived from from the brain of a patient who died with Kaposi's sarcoma and severe AIDS encephalopathy. The infectious clone JRCSF was isolated from the CSF of the same patient. Both are NSI. Also see: Pang, J., et al., <i>JAIDS</i> , 4:1082–92 (1991) and Klasse, P. J., et al., <i>ARHR</i> 12:347–350 (1996).

**Table 2 (cont.)**

Sequence	Accession	Origin	Reference
B-JRCSF	M38429	USA	O'Brien, W. A., et al., <i>Nature</i> 348: 69–73 (1990)
			This sequence is from an infectious lambda phage clone of the 1986 isolate JRCSF, derived from from the CSF of a patient who died with Kaposi's sarcoma and severe AIDS encephalopathy. The infectious clone JRFL was isolated from the brain of the same patient. Both are NSI. A SI mutant clone of this isolate has also been sequenced in env U45960. Also see: Pang, J., et al., <i>JAIDS</i> , 4:1082–92 (1991) and Klasse, P. J., et al., <i>ARHR</i> 12:347–350 (1996).
B-OYI	M26727	Gabon	Wain-Hobson, S., et al., <i>AIDS</i> 3:707 (1989)
			This sequence is derived from the Gabonese isolate OYI, isolated from a healthy HIV-1 infected individual. GA.OYI was the first viral sequence from Africa that was found to phylogenetically cluster with North American viruses. This sequence is from a lambda phage clone, and the cloned provirus was functionally defective. The vpu gene does not have a start codon.
B-CAM1	D10112	UK	McIntosh, A., Unpublished (1991)
			This sequence is from the British isolate CAM1. It has a defective vpu gene. McIntosh A, and Karpas A, Thesis (1991), Cambridge University, England.
B-NY5	M38431	USA	Willey, R., et al., <i>PNAS</i> 83:5038–5042 (1986)
			This sequence is from the 1984 New York T-cell tropic isolate NY5. It was cloned in lambda phage and is not replication competent. It has a defective vpu gene due to the loss of the start codon. See also GenBank accession number K03346, for an env gene sequence from this isolate.
B-pNL43	M19921	USA/France	Adachi, A., et al., <i>J Virol</i> 59, 284–291 (1986)
			A NY5/LAI laboratory generated chimera that has been extensively studied, with envelope from LAI(BRU) and gag-pol from NY5.
B-HXB2	K03455	France	Wong-Staal F., et al., <i>Nature</i> 313:277–284 (1985)
			This sequence was from provirus cloned in lambda phage, and is derived from the IIIB isolate related to LAI (see LAI, below). This clone has been extensively studied.
B-LAI	K02013	France	Wain-Hobson, S., et al., <i>Cell</i> 40:9–17 (1985)
			This sequence is from the French isolate LAI (formerly called BRU), and was obtained from a complete provirus cloned in lambda phage. This is an infectious molecular clone after reconstruction. Also see: Alizon, M. et al., <i>Cell</i> 46:63–74 (1986), and Wain-Hobson, S., <i>Science</i> 252:961–965 (1991), for clarification on the origins of early French isolates. The IIIB strain is also very closely related to LAI. There are several other LAI-IIIB related clones that have been fully sequenced (LAI/BRU, BH10, PV22, PM213, MCK1, LW12–3, HXB2, and TH475), but in our printed compendium we include only the two common reference strains HXB2 and LAI; our ftp site alignment includes the full set of full length LAI-related HIV sequences. These were the first HIV isolates, are T-cell tropic, and are the most extensively studied variants in terms of mutational analysis of functional domains, phenotype, vaccine design, and antigenicity. The LW12–3 sequence is a complete viral genome derived from a IIIB/LAI infected lab worker who was first infected in 1985, sampled in 1987, and who was still asymptomatic in 1994 (Reitz, M., <i>ARHR</i> 10:1143–1155 (1994)). As the use of LAI-related cultures, reagents and clones is ubiquitous in AIDS research, these sequences are occasionally found as contaminants in sequencing studies (although LAI/IIIB sequences are certainly not the only possible source of contamination, they are the most common). A very unusual QR insertion in the V3 loop of the Envelope protein has been used as red flag to signal the possibility of LAI/IIIB contaminations.

## Introduction

**Table 2 (cont.)**

Sequence	Accession	Origin	Reference
B-YU2	M93258	USA	Li, Y., et al., J Virol 66:6587–6600 (1992)
<p>YU-2 is a lambda phage clone that is replication competent. It was from the uncultured brain tissue of a patient with AIDS dementia complex. YU2 and YU10 differ by 0.26% in nucleotide sequence, YU-2 was fully replication competent after reconstruction, in both primary T lymphocytes and monocyte-macrophages. TU-2 a defective vpu gene due to the loss of the start codon. Also see Li, Y., et al., J Virol 65: 3973–3985 (1991).</p>			
B-YU10	M93259	USA	Li, Y., et al., J Virol 66:6587–6600 (1992)
<p>YU10 is a sequenced lambda phage clone from the same source as YU2, the uncultured brain tissue of a patient with AIDS dementia complex. There is a frame shift mutation in the pol gene in YU10. YU2 and YU10 differ by 0.26% in nucleotide sequence. Also see Li, Y., et al., J Virol 65:3973–3985 (1991).</p>			
B-ACH320.2A.2.1	U34604	The Netherlands	Guillon, C., et al., ARHR 11:1537–1538 (1995)
<p>This is the sequence of the complete genome of an NSI, macrophage tropic lambda phage clone from an isolate taken from the PBMC of a patient who was making a shift from NSI to SI in bulk phenotype. A syncytium inducing clone from the same isolate has also been completely sequenced, ACH320.2A.1.2. The patient, isolates and phenotype of the molecular clones are described in Groenink, M., et al., J Virol 65:1968–1975 (1991).</p>			
B-ACH320.2A.1.2	U34603	The Netherlands	Guillon, C., et al., ARHR 11:1537–1538 (1995)
<p>This is the sequence of the complete genome of an SI, syncytium-inducing lambda phage clone from an isolate taken from the PBMC a patient who was making a shift from NSI to SI in bulk phenotype. An NSI clone from the same isolate has also been completely sequenced, ACH320.2A.2.1. The patient, isolates and phenotype of the molecular clones are described in Groenink, M., et al., J Virol 65:1968–1975 (1991)</p>			
B-SF2	K02007	USA	Sanchez-Pescador, R., et al., Science 227:484–492 (1985)
<p>This sequence is from an infectious phage clone from the U.S. isolate ARV-2. ARV-2/SF2 was isolated from the PBMC of a patient with oral candidiasis after co-culture with mitogen-stimulated PBMCs, (Levy, J., et al., Science 225:840–842, (1984)). It is a standard reference strain, and has been used for vaccine studies.</p>			
B-D31	U43096	Germany	Kreutz, R., et al., ARHR 8:1619–1629 (1992)
<p>This sequence is from isolate D31, about which little information is available.</p>			
B-MANC	U23487	UK	Zhu, T., and Ho., D. Nature 374:503–504 (1995)
<p>This sequence was PCR amplified from the 1959 sample “Manchester sailor“ kidney tissue (see: Corbitt. G., et al., Lancet 336:51 (1990)). The sequence of the complete genome is available, and it is indistinguishable from contemporary subtype B HIV-1 sequences in phylogenetic analysis. This information, together with the observation that additional tissue samples were HIV PCR negative, suggests that the HIV clone that came from the MANC kidney sample was very likely to be a contemporary clinical contaminant. The sequence was assembled from multiple PCR amplified fragments. All reading frames in this sequence are intact.</p>			

**Table 2 (cont.)**

Sequence	Accession	Origin	Reference
B-HAN	U43141	Germany	Sauermann, U., et al., ARHR 6:813–823 (1990)
<p>HAN2 was isolated from a 39 year old homosexual German patient with AIDS related complex in 1986. This patient died from complications of AIDS in 1987. HAN2 was highly cytopathic in MT-2 cells. Genomic DNA from infected MT-2 cells was used to prepare a lambda phage genomic library. Two full-length clones, HAN2/2 and HAN2/3 were purified. Clone HAN2/3 was used for DNA sequencing, and has a defective env gene.</p>			
B-C18MBC	U37270	Australia	Deacon, N., et al., Science 270:988–991 (1995)
<p>A sequence from one of the six Australian long term survivors (subject C18) that were all infected via blood transfusions with blood from the same HIV+ donor, D36, a homosexual male who was infected some time between Dec. 1980 and April 1981. All were healthy, and symptom-free 10 to 14 years after infection, in 1995, at the time of sampling. C18 was born in 1912, and was infected by transfusion in August of 1983. C18 PMBC DNA was PCR amplified in overlapping fragments which were cloned and sequenced. The virus is attenuated due to extensive deletions the nef coding region, and the U3 region of the LTR. Different members of the cohort have different patterns of nef deletions. The 18MBC sequence was assembled from multiple PCR amplified fragments. The authors point out the importance of Nef in determining the pathogenicity of HIV-1 and suggest this strain of HIV-1 as a possible basis for a live attenuated vaccine, or for further study of this approach.</p>			
C-ETH2220	U46016	Ethiopia	Salminen, M. et al., ARHR 12:1329–1339 (1996)
<p>ETH2220 is the first reported near full length subtype C sequence from Ethiopia. The patient sample from which this clone was obtained was taken in 1986. In its genomic organization, this clone closely resembles subtype A, B, and D isolates except that the core promoter contains three potential binding sites for the transcription factor NF-kB instead of containing two, a feature which was preserved in other Ethiopian C subtype samples, as well as C viruses from Zambia. This sequence was cloned as a PCR amplified near full length genome, and has a defective tat gene.</p>			
C-92BR025.8	U52953	Brazil	Gao, F. et al., submitted (1997)
<p>This sequence is from a PCR clone from a primary isolate that is part of a set of isolates obtained through the WHO Global Programme on AIDS (WHO Network, ARHR 10:1327–1344 (1994)). 92BR025 was from a 23 year old male hemophiliac from Porto Alegre, Brazil. He had recently seroconverted, though sometime more than 1.2 months prior to the date this blood sample was collected in 1992. He was asymptomatic, and had not taken any anti-retroviral therapy prior to sampling. The isolate 92BR025 was established and propagated by short-term cocultivation with normal donor lymphocytes, and then the near full length genome was PCR amplified and sequenced. The HIV isolate BR025 exhibited an NSI phenotype when assayed by the WHO. The clone has two inframe stop codons in pol at positions 2141, and 3115, and a frame shift mutation at position 4131. This sequence was kindly made available prior to publication, and the manuscript is currently submitted (Gao F., et al., submitted (1997)). Additional env, nef, and ltr region sequences are available from this isolate: U09126, U09132, U51282, and U15121.</p>			
D-84ZR085	U88822	Zaire	Gao, F. et al., submitted (1997)
<p>Sample 84ZR085 was obtained from an AIDS patient from Zaire. The near full length genome was cloned in phage and sequenced. D-84ZR085 is subtype D. There was a frame shift mutation in gag/pol, position 1692. This isolate was obtained from Thomas Jefferson University, and isolate phenotyping information was not available. This sequence was kindly made available prior to publication, and the manuscript is currently submitted (Gao, F., et al., submitted (1997)).</p>			

**Table 2 (cont.)**

Sequence	Accession	Origin	Reference
D-NDK	M27323	Zaire	Spire, B. et al., <i>Gene</i> 81:275–284 (1989)
			This is an infectious molecular clone derived from a very cytopathic isolate. It was cloned in phage and is replication competent. All reading frames in this sequence are intact. Spire et al. reported that only minor sequence differences appear to be responsible for the “acute biological effect”. This sequences clusters with HIV-1 subtype D in phylogenetic analysis.
D-Z2Z6	M22639	Zaire	Theodore, T. and A. Buckler-White, Unpublished (1988)
			An infectious molecular clone of this virus was created by reconstruction. It was cloned in phage and sequenced from the isolate Z2, also called CDC-Z34. All reading frames in this sequence are intact.
D-ELI	K03454	Zaire	Alizon, M. et al., <i>Cell</i> 46:63–74 (1986)
			This sequence is of a phage clone derived from the Zairean isolate ELI. ELI was recovered in 1983 from a 24 year old woman with AIDS. All reading frames are intact. A reconstructed infectious clone is available. Gag (M27954) and env (M27949) sequences from the same isolate are also available.
D-94UG114.1	U88824	Uganda	Gao, F. et al., (1997)
			Sample 94UG114 was obtained from an asymptomatic 31-year-old man from Butuku, Uganda, as part of the WHO/UNAIDS study. He had not taken any anti-retroviral therapy prior to sampling. His risk factor for infection was heterosexual contact. The near full length genome was PCR amplified from a short term culture of a PBMC sample and sequenced. 92UG114.1 is subtype D. There were no defective gene products, and an infectious molecular clone of 94UG114.1 is available. The isolate from which this sequence was derived is NSI by an MT-2 assay. This sequence was kindly made available prior to publication, and the manuscript is currently (Gao F., et al., submitted (1997)).
F-93BR020.1	AF005494	Brazil	Gao, F. et al., submitted, (1997)
			This isolate is part of a part of a set obtained through the WHO Global Programme on AIDS (WHO Network, ARHR 10:1327–1344 (1994)) and came from a asymptomatic HIV seropositive 52 year old man from Rio de Janeiro, Brazil, sampled in 1993. The risk factor for infection was bisexual contact. The isolate 92BR020 was established and propagated by short term cocultivation with normal donor lymphocytes, and then the near full length genome was PCR amplified and sequenced. The isolate 93BR020 was described as syncytium inducing (SI) using an MT-2 assay. An envelope gene sequence from this isolate is described in Gao, F., et al., <i>J Virol</i> 70, 1651–1657 (1996). This sequence was kindly made available prior to publication, and the manuscript is currently submitted Gao, F., et al., (1997)).
H-90CF056.1	AF005496	CAR	Gao, F. et al., (1997)
			This sequence clusters with available HIV-1 subtype H sequences in phylogenetic analysis, and is the first available full length H subtype sequence . The isolate comes from Bangui, in the Central African Republic, and was sampled in 1990, from an asymptomatic individual, who had no anti-retroviral therapy. The isolate had an NSI phenotype by an MT-2 assay, and the sample was obtained from the Pasteur Institute, Bangui. The isolate 90CF056 was established and propagated by short term cocultivation with normal donor lymphocytes, and then the near full length genome was PCR amplified, cloned and sequenced. The isolate was at one point designated 90CR056, but was changed to 90CF056 as CR stands for Costa Rica, and CF for Central African Republic. The first genetic characterization of this virus isolate (an env V3 sequence designated 4056, GenBank accession number L11497, Murphy, E., et al., ARHR 9:997–1006 (1993)) left the subtype designation as unclassified, but a second study of this env region sequence classified it as subtype H (Janssens, W., ARHR 10:877–879 (1994)). This sequence was kindly made available prior to publication, and the manuscript is currently submitted (Gao, F., et al., (1997)). An additional ltr (U51290) and env and nef genes are available from this isolate (U08797 and U27401).

**Table 2 (cont.) Inter-subtype recombinants**

Sequence	Accession	Origin	Reference
AC-ZAM184	U86780	Zambia	Salminen, M., et al., <i>J Virol</i> 71:2647–2655 (1997)
<p>The envelope gene from this sample taken from a Zambian woman in 1990 (Louwagie, J., et al., <i>J Virol</i> 69:263–271, 1995) proved to be an A/C recombinant in subsequent phylogenetic analyses (D. Robertson et al., <i>Nature</i> 374:124–126 (1995)). The full length provirus was recovered using PCR, and sequenced, according to the method described in Salminen, M., et al., <i>Virology</i> 213:80–86 (1995). Additional clones from serial samples from the index case ZAM184, and from her spouse who was also HIV-1 positive ((GenBank Accession # U86768-U86781, inclusive) represent gag, env, or additional full sequences from this couple. The A/C mosaic pattern of the full length genomic sequence is presented in Salminen, M., et al., <i>J Virol</i> 71:2647–2655 (1997), and also in Robertson, D., et al., part III pages 25–30, of this compendium, 1997. All coding sequences are intact. L22955.</p>			
AC-92RW009.6	U88823	Rwanda	Gao, F., et al., submitted, (1997)
<p>The isolate 92RW009 was part of the set generated through the WHO Global Programme on AIDS (see: WHO Network, ARHR 10:1327–1343 (1994)). The virus was derived from an asymptomatic 24 year old woman from Kigali, Rwanda, whose route of infection is thought to be heterosexual contact, and who had no anti-retroviral therapy. The clone has a frameshift mutation in gag at position 213. The blood sample was taken in 1992. The original env and gag sequence from this isolate clustered with HIV-1 subtype A sequences (Gao, F., et al., ARHR 10: 1359–1368 (1994)), however subsequent in-depth analysis of the full length genome sequence from this isolate suggests it is an AC mosaic sequence with multiple crossover points. The full length sequence and the information concerning its mosaic nature was kindly made available prior to publication, and the manuscript is currently submitted (Gao, F. et al., (1997)). The breakpoints are mapped in Robertson, D., et al., part III pages 25–30, of this compendium, 1997. The cytopathic effect of both the primary isolate from this patient, and from early passage cultured virus was NSI on MT-2 cells when analyzed by two different WHO labs in 1993–1994 (WHO Network, ARHR 10:1327–1343 (1994), and De Wolf, F., et al., ARHR 10: 1387–1400 (1994)). The NIAID 1997 Reference Reagent Catalog classifies it as NSI. However, more recent papers (Zhang L., et al., <i>Nature</i> 383:768 (1996) and Zhang L., et al., ARHR 13(16), 1357–1366 (1997), classify it as SI. This sequence is from PCR-amplified proviral DNA harvested from PBMCs from the NIH NIAID reagent repository. The NSI phenotyping was determined using earlier passages, and SI was associated with later passages, (J. Bradac, personal communication). Both the full length clone 92RW009.6 and the partial gp120 clone 92RW009.14 (accession U08793) were amplified from the same DNA extracted from a short term primary PBMC culture prepared by Ogden Bioservices, stored in the NIAID Research Reagent Repository, lot 1. Other env sequences from this isolate are: U16221, U08793, U13441, U16220, and U16222, and a gag sequence is U86545.</p>			
ADI-MAL	K03456	Zaire	Alizon, M. et al., <i>Cell</i> 46:63–74 (1986)
<p>This sequence is from a lambda phage clone derived from the Zairean isolate MAL. MAL was recovered in 1985 from a 7 year old boy with ARC, probably infected by a blood transfusion in 1981, as his parents were seronegative. All reading frames are intact except for vpu, due to the loss of a start codon. A reconstructed infectious clone is available. MAL was one of the first African sequences characterized, and soon after the initial characterization it was determined to be a mosaic (Li, W.-H., et al., <i>Genetics</i> 116:s44 (1987)). Recent analysis suggests that it has three different distinct subtype associations, A, D and I, and some regions that are difficult to characterize as associated with any known subtype (see Robertson, D., et al., part III pages 25–30, of this compendium, 1997). The I association is based on phylogenetic associations with a clone from Cyprus isolate that was the first I subtype characterized (94CY032), the 94CY032 clone appears to be an A-G-I mosaic (Robertson, D., et al., part III pages 25–30, of this compendium, 1997).</p>			

**Table 2 (cont.)**

Sequence	Accession	Origin	Reference
AE-93TH253.3	U51189	Thailand	Gao, F. et al., <i>J Virol</i> 70:7013–7029 (1996)
<p>This virus was isolated in 1993 from a 21 year old man from Chiang Mai, Thailand, who had end-stage AIDS. The isolate was previously designated CMU010, or 302053. The isolate was expanded in donor PBMCs, then in H9 cells, then a lambda phage clone was generated and sequenced. The sequence has a defective vpu due to the lack of a start codon, and a defective env gene. Like other "E" subtype viruses from both Asia and Africa, large stretches of the genome are associated with the A subtype, and all share a common mosaic pattern of A/E breakpoints, suggesting that the currently identified A-E recombinants all share a common ancestor. The breakpoints are mapped in Robertson, D., et al., part III pages 25–30, of this compendium, 1997.</p>			
AE-CM240	U54771	Thailand	Carr, J. et al., <i>J Virol</i> 70:5935–5943 (1996)
<p>Blood from an asymptomatic heterosexual 21-year-old Thai man was transported from Thailand to the USA where PBMCs were separated and co-cultivated with PHA-stimulated donor PBMCs. DNA from p24 antigen-positive culture was used to PCR amplify the proviral DNA. The complete genomic sequence of the provirus was determined by the compilation of three clones containing different parts of the viral genome. CM240 is an example of a Thai subtype E virus, which is a mosaic of a clade A virus and clade E virus, with the gag gene (and other regions) of subtype E viral genome falling within clade A in phylogenetic analysis. This is the pattern of A-E sequences found throughout Asia and Africa, and no full length E subtype reference strain has been identified (as for 93TH253). Carr et al. provide detailed analysis of the breakpoints, and point out that the A/E mosaic genomes have a natural pseudotype structure where the external envelope protein spikes on the virion essentially are contributed by the E subtype, and the rest of the viral proteins have a subtype A origin. The breakpoints are also mapped in Robertson, D., et al., part III pages 25–30, of this compendium, 1997. See also the env sequence from the same isolate (L14572), Mascola, J., et al., <i>JID</i> 169:48–54 (1993)</p>			
AE-90CF402.1	U51188	CAR	Gao, F. et al., <i>J Virol</i> 70:7013–7029 (1996)
<p>The isolate 90CF402 was sampled from a man from Bangui, the Central African Republic, who was suffering from AIDS-related conditions. His risk factor for infection was heterosexual contact. The isolate was first adapted to growth in chimpanzee cells, then re-expanded in human PBMC before a lambda phage clone was generated and sequenced. The sequence has a defective vpu due to the lack of a start codon, and a defective vif gene. A reconstructed infectious molecular clone is available. The pattern of subtype A-E recombination breakpoints is shared between A-E subtype sequences from Thailand and from the Central African Republic, suggesting a shared ancestral recombined virus that arose prior to the subsequent epidemics in the two areas. The breakpoints are mapped in Robertson, D., et al., part III pages 25–30, of this compendium, 1997.</p>			
AGI-Z321	U76035	Zaire	Choi, D. et al., <i>ARHR</i> 13:357–361 (1997)
<p>Z321B is a later passage of isolate Z321 (see GenBank Accession Numbers M15896, U50208, and U50207); Z321 was isolated from a 1976 Zairean serum sample. Z321 was grown to industrial scale as a chronically infected T-cell line to manufacture an inactive, therapeutic HIV-1 immunogen. Z321B was then established from this industrial scale stock. The genomic sequence was derived from multiple PCR clones amplified from Z321B. Z321B contains a mutation in the termination codon of the tat gene, (bases 2294–2296) so that the tat gene of HIVZ321 extends further downstream (bases 2342–2344), and has a defective vpr and vpu. Recent analysis suggests that it has three different distinct subtype associations, A, G and I, and some regions that are difficult to characterize as part of any known subtype (see Robertson, D., et al., part III page 25–30 of this compendium, 1997). The I association is based on phylogenetic associations with a clone from Cyprus isolate that was the first I subtype characterized (94CY032). The 94CY032 clone also appears to be an A-G-I mosaic (Robertson, D., et al., part III pages 25–30, of this compendium, 1997).</p>			

**Table 2 (cont.)**

Sequence	Accession	Origin	Reference
AG-92NG083.2	U88826	Nigeria	Gao, F. et al., submitted (1997)
<p>This sequence is from a PCR clone from a primary culture from the NSI isolate 92NG083; the sample was taken in 1992 from an AIDS patient from Jos, Nigeria. The isolate was originally called JV1083, but was renamed 92NG083 to be consistent with WHO nomenclature. While originally described as subtype G in env (Abimiku, A., et al., ARHR 10:1581–1583 (1994)), analysis of the full length genome revealed mosaic A/G regions. The breakpoints are mapped in Robertson, D., et al., part III pages 25–30, of this compendium, 1997. The full length clone has an altered initiation codon at position 157 and an inframe stop codon at position 360 in gag, and a vpu frameshift mutation at position 5462. This sequence was kindly made available prior to publication, and the manuscript is currently submitted (Gao F., et al., submitted (1997)).</p>			
AG-92NG003.1	U88825	Nigeria	Gao, F. et al., submitted (1997)
<p>This sequence is from a PCR clone from an NSI primary culture from isolate G3, renamed 92NG003 to be consistent with WHO nomenclature. The sample was taken in 1992 from a 27 year old, asymptomatic HIV seropositive female prostitute from Jos, Nigeria. (Abimiku, A., ARHR 10:1581–1583 (1994), env sequence accession number U13208). The isolate came from the Institute of Human Virology, Baltimore, MD. This sequence was kindly made available prior to publication, and the manuscript is currently submitted (Gao F., et al., submitted (1997)). While originally described as subtype G in env, this genome has mosaic A/G regions. The breakpoints are mapped in Robertson, D., et al., part III pages 25–30, of this compendium, 1997. There are frameshift mutations associated with 10–16 bp deletions in vpr and vpu, at positions 5024 and 5485, as well as deletions totaling 33 bp near the 3' end of the V3 loop. Nef has an altered initiation codon at position 8113.</p>			
AG-IbNg	L39106	Nigeria	Howard, T. et al., ARHR 12:1413–1425 (1996)
<p>HIV-1 sample IbNg was isolated from the PBMCs of an asymptomatic 23 year old man from Nigeria. DNA from this isolate was PCR amplified and cloned, with the complete genome sequence derived from multiple PCR amplification products. The partial env gene sequence (U48628) was originally designated subtype A (Howard, T. et al., ARHR 10:1755–1757 (1994)), as was the full length genome. The full length sequence was eventually shown to be an A-G recombinant with multiple cross-over points. The breakpoints are mapped in Robertson, D., et al., part III pages 25–30, of this compendium, 1997. The IbNg sequence has a 16 bp insertion within the Lys-tRNA primer binding site, just 3' of the 5' LTR. It also has a single nucleotide deletion in tat cds at position 5449.</p>			
BF-93BR029.4	AF005495	Brazil	Gao, F. et al., submitted, (1997)
<p>The isolate 93BR029 is part of a set of isolates obtained through the WHO Global Programme on AIDS (WHO Network, ARHR 10:1327–1344 (1994)), and came from an asymptomatic HIV seropositive 17 year old male, with unknown risk factor, from Sao Paulo, Brazil, sampled in 1993. The isolate had an NSI phenotype by an MT-2 assay. The isolate 93BR029 was established and propagated by short term cocultivation with normal donor lymphocytes, and then the near full length genome was PCR amplified and sequenced. An envelope gene sequence from this isolate is described in Gao, F., et al., J Virol 70, 1651–1657 (1996), U27413, and an LTR sequence is also available U51291. The envelope gene was first described as an F subtype, however subsequent phylogenetic analysis of the full length genome indicated that the clone is a mosaic with regions of B and F subtype. The breakpoints are mapped in Robertson, D., et al., part III pages 25–30, of this compendium, 1997. There are two frame shift mutations in gag, in positions 269 and 472. This sequence was kindly made available prior to publication, and the manuscript is currently submitted (Gao, F. et al., (1997)).</p>			

## Introduction

**Table 2 (cont.)**

Sequence	Accession	Origin	Reference
O-MVP5180	L20571	Cameroon	Gurtler, L. et al., J Virol 68:1581–1585 (1994) The isolate MVP5180 was derived from a Cameroonian woman who had AIDS in 1991; she died of AIDS in 1992. The isolate from which the clone was derived was grown in several T cell lines, and could also grow in the monocytic U937 cells.
O-ANT70	L20587	Cameroon	Vanden Haesevelde, M. et al., J Virol 68:1586–1596 (1994) ANT70 was isolated from the first O group infection discovered, and the very divergent LTR sequence was first published in 1990 (de Leys, R., et al., J Virol 64:1207–1216, 1990). The isolate came from a CDC stage III infected individual with unusual serological reactivity. O group viruses have the same genetic organization as M group viruses, which dominate the epidemic, but are quite distinct in terms of their genetic sequences. For a review, See Korber B., et al., Human Retroviruses and AIDS Database, Part III, 41–56, 1996
SIV-CPZANT	U42720	Zaire	Vanden Haesevelde, M. et al., Virology 221:346–350 (1996) The CPZANT was isolated from a wild caught chimpanzee from Zaire. Two additional SIV CPZ (chimp) viral isolates have been generated from chimps caught in Gabon (U11495, X52154). The chimpanzee viral sequences are genetically more closely related to the HIV-1 sequences derived from infected humans than are HIV-2 strains or other SIVs.
SIV-CPZGAB	X52154	Gabon	Huet, T. et al., Nature 345:356–359 (1990) The CPZGAB virus was isolated from a chimpanzee caught in Gabon. The genome is more closely related to HIV-1 than are HIV-2 or other SIV viral sequences. Also see CPZGAB2, U11495 for a sequence fragment from an additional chimpanzee caught in Gabon.