Table 9: gp120

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
223	M85	gp120(C1 30-51 LAI)	gp120(29-50)	ATEKLWVTVYYGVPVW-KEATTT	N	451 Env	murine(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996), Ditzel et al.(1997), Wyatt et al.(1997)]
NOTES:

- M85: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese et al.(1992)]
- M85: C1 domain – mutation 40 Y/D impairs binding – the relative affinity for denatured/native gp120 is $< .01$, suggesting conformational component [Moore et al.(1994c)]
- M85: Binding inhibited by MAb 4D4#85, enhanced by conformationally sensitive anti-V3 MAb 5G11, and some anti-18 MAbs [Moore & Sodroski(1996)]
- M85: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt et al.(1997)]

224	7E2/4	gp120(C1 31-50 LAI)	gp120(30-49)	TEKLWVTVYYGVPVWK-EATT		Env glycopro	murine(IgG)

Donor: S. Ranjbar, NIBSC, UK
References: [Moore et al.(1994c)]
NOTES:

- 7E2/4: C1 domain – the relative affinity for denatured/native gp120 is .07, suggesting conformational component [Moore et al.(1994c)]
- 7E2/4: UK Medical Research Council AIDS reagent: ARP3050

225	M92	gp120(C1 31-50 LAI)	gp120(40-49)	GVPVWKEATT	N	451 Env	rat(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d)]
NOTES:

- M92: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)]
- M92: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
226	4D4#85	gp120(C1 41-50 LAI)	gp120(40-49)	GVPVWKEATT		Env	murine(IgG)

Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA
References: [Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996), Wyatt et al.(1997), Binley et al.(1998)]
NOTES:

- 4D4#85: C1 domain – the relative affinity, denatured/native gp120 is 0.1 – mutation 45 W/S impairs binding [Moore et al.(1994c)]
- 4D4#85: Inhibits binding of C1 MAb M85, C1-C5 discontinuous epitope MAbs 181 and 212A, and CD4 binding induced MAbs 48d and 17b [Moore & Sodroski(1996)]
- 4D4#85: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-50, are deleted [Wyatt et al.(1997)]
- 4D4#85: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

227	M86	gp120(C1 42-61 LAI)	gp120(41-60)	VPVWKEATTTLFCASDA-KAY	N	451 Env	murine(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [di Marzo Veronese et al.(1992), Moore et al.(1994c)]
NOTES:

- M86: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese et al.(1992)]
- M86: C1 domain – the relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]

228	133/11	gp120(C1 64-78)	gp120(63-77)	EVHNVWATHACVPTD	L	IIIB gp120	murine(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [Niedrig et al.(1992b)]
NOTES:

- 133/11: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig et al.(1992b)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
229	133/237	gp120(C1 51-70 LAI)	gp120(60-69)	YDTEVHNVWA	L	IIIB gp120	murine(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [Niedrig et al.(1992b), Moore et al.(1994c), Moore et al.(1994d)]
NOTES:
- 133/237: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig et al.(1992b)]
- 133/237: The relative affinity, denatured/native gp120 is 1.4 – mutation of position 69 W/L impairs binding [Moore et al.(1994c)]

230	133/290	gp120(C1 61-70 LAI)	gp120(60-69)	YDTEVHNVWA	L	IIIB gp120	murine(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [Niedrig et al.(1992b), Thali et al.(1993), Moore et al.(1994c), Moore et al.(1994d), Wyatt et al.(1995), Binley et al.(1997), Wyatt et al.(1997), Binley et al.(1998)]
NOTES:
- 133/290: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig et al.(1992b)]
- 133/290: The relative affinity for denatured/native gp120 is 2.2 – mutation in position 69 W/L impairs binding [Moore et al.(1994c)]
- 133/290: Used for antigen capture assay, either to bind gp120 to the ELISA plate, or to quantitate bound gp120 [Wyatt et al.(1995)]
- 133/290: Reciprocal binding inhibition with the antibody 522-149, that binds to a discontinuous epitope – binding is enhanced by some C5 and C1 binding site antibodies [Moore & Sodroski(1996)]
- 133/290: A high avidity antibody as assessed by urea elution
- 133/290: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt et al.(1997)]
- 133/290: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

231	D/3G5	gp120(C1 73-82 LAI)	gp120(72-81)	ACVPTDPNPQ	N	Baculovirus-expressed rgp120 LAI	murine(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [Bristow et al.(1994)]
NOTES:
- D/3G5: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
232	D/6A11	gp120(C1 73-82 LAI)	gp120(72-81)	ACVPTDPNPQ	N	Baculovirus-expressed rgp120 LAI	murine

Donor: Fulvia di Marzo Veronese
References: [Bristow et al.(1994)]
NOTES:
- D/6A11: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]

233	D/5E12	gp120(C1 73-92 LAI)	gp120(72-91)	ACVPTDPNPQEVVLVNV-TEN	N	Baculovirus-expressed rgp120 LAI	murine

Donor: Fulvia di Marzo Veronese
References: [Bristow et al.(1994)]
NOTES:
- D/5E12: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]

234	4A7C6	gp120(C1 81-90 LAI)	gp120(80-89)	PQEVVLVNVT		Env glycopro	murine(IgG)

Donor: R. Tedder
References: [Thiriart et al.(1989), Thali et al.(1993), Moore & Ho(1993), Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996)]
NOTES:
- 4A7C6: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- 4A7C6: The relative affinity for denatured/native gp120 is 7.9 – mutation 88 N/P impairs binding [Moore et al.(1994c)]
- 4A7C6: C1 region epitope (88 N/P substitutions abrogates binding), but substitutions 380 G/F and 420 I/R also impaired binding [Moore et al.(1994d)]
- 4A7C6: Reciprocal binding inhibition with the antibody 133/192 – enhanced by anti-C5 antibodies, and C1 antibody 135/9[Moore & Sodroski(1996)]
- 4A7C6: UK Medical Research Council AIDS reagent: ARP 360

235	B242	gp120(C1 83-92 LAI)	gp120(82-91)	EVVLVNVTEN	N	Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	murine(IgG_1)

Donor: R. Tedder
References: [Bristow et al.(1994)]
NOTES:
- B242: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
236	1D10	gp120(C1 81-100 LAI)	gp120(80-99)	PQEVVLVNVTENFDMW-KNDM	L	IIIB-rgp120	rat

Donor: R. Tedder
References: [Dowbenko et al.(1988), Berman et al.(1991), Nakamura et al.(1992), Moore et al.(1994c)]
NOTES:
- 1D10: Cross-blocks 5B3 in IIIB-rsgp160 ELISA – type specific in rgp120 ELISA binding [Nakamura et al.(1992)]
- 1D10: The relative affinity for denatured/native gp120 is 13 – mutation 88 N/P impairs binding [Moore et al.(1994c)]

237	133/192	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	L	IIIB gp120	murine(IgG_1)

Donor: Matthias Niedrig
References: [Niedrig et al.(1992b), Moore et al.(1993b), Moore et al.(1994c), Moore & Sodroski(1996), Trkola et al.(1996a), Binley et al.(1997), Binley et al.(1998)]
NOTES:
- 133/192: Epitope seems complex, binds multiple peptides – weak neutralization of lab strain [Niedrig et al.(1992b)]
- 133/192: The relative affinity for denatured/native gp120 is 1.8 [Moore et al.(1994c)]
- 133/192: C1 region – substitutions 76P/Y, 113 D/A or R, 117 K/W, 420 I/R, 427 W/S impair binding, other substitutions enhanced binding [Moore et al.(1994d)]
- 133/192: Reciprocal binding inhibition with the antibody 4A7C6 – enhanced by some anti-C5 and-C1 antibodies [Moore & Sodroski(1996)]
- 133/192: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 133/192: A low avidity C1 antibody as assessed by urea elution
- 133/192: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
238	C6	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		mis-folded LAI rgp160	murine(IgG_1)

Donor: Matthias Niedrig
References: [Pincus & McClure(1993), Abacioglu et al.(1994), Moore et al.(1994c), Pincus et al.(1996)]
NOTES:
- C6: Also called Ch6?
- C6: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu et al.(1994)]
- C6: The relative affinity for denatured/native gp120 is 0.9 [Moore et al.(1994c)]
- C6: There is FNM/FDM polymorphism in LAI-based peptides – N is essential (J. P. Moore, per. comm.)
- C6: Called Ch6 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)]
- C6: NIH AIDS Research and Reference Reagent Program: 810

239	B2	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		mis-folded LAI rgp160	murine(IgG_{2b})

Donor: Matthias Niedrig
References: [Thali et al.(1993), Abacioglu et al.(1994), Moore et al.(1994c), Moore et al.(1994d), Binley et al.(1997)]
NOTES:
- B2: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu et al.(1994)]
- B2: The relative affinity for denatured/native gp120 is 1.4 [Moore et al.(1994c)]
- B2: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.)
- B2: A low avidity antibody as assessed by urea elution

240	GV4D3	gp120(92-100 IIIB)	gp120(91-99)	NFNMWKNDM		gp120 complexed with MAb M77	murine

Donor: Matthias Niedrig
References: [Denisova et al.(1996)]
NOTES:
- GV4D3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV4H4 and GV5F9 are homologous to GV4D3 and were generated in the same experiment [Denisova et al.(1996)]

241	D/4B5	gp120(C1 93-101 LAI)	gp120(92-100)	FNMWKNDMV	N	Baculovirus-expressed rgp120 LAI	murine

Donor: Matthias Niedrig
References: [Bristow et al.(1994)]
NOTES:
- D/4B5: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
242	D/6B2	gp120(C1 93-101 LAI)	gp120(92-100)	FNMWKNDMV	N	Baculovirus-expressed rgp120 LAI	murine(IgG_1)
		Donor: Matthias Niedrig **References:** [Bristow et al.(1994)] **NOTES:** • D/6B2: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]					
243	D/5A11	gp120(C1 93-101 LAI)	gp120(92-100)	FNMWKNDMV	N	Baculovirus-expressed rgp120 LAI	murine
		Donor: Matthias Niedrig **References:** [Bristow et al.(1994)] **NOTES:** • D/5A11: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]					
244	B9	gp120(C1 93-96 LAI)	gp120(92-95)	FNMW		mis-folded LAI rgp160	murine(IgG_1)
		Donor: Matthias Niedrig **References:** [Abacioglu et al.(1994)] **NOTES:** • B9: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
245	B10	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		mis-folded LAI rgp160	murine(IgG_1)
		Donor: Matthias Niedrig **References:** [Abacioglu et al.(1994), Moore et al.(1994c)] **NOTES:** • B10: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu et al.(1994)] • B10: The relative affinity for denatured/native gp120 is 0.4 [Moore et al.(1994c)] • B10: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.)					
246	L5.1	gp120(C1 89-103 IIIB)	gp120(78-92)	PNPQEVVLVNVTENF		vaccinia gp160	murine(IgG)
		Donor: Matthias Niedrig **References:** [Akerblom et al.(1990)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
247	B27	gp120(C1 94-97 BH10)	gp120(92-95)	FNMW	N	Baculovirus-expressed mis-folded rgp160 IIIB: NL43, MicroGenSys	murine(IgG_1)
		Donor: Matthias Niedrig **References:** [Abacioglu et al.(1994), Bristow et al.(1994)] **NOTES:** • B27: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)] • B27: MAbs generated in the context of a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]					
248	B35	gp120(C1 94-99 BH10)	gp120(92-97)	FNMWKN		mis-folded LAI rgp160	murine(IgG_1)
		Donor: Matthias Niedrig **References:** [Abacioglu et al.(1994)] **NOTES:** • B35: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
249	489.1(961)	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		Env	murine(IgG)
		Donor: C. Bruck, SKB, Belgium **References:** [Moore et al.(1994c)] **NOTES:** • 489.1(961): The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)] • 489.1(961): NIH AIDS Research and Reference Reagent Program: 961					
250	T1.1	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		vaccinia gp160	murine(IgG)
		Donor: C. Bruck, SKB, Belgium **References:** [Akerblom et al.(1990), Broliden et al.(1990), Moore et al.(1994c)] **NOTES:** • T1.1: Also reacted in solid phase with gp120(234-248) NGTGPCTNVSTQCT [Akerblom et al.(1990)] • T1.1: No ADCC activity – reactive peptide: NVTENFNMWKNDMVEQ, IIIB [Broliden et al.(1990)] • T1.1: C1 region – the relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]					
251	T7.1	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		Env	murine(IgG)
		Donor: C. Bruck, SKB, Belgium **References:** [Akerblom et al.(1990), Bolmstedt et al.(1990), Moore et al.(1994c), Moore et al.(1994d)] **NOTES:** • T7.1: The relative affinity of denatured/native gp120 is 4.0 [Moore et al.(1994c)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
252	T9	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		Env	murine(IgG)

Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Akerblom et al.(1990), Bolmstedt et al.(1990), Moore et al.(1994c), Moore et al.(1994d), Binley et al.(1997)]
NOTES:
- T9: There appear to be two T9s
- T9: The relative affinity of denatured/native gp120 is 7.9 [Moore et al.(1994c)]
- T9: C1 region – 45 W/S, 88 N/P, 256 S/Y, 262 N/T, 475 M/S, 485 1.83, and 491 I/F enhanced binding, no substitution tested significantly inhibited [Moore et al.(1994d)]
- T9: A low avidity antibody as assessed by urea elution

253	5B3	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	N	IIIB-rspg160	murine(IgG)

Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Berman et al.(1991), Nakamura et al.(1992), Beretta & Dalgleish(1994), Moore et al.(1994c)]
NOTES:
- 5B3: Blocks gp120 -CD4 binding [Berman et al.(1991)]
- 5B3: Cross-blocks 1D10 in competitive IIIB-rsgp160 ELISA – no neutralization – blocks IIIB-gp120 sCD4 binding – localized binding to residues 72-106 [Nakamura et al.(1992)]
- 5B3: The relative affinity of denatured/native gp120 is 8.3 [Moore et al.(1994c)]

254	MF49.1	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		Env	murine(IgG)

Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Thiriart et al.(1989), Moore et al.(1994c)]
NOTES:
- MF49.1: The relative affinity of denatured/native gp120 is 3.8 [Moore et al.(1994c)]

255	B20	gp120(C1 101-110 LAI)	gp120(100-109)	VEQMHEDIIS		mis-folded LAI rgp160	murine(IgG_{2a})

Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Abacioglu et al.(1994), Moore et al.(1994c)]
NOTES:
- B20: C1 region – epitope boundaries mapped by peptide scanning – HEDII core [Abacioglu et al.(1994)]
- B20: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
256	B18	gp120(C1 101-110 LAI)	gp120(100-109)	VEQMHEDIIS		mis-folded LAI rgp160	murine(IgG_{2a})

Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Abacioglu et al.(1994), Moore et al.(1994c)]
NOTES:
- B18: C1 region – epitope boundaries mapped by peptide scanning, HEDII core [Abacioglu et al.(1994)]
- B18: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]

257	MF39.1	gp120(C1 101-110 LAI)	gp120(100-109)	VEQMHEDIIS		Env	murine(IgG)

Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Thiriart et al.(1989), Cook et al.(1994), Moore et al.(1994c)]
NOTES:
- MF39.1: Called 39.1, and is probably the same as MF39.1 – MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]
- MF39.1: The relative affinity of denatured/native gp120 is 30 [Moore et al.(1994c)]

258	T2.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV		Env	murine(IgG)

Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Akerblom et al.(1990), Bolmstedt et al.(1990), Moore et al.(1994c), Moore et al.(1994d)]
NOTES:
- T2.1: The relative affinity for denatured/native gp120 is .27 – mutations 113 D/R, 106 E/A, and 117 D/A impair binding [Moore et al.(1994c)]

259	11/65	gp120(311-321 HXB10)	gp120(101-120)	EQMHEDIISLWDQSLKP-CVK		rgp120 BH10	rat(IgG_{2b})

Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [McKeating et al.(1992a)]
NOTES:
- 11/65: Binds only soluble gp120, not virion bound – used to quantitate gp120 shedding – (numbering is incorrect in original?) [McKeating et al.(1992a)]
- 11/65: UK Medical Research Council AIDS reagent: ARP3076

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
260	6D8	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV		IIIB-rgp120	rat

Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Dowbenko et al.(1988), Nakamura et al.(1992), Moore et al.(1994c)]
NOTES:
- 6D8: Highly cross reactive with multiple stains by rgp120 ELISA [Nakamura et al.(1992)]
- 6D8: The relative affinity for denatured/native gp120 is 15 – mutations 113 D/R and 113 D/A impair binding [Moore et al.(1994c)]

261	M96	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV	N	451 Env	rat(IgG_{2a})

Donor: Fulvia di Marzo Veronese
References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d)]
NOTES:
- M96: Immunoblot reactive for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)]
- M96: C1 region – the relative affinity for denatured/native gp120 is 6 [Moore et al.(1994c)]

262	37.1.1 (ARP 327)	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV		Env glycopro	murine(IgG)

Donor: Claudine Bruck
References: [Thiriart et al.(1989), Moore & Ho(1993), Moore et al.(1994c)]
NOTES:
- 37.1.1: Called 37.1 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- 37.1.1: The relative affinity for denatured/native gp120 is 8.6 – mutations 113 D/R (not D/A) and 117 K/W impair binding [Moore et al.(1994c)]
- 37.1.1: UK Medical Research Council AIDS reagent: ARP327

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
263	187.2.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV		Env glycopro	murine(IgG)

Donor: Claudine Bruck and Clothilde Thiriart
References: [Thiriart et al.(1989), Moore & Ho(1993), Cook et al.(1994), Moore et al.(1994c), Moore et al.(1994d)]
NOTES:

- 187.2.1: Called 187.1, and is probably the same as 187.2.1 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- 187.2.1: Called 187.1, and is probably the same as 187.2.1 – MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]
- 187.2.1: The relative affinity for denatured/native gp120 is 7 – mutations 113 D/A (not D/R) and 117 K/W impair binding [Moore et al.(1994c)]
- 187.2.1: UK Medical Research Council AIDS reagent: ARP332

264	MF58.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV		Env	murine(IgG)

Donor: Claudine Bruck and Clothilde Thiriart
References: [Thiriart et al.(1989), Moore et al.(1994c)]

265	MF77.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV		Env	murine(IgG)

Donor: Claudine Bruck and Clothilde Thiriart
References: [Thiriart et al.(1989), Moore et al.(1994c)]
NOTES:

- MF77.1: The relative affinity for denatured/native gp120 is 11 [Moore et al.(1994c)]

266	MF119.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV		Env	murine(IgG)

Donor: Claudine Bruck and Clothilde Thiriart
References: [Thiriart et al.(1989), Moore et al.(1994c)]
NOTES:

- MF119.1: The relative affinity for denatured/native gp120 is 30 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore et al.(1994c)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
267	MF4.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV		Env	murine(IgG)

Donor: Claudine Bruck and Clothilde Thiriart
References: [Thiriart et al.(1989), Moore et al.(1994c)]
NOTES:
- MF4.1: The relative affinity for denatured/native gp120 is 8 [Moore et al.(1994c)]

268	MF53.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK-PCV		Env	murine(IgG)

Donor: Claudine Bruck and Clothilde Thiriart
References: [Thiriart et al.(1989), Moore et al.(1994c)]
NOTES:
- MF53.1: The relative affinity for denatured/native gp120 is 10 [Moore et al.(1994c)]

269	GV1A8	gp120(105-113 IIIB)	gp120(104-112)	HEDIISLWD		gp120 complexed with MAb M77	murine

Donor: Claudine Bruck and Clothilde Thiriart
References: [Denisova et al.(1996)]
NOTES:
- GV1A8: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV7A4 and GV5H5 are homologous to GV1A8 and were generated in the same experiment [Denisova et al.(1996)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
270	135/9	gp120(C1 111-120 LAI)	gp120(110-119)	LWDQSLKPCV	L	IIIB gp120	murine(IgG_1)

Donor: Matthias Niedrig

References: [Niedrig et al.(1992b), Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996), Trkola et al.(1996a), Binley et al.(1997)]

NOTES:

- 135/9: Defines the epitope as gp120(114-123) MHEDIISLWD (core LWD?) – weak neutralization of lab strain [Niedrig et al.(1992b)]
- 135/9: The relative affinity for denatured/native gp120 is 15 – mutation 113 D/R impairs binding to native and denatured, 113 D/A only to denatured [Moore et al.(1994c)]
- 135/9: Substitutions 106 E/A, 113 D/A or R, and 117 K/W impair binding, some substitutions enhance binding [Moore et al.(1994d)]
- 135/9: Binding is enhanced by some anti-C1 and anti-C5 antibodies – enhances binding of some anti-V3, anti-C4 and anti-V2 MAbs – 135/9 binds to predicted alpha-helix in C1 [Moore & Sodroski(1996)]
- 135/9: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 135/9: A high avidity antibody as assessed by urea elution
- 135/9: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

271	MF46.1	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCV		Env	murine(IgG)

Donor: Matthias Niedrig

References: [Thiriart et al.(1989), Moore et al.(1994c)]

NOTES:

- MF46.1: The relative affinity for denatured/native gp120 is 8.5 [Moore et al.(1994c)]

272	C4	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCV		mis-folded LAI rgp160	murine(IgG_1)

Donor: George Lewis

References: [Abacioglu et al.(1994), Moore & Ho(1993), Moore et al.(1994c)]

NOTES:

- C4: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- C4: C1 region – epitope boundaries mapped by peptide scanning, BH10 core IISLW [Abacioglu et al.(1994)]
- C4: The relative affinity for denatured/native gp120 is 10 [Moore et al.(1994c)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
273	11	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCV		Env	murine(IgG)
		Donor: George Lewis **References:** [Thiriart et al.(1989), Moore et al.(1994c)] **NOTES:** • 11: The relative affinity for denatured/native gp120 is 7.8 – mutation 113 D/R impairs binding [Moore et al.(1994c)]					
274	12G10	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCV		Env	murine(IgG)
		Donor: George Lewis **References:** [Thiriart et al.(1989), Moore et al.(1994c)] **NOTES:** • 12G10: The relative affinity for denatured/native gp120 is 17 – mutation 117 K/W impairs binding [Moore et al.(1994c)]					
275	7C10	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCV		Env	murine(IgG)
		Donor: George Lewis **References:** [Thiriart et al.(1989), Moore et al.(1994c)] **NOTES:** • 7C10: The relative affinity for denatured/native gp120 is 5.8 – mutation 117 K/W impairs binding [Moore et al.(1994c)]					
276	W1	gp120(C1 102-121 LAI)	gp120(101-120)	EQMHEDIISLWDQSLKP-CVK		Env	murine(IgG)
		Donor: D. Weiner, U. Penn. **References:** [Moore et al.(1994c)] **NOTES:** • W1: The relative affinity for denatured/native gp120 is 6 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore et al.(1994c)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
277	T11	(C1 102-125)	gp120(101-124)	EQMHEDIISLWDQSLKP-CVKLTPL		rec gp140	murine

Donor: R. Doms, Univ. of Pennsylvania
References: [Earl et al.(1994), Jagodzinski et al.(1996)]
NOTES:
- T11: Generated during a study of the influence of the oligomeric structure of Env in determining the repertoire of the Ab response – an oligomer with no gp120/gp41 cleavage site was used as the immunogen [Earl et al.(1994)]
- T11: The sulfated polysaccharide, curdlan sulfate (CRDS), binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop from gp120 results in more potent T11 inhibition by CRDS [Jagodzinski et al.(1996)]

278	B33	gp120(V2 123-142 LAI)	gp120(122-146)	TPLCVSLKCTDLGNATN-TNS	N	Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	murine($IgG_{2b\kappa}$)

Donor: Daniels
References: [Abacioglu et al.(1994), Bristow et al.(1994)]
NOTES:
- B33: There are two MAbs in the literature named B33. See also gp41, LAI 123-142 [Abacioglu et al.(1994)]
- B33: MAbs generated in the context of a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]
- B33: UK Medical Research Council AIDS reagent: ARP304, gp160/41 binding

279	6D5	gp120(V2 122-141 LAI)	gp120(121-145)	LTPLCVSLKCTDLKNDT-NTN		Env	murine(IgG)

Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA
References: [Moore et al.(1994c), Moore et al.(1994d)]
NOTES:
- 6D5: The relative affinity for denatured/native gp120 is 15 – mutations Δ119-205 and 125 L/G impair binding [Moore et al.(1994c)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
280	C108G	gp120(V2 162-169 HXB2)	gp120(166-173)	STSIRGKV	L	IIIB infection	chimpanzee($IgG_{1\kappa}$)

Donor: S. Tilley, Public Health Research Institute, NY, NY

References: [Warrier et al.(1994), Wu et al.(1995), Warrier et al.(1995), Warrier et al.(1996), Ugolini et al.(1997), Mondor et al.(1998), Alsmadi & Tilley(1998)]

NOTES:

- C108G: High affinity, potent neutralization of HIV-1 IIIB – binding not affected by reduction of disulfide bonds – binding disrupted by removal of N-linked glycans – peptide binding lower affinity than glycosylated Env [Warrier et al.(1994)]
- C108G: Strain specificity: LAI, Bal, HXB2 – conformational character – glycosylation site at 160 critical – mutation of conserved glycosylation site at 156 increased epitope exposure [Wu et al.(1995)]
- C108G: Characterization of MAb variable region [Warrier et al.(1995)]
- C108G: Synergistic neutralization of HIV-1 when combined with anti-V3 MAbs 0.5β and C311E, or anti-CD4BS MAbs, 1125H and 5145A – neutralization further enhanced by presence of both 1125H and 0.5β [Warrier et al.(1996)]
- C108G: Viral binding inhibition by C108G was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- C108G: Inhibits HX10 binding to both CD4 positive and negative HeLa cells[Mondor et al.(1998)]
- C108G: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against only IIIB – this is first demonstration of ADCC directed by a V2 specific MAb [Alsmadi & Tilley(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
281	10/76b	gp120(V2 162-171 BH10)	gp120(166-174)	STSIRGKVQ	L (HXB10)	BH10 rgp120	rat(IgG_{2a})

Donor: S. Tilley, Public Health Research Institute, NY, NY

References: [McKeating et al.(1993b), McKeating et al.(1993a), Shotton et al.(1995), Wu et al.(1995), McKeating et al.(1996)]

NOTES:

- 10/76b: R to L substitution abrogated binding – human sera recognize epitope [McKeating et al.(1993b)]
- 10/76b: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton et al.(1995)]
- 10/76b: Included in cross-competition and neutralization studies [Shotton et al.(1995)]
- 10/76b: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)]
- 10/76b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]
- 10/76b: UK Medical Research Council AIDS reagent: ARP3077

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
282	11/4c	gp120(V2 162-171)	gp120(166-174)	STSIRGKVQ	L (HXB2)	BH10 rgp120	rat(IgG_{2a})

Donor: S. Tilley, Public Health Research Institute, NY, NY
References: [McKeating et al.(1993b), Wu et al.(1995), Shotton et al.(1995)]
NOTES:
- 11/4c: R to L substitution abrogated binding – human sera recognize epitope [McKeating et al.(1993b)]
- 11/4c: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)]
- 11/4c: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton et al.(1995)]
- 11/4c: UK Medical Research Council AIDS reagent: ARP3035

283	11/41e	gp120(V2 162-171)	gp120(166-174)	STSIRGKVQ	L (HXB10)	rgp120 LAI:BH10	rat(IgG_1)

Donor: S. Tilley, Public Health Research Institute, NY, NY
References: [McKeating et al.(1993b), Shotton et al.(1995), Wu et al.(1995)]
NOTES:
- 11/41e: R to L abrogated binding – human sera recognize the epitope [McKeating et al.(1993b)]
- 11/41e: Included in cross-competition and neutralization studies [Shotton et al.(1995)]
- 11/41e: HX10 strain specificity – binds native and deglycosylated gp120 [Wu et al.(1995)]

284	11/4b	gp120(V2 162-171)	gp120(166-174)	STSIRGKVQ	L (HXB10)	rgp120 LAI:BH10	rat(IgG_{2a})

Donor: S. Tilley, Public Health Research Institute, NY, NY
References: [McKeating et al.(1993b), Shotton et al.(1995), Wu et al.(1995), Moore & Sodroski(1996)]
NOTES:
- 11/4b: A change from R to L abrogated binding – human sera recognize epitope [McKeating et al.(1993b)]
- 11/4b: Cross-competes with MAbs 10/76b and 11/4c – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton et al.(1995)]
- 11/4b: HXB10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)]
- 11/4b: Linear V2 epitope – reciprocal binding enhancement of anti-V2 discontinuous epitope antibodies (in contrast to BAT085) and CD4 inducible antibody 48d. Reciprocal inhibits BAT085 binding – inhibits CRA-3 binding CRA-3 does not inhibit 11/4b [Moore & Sodroski(1996)]

285	RSD-33	gp120(V2 162-171 BH10)	gp120(166-174)	STSIRGKVQ		BH10 gp120	

Donor: R. Daniels (NIMR, UK)
References: [Moore et al.(1993a)]

286	6C4/S	gp120(V2 162-170 BH10)	gp120(166-173)	STSIRGKV		BH10 gp120	

Donor: S. Ranjbar (NIBSC, UK)
References: [Moore et al.(1993a)]
NOTES:
- 6C4/S: UK Medical Research Council AIDS reagent: ARP3049

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
287	G3-4	gp120(V2 170-180 BH10)	gp120(174-184)	QKEYAFFYKLD	L	IIIB gp120	murine($IgG_{2b\kappa}$)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Ho et al.(1991a), Ho et al.(1992), Fung et al.(1992), McKeating et al.(1992a), Moore & Ho(1993), Sullivan et al.(1993), Sattentau et al.(1993), Thali et al.(1993), Moore et al.(1993a), Moore et al.(1994b), Gorny et al.(1994), Thali et al.(1994), Yoshiyama et al.(1994), Wu et al.(1995), Sattentau & Moore(1995), Jagodzinski et al.(1996), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Stamatatos et al.(1997), Ditzel et al.(1997), Wyatt et al.(1997), Parren et al.(1998)]

NOTES:

- G3-4: Also called G3.4
- G3-4: Binding is sensitive to removal of glycans by endo H – 50% neutralization of 4/9 primary isolates – has conformational features [Ho et al.(1991a)]
- G3-4: Neutralizes IIIB and RF, not MN – blocks sCD4-gp120, not as potent as MAb 15e – V2 binding MAbs BAT085 and G3-136 block G3-4 gp120 binding – sensitive to reduction of gp120 by DTT [Ho et al.(1992)]
- G3-4: Substitutions in residues 176 to 184 affect MAb recognition – substitutions in V2 can result in gp120-gp41 dissociation [Sullivan et al.(1993)]
- G3-4: Increased binding in the presence of sCD4 [Sattentau et al.(1993)]
- G3-4: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- G3-4: V2 region, marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore et al.(1993a)]
- G3-4: Conformationally sensitive – sporadic cross-reactivity among, and outside, B clade gp120s [Moore et al.(1994b)]
- G3-4: Weakly neutralizing, IC_{50} = 53 μg/ml [Gorny et al.(1994)]
- G3-4: gp41 mutation (582 A/T) that reduces neutralization of anti-CD4 binding site MAbs does not alter G3-4s ability to neutralize [Thali et al.(1994)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)

287 cont.

NOTES:

- G3-4: Neutralizes RF – substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity and result in neutralization escape [Yoshiyama et al.(1994)]
- G3-4: Reactive with BH10, RF, and MN – binds native, but not denatured or deglycosylated gp120, binds to deglycosylated V1V2 fusion protein, suggesting importance of glycans outside the V1V2 region [Wu et al.(1995)]
- G3-4: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes Hx10 cell-free virus [Sattentau & Moore(1995)]
- G3-4: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop from gp120 results in more potent G3-4 binding inhibition by CRDS – G3-4 epitope described as 176-184 FYKLDIIPI and 191-193 YSL [Jagodzinski et al.(1996)]
- G3-4: Binding enhanced by selected antibodies to C1, C4, C5, V3 and anti-CD4 binding site MAbs – enhances binding of selected V3, C4 and anti-CD4 binding site MAbs [Moore & Sodroski(1996)]
- G3-4: Described epitope as STSIRGKVKEYAFFYKLDI – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT085 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50-69, in contrast to anti-V3 MAbs [Poignard et al.(1996a)]
- G3-4: A low avidity antibody as assessed by urea elution
- G3-4: Called G3.4 – mediates gp120 virion dissociation in contrast to anti-V2 MAb G3-136 – not neutralizing for SF162 or SF128A in either primary macrophages or PBMC [Stamatatos et al.(1997)]
- G3-4: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)]
- G3-4: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
288	BAT085	gp120(V2 170-180 IIIB)	gp120(175-184)	KEYAFFYKLD	L	Inact IIIB	murine(IgG_1)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Fung et al.(1987), Fung et al.(1992), Moore & Ho(1993), Pirofski et al.(1993), Thali et al.(1993), Moore et al.(1993a), D'Souza et al.(1994), Moore et al.(1994d), Gorny et al.(1994), Yoshiyama et al.(1994), Wu et al.(1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Ditzel et al.(1997), Parren et al.(1998)]

NOTES:

- BAT085: Also called BAT-085
- BAT085: V2 region – sCD4 does not block – neutralizes IIIB and some primary isolates, but not MN or RF – binds MN – deglycosylation or DDT reduction of gp120 does not diminish reactivity [Fung et al.(1992)]
- BAT085: Called BAT-85 – conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- BAT085: 7/8 V2 murine MAbs required gp120 native structure to bind, but BAT085 was the exception – type-specific [Moore et al.(1993a)]
- BAT085: Peptide affinities of G3-136 and G3-4 are 100-fold less than BAT085, but BAT085 has lower affinity for BH10 gp120 and is weaker at neutralization [Moore et al.(1993a)]
- BAT085: Multi-lab study for antibody characterization and assay comparison – did not bind MN or SF2 [D'Souza et al.(1994)]
- BAT085: Interacts with two overlapping peptides with region of overlap KEYAFFYKLD [Gorny et al.(1994)]
- BAT085: Neutralizes RF – substitution 177 Y/H in the V2 loop of RF does not inhibit neutralization, in contrast to MAbs G3-4 and SC258 [Yoshiyama et al.(1994)]
- BAT085: HXB10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)]
- BAT085: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)]
- BAT085: Binding is blocked by other V2 region antibodies, enhanced by several anti-C1 MAbs, and anti-V3 MAb G511 – reciprocal enhancement of CD4i MAb 48d binding [Moore & Sodroski(1996)]
- BAT085: Epitope suggested to be QKEYAFFYKLD – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT123 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50-69, in contrast to anti-V3 MAbs [Poignard et al.(1996a)]
- BAT085: An antibody with moderate avidity as assessed by urea elution
- BAT085: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
289	G3-136	gp120(V2 170-180 IIIB)	gp120(174-184)	QKEYAFFYKLD	L	purified IIIB gp120	murine(IgG)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Fung et al.(1992), Pirofski et al.(1993), Thali et al.(1993), Moore & Ho(1993), Moore et al.(1993a), Yoshiyama et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Stamatatos et al.(1997), Ditzel et al.(1997), Wyatt et al.(1997), Parren et al.(1998)]

NOTES:

- G3-136: V2 region – binds and neutralizes IIIB and RF in CEM-SS cells, but not MN – neutralization activity against a few primary isolates in PBMC – sCD4 binding inhibits binding (contrast with BAT085) – deglycosylation or reduction of gp120 by DTT diminishes reactivity [Fung et al.(1992)]
- G3-136: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- G3-136: Marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore et al.(1993a)]
- G3-136: Binding enhanced by selected antibodies to C1, C4, C5, V3 and anti-CD4 binding site MAbs – enhances binding of selected V3, C4 and anti-CD4 binding site MAbs [Moore et al.(1993a)]
- G3-136: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity [Yoshiyama et al.(1994)]
- G3-136: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)]
- G3-136: Described epitope as STSIRGKVKEYAFFYKLDI – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT123 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50-69, in contrast to anti-V3 MAbs [Poignard et al.(1996a)]
- G3-136: A low avidity antibody as assessed by urea elution
- G3-136: Called G3.136 – does not mediate gp120 virion dissociation in contrast to anti-V2 MAb G3-4 – not neutralizing for SF162 or SF128A in either primary macrophages or PBMC [Stamatatos et al.(1997)]
- G3-136: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)]
- G3-136: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

290	38/12b	gp120(V2 172-191 HXB2)	gp120(176-195)	EYAFFYKLDIIPIDNDT-TSY		BH10 gp120	rat

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Wu et al.(1995)]

NOTES:

- 38/12b: Broad specificity: HXB2, MN, SF162 – binds native and deglycosylated gp120 [Wu et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
291	697-D	gp120(V2 161-180 IIIB)	gp120(165-184)	ISTSIRGKVQKEYAFFY-KLD	P (weak)	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Cellular Products Inc, Buffalo NY
References: [Gorny et al.(1994), Forthal et al.(1995), Moore & Ho(1995), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997), Parren et al.(1997b)]
NOTES:

- 697-D: Also called 697D
- 697-D: Conformational with weak reactivity to V2 peptide ISTSIRGKVQKEYAFFYKLD – neutralized 3/4 primary isolates, but none of 4 lab strains – V2 substitutions 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS abrogate binding – anti-C4 MAbs G3-536 and G45-60 enhance binding – mild oxidation of carbohydrate moieties inhibits binding [Gorny et al.(1994)]
- 697-D: Not neutralizing, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]
- 697-D: Review: called 697/30D – neutralizes some primary, but not lab adapted strains [Moore & Ho(1995)]
- 697-D: Partial inhibition of gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 697-D: A low avidity antibody as assessed by urea elution
- 697-D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 697-D bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- 697D: Does not neutralize TCLA strains but neutralizes some primary isolates weakly [Parren et al.(1997b)]

292	12b	gp120(V2 162-181)	gp120(166-185)	STSIRGKVQKEYAFFYK-LDI	L (HXB10)	BH10 rgp120	rat(IgG_{2a})

Donor: Cellular Products Inc, Buffalo NY
References: [Shotton et al.(1995), McKeating et al.(1996)]
NOTES:

- 12b: V2 MAb neutralized HXB2 – position 179-180 LD to DL abrogates binding – competes with 60b, but not 74 [Shotton et al.(1995)]
- 12b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]

293	38/60b	gp120(V2 172-191 HXB2)	gp120(176-195)	EYAFFYKLDIIPIDNDT-TSY		BH10 gp120	rat

Donor: Cellular Products Inc, Buffalo NY
References: [Wu et al.(1995)]
NOTES:

- 38/60b: Strain specificity: HXB2 – binds native and deglycosylated gp120 [Wu et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
294	60b	gp120(V2 172-181 HXB2)	gp120(176-185)	EYAFFYKLDI	N	BH10 rgp120	rat(IgG_{2b})
		Donor: Cellular Products Inc, Buffalo NY **References:** [Shotton et al.(1995)] **NOTES:** • 60b: V2 MAb did not neutralize HXB2 – bound to rgp120 in ELISA – substitutions 179-180 LD/DL and 191-193 YSL/GSS abrogate binding, as do changes outside the minimum epitope – competes with 12b, but not 74 [Shotton et al.(1995)]					
295	74	gp120(V2 172-181)	gp120(176-185)	EYAFFYKLDI	N	BH10 rgp120	rat(IgG_1)
		Donor: Cellular Products Inc, Buffalo NY **References:** [Shotton et al.(1995)] **NOTES:** • 74: V2 MAb did not neutralize HXB2 – did not bind rgp120 ELISA – position 179-180 LD to DL abrogates binding, as do changes outside the minimum epitope – does not compete with 60b or 12b, and is enhanced by two conformation dependent MAbs [Shotton et al.(1995)]					
296	1088	gp120(V2)	gp120				
		Donor: Cellular Products Inc, Buffalo NY **References:** [Berman et al.(1997)] **NOTES:** • 1088: Binds weakly to 2/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)]					
297	3D3.B8	gp120(211-220 LAI)	gp120(215-225)	EPIPIHYCAPA		Env glycopro	murine(IgG)
		Donor: Cellular Products Inc, Buffalo NY **References:** [Bolmstedt et al.(1990), Moore et al.(1994c)] **NOTES:** • 3D3.B8: The relative affinity denatured/native gp120 is greater than 10 [Moore et al.(1994c)]					
298	4C11.D8	gp120(211-220 LAI)	gp120(215-225)	EPIPIHYCAPA		Env glycopro	murine(IgM)
		Donor: Cellular Products Inc, Buffalo NY **References:** [Bolmstedt et al.(1990), Moore et al.(1994c)] **NOTES:** • 4C11.D8: The relative affinity denatured/native gp120 is greater than 10 [Moore et al.(1994c)]					
299	322-151	gp120(201-220 LAI)	gp120(215-225)	EPIPIHYCAPA		Env glycopro	murine(IgG)
		Donor: G. Robey, Abbot Labs **References:** [Moore et al.(1994c), Moore et al.(1994d)] **NOTES:** • 322-151: The relative affinity denatured/native gp120 is 30 [Moore et al.(1994c)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
300	110.1	gp120(200-217)	gp120(216-225)	PIPIHYCAPA?	N	Env glycoprotein	human
		Donor: G. Robey, Abbot Labs **References:** [Pincus & McClure(1993), Pincus et al.(1996), Valenzuela et al.(1998)] **NOTES:** • 110.1: There is another antibody with this ID that binds to Env at positions 491-500 in LAI, see [Gosting et al.(1987)] • 110.1: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding – 110.1-RAC did not mediate cell killing, and sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)]					
301	493-156	gp120(211-230 LAI)	gp120(215-234)	EPIPIHYCAPAGFAILK-CNN		Env glycopro	murine(IgG)
		Donor: G. Robey, Abbot Labs **References:** [Moore et al.(1994c)] **NOTES:** • 493-156: The relative affinity denatured/native gp120 is >10 [Moore et al.(1994c)]					
302	GV4H3	gp120(219-226 IIIB)	(223-230)	APAGFAIL		gp120 complexed with MAb M77	murine
		Donor: G. Robey, Abbot Labs **References:** [Denisova et al.(1996)] **NOTES:** • GV4H3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes [Denisova et al.(1996)]					
303	J1	gp120(222-231 LAI)	gp120(226-235)	GFAILKCNNK		peptide	murine(IgG_1)
		Donor: J. Hoxie, U. Penn. **References:** [Moore et al.(1994c), Moore et al.(1994d), Cook et al.(1994)] **NOTES:** • J1: The relative affinity denatured/native gp120 is 30 [Moore et al.(1994c)] • J1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
304	J3	gp120(222-231 LAI)	gp120(226-235)	GFAILKCNNK		peptide	murine(IgG_1)

Donor: J. Hoxie, U. Penn.
References: [Moore et al.(1994c), Cook et al.(1994)]
NOTES:
- J3: The relative affinity denatured/native gp120 is 30 [Moore et al.(1994c)]
- J3: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]

305	MF87.1	gp120(242-261 LAI)	gp120(256-265)	RPVVSTQLLL		Env	murine(IgG)

Donor: J. Hoxie, U. Penn.
References: [Thiriart et al.(1989), Moore et al.(1994c)]
NOTES:
- MF87.1: The relative affinity denatured/native gp120 is 10 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore et al.(1994c)]

306	MF169.1	gp120(242-261 LAI)	gp120(256-265)	RPVVSTQLLL		Env	murine(IgG)

Donor: J. Hoxie, U. Penn.
References: [Thiriart et al.(1989), Moore et al.(1994c), Moore et al.(1994d)]
NOTES:
- MF169.1: The relative affinity denatured/native gp120 is 11 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore et al.(1994c)]

307	MF170.1	gp120(242-261 LAI)	gp120(256-265)	RPVVSTQLLL		Env	murine(IgG)

Donor: J. Hoxie, U. Penn.
References: [Thiriart et al.(1989), Moore et al.(1994c), Moore et al.(1994d)]
NOTES:
- MF170.1: The relative affinity denatured/native gp120 is 15 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding to denatured and native gp120, and 262N/T, 269 E/L and 281 A/V to only native gp120 [Moore et al.(1994c)]

308	213.1	gp120(242-261 LAI)	gp120(256-265)	RPVVSTQLLL		Env glycopro	murine(IgG1)

Donor: Claudine Bruck
References: [Thiriart et al.(1989), Moore & Ho(1993), Moore et al.(1994c)]
NOTES:
- 213.1: Bound preferentially to denatured IIIB and SF2 gp120 [Moore & Ho(1993)]
- 213.1: The relative affinity denatured/native gp120 is 100 – mutations 252 R/W, 257 T/G or T/R impair binding [Moore et al.(1994c)]
- 213.1: UK Medical Research Council AIDS reagent: ARP334

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
309	M89	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQLLLNGSLAEE-EVV	N	451 Env	murine(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d)]
NOTES:
- M89: Immunoblot reactive, RIP negative, for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)]
- M89: C2 region – the relative affinity for denatured/native gp120 is >30 – mutations 257 T/R and 269 E/L impair binding [Moore et al.(1994c)]

310	B12	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQLLLNGSLAEE-EVV		mis-folded LAI rgp160	murine(IgG)

Donor: Fulvia di Marzo Veronese
References: [Moore et al.(1994c)]
NOTES:
- B12: C2 region – the relative affinity for denatured/native gp120 is 27 – mutations 257 T/R and 262 N/T impair binding [Moore et al.(1994c)]

311	B13	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQLLLNGSLAEE-EVV		mis-folded LAI rgp160	murine(IgG_{2a})

Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA
References: [Pincus & McClure(1993), Moore & Ho(1993), Moore et al.(1994c), Abacioglu et al.(1994), Moore et al.(1994d), Pincus et al.(1996), Connor et al.(1998)]
NOTES:
- B13: Also called Bh13
- B13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- B13: The relative affinity for denatured/native gp120 is 30 – mutations 257 T/R and 269 E/L impair binding [Moore et al.(1994c)]
- B13: C2 region – epitope boundaries mapped by peptide scanning, core epitope: TQLLLN [Abacioglu et al.(1994)]
- B13: Called Bh13 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)]

312	B24	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLLN		mis-folded LAI rgp160	murine(IgG_{2a})

Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA
References: [Abacioglu et al.(1994)]
NOTES:
- B24: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
313	B3	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLLN		mis-folded LAI rgp160	murine(IgG_1)
		Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA **References:** [Abacioglu et al.(1994)] **NOTES:** • B3: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
314	B21	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLLN		mis-folded LAI rgp160	murine(IgG_1)
		Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA **References:** [Abacioglu et al.(1994)] **NOTES:** • B21: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
315	B23	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLLN		mis-folded LAI rgp160	murine(IgG_{2a})
		Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA **References:** [Abacioglu et al.(1994)] **NOTES:** • B23: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
316	B25	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLLN		mis-folded LAI rgp160	murine(IgG_1)
		Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA **References:** [Abacioglu et al.(1994)] **NOTES:** • B25: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
317	B29	gp120(C2 257-263 BH10)	gp120(261-267)	TQLLLNG		mis-folded LAI rgp160	murine(IgG_{2a})
		Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA **References:** [Abacioglu et al.(1994)] **NOTES:** • B29: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
318	B26	gp120(C2 257-263 BH10)	gp120(261-267)	TQLLLNG		mis-folded LAI rgp160	murine(IgG_1)
		Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA **References:** [Abacioglu et al.(1994)] **NOTES:** • B26: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
319	B36	gp120(C2 257-263 BH10)	gp120(261-267)	TQLLLNG		mis-folded LAI rgp160	murine(IgG_1)
		Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA **References:** [Abacioglu et al.(1994)] **NOTES:** • B36: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
320	C13	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQLLLNGSLAEE-EVV		mis-folded LAI rgp160	murine(IgG_1)
		Donor: George Lewis **References:** [Moore & Ho(1993), Moore et al.(1994c), Abacioglu et al.(1994)] **NOTES:** • C13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • C13: The relative affinity for denatured/native gp120 is 36 – mutations 257 T/R, 267 E/L, and 269 E/L impair binding [Moore et al.(1994c)] • C13: Epitope boundary extended to RPVVSTQLLLNGSLAEEEVVIR, to take into account the effect of a point mutation [Abacioglu et al.(1994)] • C13: NIH AIDS Research and Reference Reagent Program: 1209					
321	110.E	gp120(C2 262-281 LAI)	gp120(266-285)	NGSLAEEEVVIRSVNFT-DNA		Env glycopro	murine(IgG)
		Donor: F. Traincard **References:** [Moore et al.(1994c), Moore et al.(1994d)] **NOTES:** • 110.E: The relative affinity for denatured/native gp120 is 7.3 [Moore et al.(1994c)]					
322	110.C	gp120(C2 261-280 LAI)	gp120(275-284)	VIRSVNFTDN		Env glycopro	murine(IgG)
		Donor: F. Traincard, Hydridolabs, Institut Pasteur **References:** [Moore et al.(1994c), Moore et al.(1994d), Valenzuela et al.(1998)] **NOTES:** • 110.C: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)] • 110.C: Only slightly reduces LAI viral binding or entry into CEM cells [Valenzuela et al.(1998)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
323	IIIB-V3-21	gp120(V3 299-304 IIIB)	gp120(298-303)	INCTRP	N	Peptide	murine(IgG_1)

Donor: J. Laman
References: [Laman et al.(1992), Laman et al.(1993), Valenzuela et al.(1998)]
NOTES:
- IIIB-V3-21: Also called V3-21
- IIIB-V3-21: Binds to the base of the V3 loop on denatured gp120 [Laman et al.(1992)]
- IIIB-V3-21: Binds to NP40 treated gp120, and epitope is probably obscured by local glycosylation [Laman et al.(1993)]
- IIIB-V3-21: Does not block HIV-1 LAI binding or entry into CEM cells [Valenzuela et al.(1998)]
- IIIB-V3-21: UK Medical Research Council AIDS reagent: ARP3048
- IIIB-V3-21: NIH AIDS Research and Reference Reagent Program: 1725

324	IIIB-V3-26	gp120(V3 299-304 IIIB)	gp120(295-311)	SVEINCTRPNNNTRKSI	N	Peptide	murine(IgG_1)

Donor: J. Laman
References: [Laman et al.(1992)]
NOTES:
- IIIB-V3-26: Binds to the base of the V3 loop on denatured gp120 [Laman et al.(1992)]

325	MO97/V3	gp120(V3 299-308 IIIB)	gp120(303-312)	PNNNTRKSIR	N	rpB1 (IIIB Env 286-467)	human(IgM)

Donor: J. Laman
References: [Ohlin et al.(1992)]
NOTES:
- MO97: Generated through *in vitro* “immunization” of uninfected-donor lymphocytes [Ohlin et al.(1992)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
326	8/38c	gp120(V3 300-315 HXB10)	gp120(304-317)	NNNTRKRIRIQRGPGR	L	rBH10 gp120	rat(IgG_{2a})

Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK
References: [McKeating et al.(1992a), Sattentau & Moore(1995), Jeffs et al.(1996), Parren et al.(1998)]
NOTES:

- 8/38c: Also called 8/38/1c
- 8/38c: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating et al.(1992a)]
- 8/38c: Binds equally well to monomer and oligomer, less rapid association rate than other anti-V3 antibodies, and an associated less potent neutralization of lab strains [Sattentau & Moore(1995)]
- 8/38c: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)]
- 8/38c: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- 8/38c: UK Medical Research Council AIDS reagent: ARP3039

327	8/64b	gp120(V3 300-315 HXB10)	gp120(304-317)	NNNTRKRIRIQRGPGR	L	rBH10 gp120	rat(IgM)

Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK
References: [McKeating et al.(1992a)]
NOTES:

- 8/64b: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating et al.(1992a)]
- 8/64b: UK Medical Research Council AIDS reagent: ARP3036

328	polyclonal	gp120(V3 IIIB)	gp120(305-328)	NNTRKSIRIQRGPGRAF-VTIGKIGN	L	oral immunization – peptide plus cholera toxin adjuvant	murine(IgA)

Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK
References: [Bukawa et al.(1995)]
NOTES:

- Polyclonal secretory IgA antibody raised by mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
329	polyclonal	gp120(V3 IIIB)	gp120(305-328)	NNTRKSIRIQRGPGRAF-VTIGKIGN	L	DNA vaccine IIIB env + rev	murine(IgA22a)
		Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK **References:** [Sasaki et al.(1998)] **NOTES:** • The env response is what is being sought, but co-expression of Rev is required – intramuscular versus nasal vaccination with DNA vaccine with a QS-21 adjuvant was studied – QS-21 enhanced the IgG2a response mediated via Th1 cytokines IFNγ and IL-2 [Sasaki et al.(1998)]					
330	polyclonal	gp120(V3 MN)	gp120(304-328)	CNYNKRKRIHIGPGRAF-YTTKNIIGTIC	L		rabbit(IgA and IgG)
		Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK **References:** [FitzGerald et al.(1998)] **NOTES:** • Polyclonal response to MN, or Thai E V3 loop inserted into *Pseudomonas* Exotoxin for vaccination – inserts of 14 or 26 amino acids were used from MN or a Thai E strain, constrained by disulfide bond – sera from vaccinated rabbit were reactive with strain-specific gp120 – administration to mucosal surfaces elicits IgA [FitzGerald et al.(1998)]					
331	polyclonal	gp120(V3 IIIB)	gp120(305-325)	CNNTRKSIRIQRGPGRA-FVTIGK	L	?	Guinea pig IgG
		Donor: D. Bolognesi and T. Matthews, Duke University **References:** [Allaway et al.(1993)] **NOTES:** • Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
332	1324E	gp120(V3 E clade)	gp120(307-312)	TRTSVR	L	HIV-1 E clade infection	human($IgG_{1\kappa}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Gorny et al.(1998)]
NOTES:
- A hu MAb derived from an HIV-1 E subtype infected service man from the US who had served in Thailand – cross-reactive with V3 peptides, rgp120, and monomeric gp120 from E, C and A clades, as well as cells infected with a C-clade primary isolate, but not with B and D clade V3 peptides or rgp120 – neutralizes E clade virus adapted for growth in H9 cells, but not 5 primary E clade isolates, including the autologous isolate – kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 1324E was comparable to 447-52D [Gorny et al.(1998)]

333	MO99/V3	gp120(V3 304-308 IIIB)	gp120(308-312)	RKSIR	N	rpB1 (IIIB Env 286-467)	human(IgM)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Ohlin et al.(1992)]
NOTES:
- MO99: Generated through *in vitro* "immunization" of uninfected-donor lymphocytes [Ohlin et al.(1992)]

334	DO 142-10	gp120(V3 MN)	gp120(309-322)	KRIHIGPGRAFYTT		HIV-1 infection	human

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Seligman et al.(1996)]
NOTES:
- DO 142-10: Fab fragment – competition ELISAs with serial deletions defined the epitope KRIHIGPGRAFYTT [Seligman et al.(1996)]

335	polyclonal	gp120(V3 C subtype)	gp120(310-321)	CKRKIHIGPGQAFYT		Peptide-ISCOM	murine($IgG_{2a,b}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Ahluwalia et al.(1997)]
NOTES:
- A V3 loop peptide modified to resemble an Indian form (GPGQ) was incorporated into ISCOMS (immune stimulating complexes) or liposomes, and used to immunize mice – the IgG2a/IgG2b antibody response was enhanced by the presentation in the ISCOM suggestive of a Th1 response

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
336	polyclonal	gp120(V3 MN)	gp120(308-322)	RKRIHIGPGRAFYTT	L (MN ALA-1)	HIV-1 infection	human
		Donor: Susan Zolla-Pazner (NYU Med. Center) **References:** [Spear et al.(1994)] **NOTES:** • 40% of antibody in serum that can bind to native viral proteins on MN-infected cells can be blocked by the peptide RKRIHIGPGRAFYTT, which can also block 75-95% of the complement activation on HIV infected cells [Spear et al.(1994)]					
337	polyclonal	gp120(V3 MN)	gp120(313-324)	IGPGRAFYTTKN	L (MN ALA-1)	IGPGRAFYTTKN HRV14: HIV-1 chimera	guinea pigs
		Donor: Susan Zolla-Pazner (NYU Med. Center) **References:** [Smith et al.(1998)] **NOTES:** • The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunos-elected, and chimeric viruses were neutralized by anti-V3 loop antibodies – chimeric viruses elicited potent NAbs against ALA-1 and MN [Smith et al.(1998)]					
338	TH1	gp120(V3)		unk	L (MN,JRCSF)		human($IgG_{1\lambda}$)
		Donor: Michael Fung, Tanox Biosystem, USA **References:** [D'Souza et al.(1995), Yang et al.(1998)] **NOTES:** • TH1: Found to neutralize MN and JRCSF, but not two B subtype primary isolates, nor a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs [D'Souza et al.(1995)] • TH1: A neutralization assay was developed based on heminested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang et al.(1998)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
339	D47	(V3 IIIB)		unk		IIIB vaccinia expressed Env	murine

Donor: Michael Fung, Tanox Biosystem, USA
References: [Richardson Jr et al.(1996), Wyatt et al.(1997), Earl et al.(1997), Otteken et al.(1996)]
NOTES:

- D47: Used for capture of oligomeric Env for antigen capture ELISA – binding of this antibody to oligomeric Env IIIB was not blocked by human sera from the US, consistent with a low prevalence of IIIB-like V3 strains [Richardson Jr et al.(1996)]
- D47: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)]
- D47: Used for comparison in a study of gp41 antibodies – D47 binds to a greater extent to cell surface expressed Env than any of 38 conformation dependent anti-gp41 MAbs [Earl et al.(1997)]
- D47: Pulse label experiments of MAb binding to noncleavable gp160 revealed that this anti-V3 MAb bound immediately and binding stayed constant through chase period [Otteken et al.(1996)]

340	F19.48-3	gp120(V3 312-324 LAI)	gp120(309-321)	IRIQRGPGRAFVT	L	IIIB rgp120 294-474	murine($IgG_{2a\kappa}$)

Donor: ?
References: [Boudet et al.(1994)]
NOTES:

- F19.48-3: Strain specific – used to raise anti-idiotype antibodies [Boudet et al.(1994)]

341	F19.26-4	gp120(V3 312-324 LAI)	gp120(309-321)	IRIQRGPGRAFVT	L	IIIB rgp120 294-474	murine($IgG_{2a\kappa}$)

Donor: ?
References: [Boudet et al.(1994)]
NOTES:

- F19.26-4: Strain specific – used to raise anti-idiotype antibodies [Boudet et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
342	F19.57-11	gp120(V3 312-324 LAI)	gp120(309-321)	IRIQRGPGRAFVT	L (LAI)	IIIB rgp120 294-474	murine($IgG_{1\kappa}$)
		Donor: ? **References:** [Boudet et al.(1991), Boudet et al.(1994), Boudet et al.(1995)] **NOTES:** • F19.57-11: MAb F19.57-11 is strain specific for LAI – used to raise anti-idiotype rabbit antibodies (called 57-B Ab2) [Boudet et al.(1994)] • F19.57-11: Anti-anti-idiotypic antibodies (Ab3) were raised in BALB/c mice that had greater breadth of reactivity than the original F19.57-11 (Ab3 could also recognize 1282 and SF2, with aa TRK(R or S)IYIGPGRA(WY or FH)T) [Boudet et al.(1995)]					
343	M096/V3	gp120(309-318 + 329-338)	gp120	IQRGPGRAFV + AHCN-ISRAKW		rIIIB Env 286-467	human(IgM)
		Donor: ? **References:** [Ohlin et al.(1992)] **NOTES:** • M096: Generated through *in vitro* “immunization” of uninfected-donor lymphocytes [Ohlin et al.(1992)]					
344	MO101/-V3,C4	gp120(314-323 + 494-503)	gp120	GRAFVTIGKI + LGVA-PTKAKR		rIIIB Env 286-467	human(IgM)
		Donor: ? **References:** [Ohlin et al.(1992)] **NOTES:** • MO101: generated through *in vitro* “immunization” of uninfected-donor lymphocytes – reacts with peptides from the V3 and C4 regions [Ohlin et al.(1992)]					
345	N70-1.9b	gp120(V3 316-322)	gp120(315-320)	PGRAFY	L	HIV-1 infection	human(IgG_1)
		Donor: ? **References:** [Robinson et al.(1990), Scott Jr et al.(1990)] **NOTES:** • N70-1.9b: Type specificity [Robinson et al.(1990)] • N70-1.9b: Type specific neutralization, ADCC directed against MN infected cells [Scott Jr et al.(1990)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
346	MAG 49	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRAFV-TIG	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: ?
References: [Kang et al.(1994), Moore & Sodroski(1996)]
NOTES:
- MAG 49: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]
- MAG 49: Called #49 in this text. Binding enhanced by anti-C1 MAbs 133/290, 135/9, and by many anti-CD4 binding site MAbs – reciprocal enhancement of some anti-V2 MAbs – reciprocal binding inhibition of anti-V3 MAbs [Moore & Sodroski(1996)]

347	MAG 53	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRAFV-TIG	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: ?
References: [Kang et al.(1994)]
NOTES:
- MAG 53: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]

348	MAG 56	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRAFV-TIG	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: ?
References: [Kang et al.(1994)]
NOTES:
- MAG 56: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]

349	MAG 109	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRAFV-TIG	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: ?
References: [Kang et al.(1994)]
NOTES:
- MAG 109: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
350	polyclonal	gp120(V3 306-338 BH10)	gp120(303-334)	PNNNTRKSIRIQRGPGR-AFVTIGKIGNMRQAHC	L	Peptide	rabbit(IgG)

Donor: ?
References: [Neurath & Strick(1990)]
NOTES:
- 21 V3 loop variant peptides spanning this region were tested and serological cross-reactivity correlated with divergence [Neurath & Strick(1990)]

351	1026	gp120(V3 tip MN)	gp120(314-319)	GPGRAF?	L	rgp120 MN	murine(IgG)

Donor: ?
References: [Nakamura et al.(1993), Bou-Habib et al.(1994)]
NOTES:
- 1026: Bound diverse strains, neutralizing activity against MN, close to GPGRAF [Nakamura et al.(1993)]
- 1026: Greater affinity for T cell-tropic strain T-CSF, derived from JR-CSF, than to the primary isolate JR-CSF [Bou-Habib et al.(1994)]

352	9284	gp120(V3 307-318 IIIB)	gp120(305-316)	NNTRKSIRIQRG	L	disrupted IIIB virion	murine(IgG_1)

Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware
References: [Skinner et al.(1988b), Skinner et al.(1988a), Sattentau & Moore(1991), Wyatt et al.(1992), McKeating et al.(1992a), Sattentau et al.(1993), Moore et al.(1993b), Trujillo et al.(1993), Thali et al.(1993), VanCott et al.(1994), Thali et al.(1994), Cook et al.(1994), Okada et al.(1994), Sorensen et al.(1994), Sattentau & Moore(1995), VanCott et al.(1995), Fontenot et al.(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Cao et al.(1997), Binley et al.(1997), Parren et al.(1998)]
NOTES:
- 9284: IIIB type-specific binding and neutralization [Skinner et al.(1988b)]
- 9284: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 9284: Single amino acid substitutions in the C4 region (427 W/V or W/S) or at the base of the V3 loop (298 R/G) can significantly increase binding and neutralization– position 427 is also important for CD4 binding and anti-CD4 binding site MAbs [Wyatt et al.(1992)]
- 9284: Increased binding in the presence of sCD4 [Sattentau et al.(1993)]
- 9284: Inhibits C4 region antibodies (G3-299, G3-519) which have conformational requirements [Moore et al.(1993b)]
- 9284: Peptide RIQRGPGRAFVTIGKIGNMRQA – Reacts with three human brain proteins of 35, 55, 110 kDa – called NEA-9284 [Trujillo et al.(1993)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
352 (cont.)						

NOTES:

- 9284: Does not bind MN gp120, just IIIB [VanCott et al.(1994)]
- 9284: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali et al.(1994)]
- 9284: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook et al.(1994)]
- 9284: Binding domain aa 301-310: TRKSIRIQRG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: 306 R/T,309 R/T and 313 R/G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5β – called NEA9284 [Okada et al.(1994)]
- 9284: Did not neutralize infection of HIV/HTLV-I pseudotype [Sorensen et al.(1994)]
- 9284: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains – neutralizes cell-free virus Hx10 [Sattentau & Moore(1995)]
- 9284: Used to monitor HIV-1 Env expression in infected H9 cells, binds native and reduced gp120s similarly [VanCott et al.(1995)]
- 9284: Binds V3 loop – anti-C1 MAbs 133/290 and 135/9 enhance binding – reciprocal binding inhibition of other anti-V3 MAbs [Moore & Sodroski(1996)]
- 9284: V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)]
- 9284: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao et al.(1997)]
- 9284: A high avidity antibody as assessed by urea elution
- 9284: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
353	1034	gp120(V3 tip MN)	gp120(314-319)	GPGRAF?	L	rgp120 MN	murine(IgG)
	Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware **References:** [Bou-Habib et al.(1994), Berman et al.(1997)] **NOTES:** • 1034: Greater affinity for T cell tropic T-CSF, derived from JR-CSF, than to the primary isolate JR-CSF, close to GPGRAF [Bou-Habib et al.(1994)] • 1034: Binds to 5/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)]						
354	polyclonal	gp120(V3 304-318 LAI)	gp120(306-320)	RKSIRIQRGPGRAFV		?	human(IgG, IgM)
	Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware **References:** [Chin et al.(1995)] **NOTES:** • Mimicking the humoral immune response *in vitro* supports isotype switching – human IgG MAbs were generated from naive donors [Chin et al.(1995)]						
355	polyclonal	gp120(V3 304-318 LAI)	gp120(306-320)	RKSIRIQRGPGRAFV		peptide	human(IgG, IgM)
	Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware **References:** [Andersson et al.(1997)] **NOTES:** • IgG to IgM isotype switching in response to primary and secondary peptide vaccinations was studied – the immunogen contained a V3 loop fragment and a tetanus toxin helper epitope [Andersson et al.(1997)]						
356	Aw	gp120(V3 tip, Gun-1wt)	gp120(309-322)	KSITIGPGRAFHAI	L	V3 peptide	rat
	Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware **References:** [McKnight et al.(1995)] **NOTES:** • Aw: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – Aw gives weak neutralization of both wt and v strains [McKnight et al.(1995)]						
357	Bw	gp120(V3 tip, Gun-1wt)	gp120(309-322)	KSITIGPGRAFHAI	L	V3 peptide	rat
	Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware **References:** [McKnight et al.(1995)] **NOTES:** • Bw: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – Bw gives weak neutralization of only the wt strain, does not bind to variant [McKnight et al.(1995)]						

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
358	Dv	gp120(V3 tip, Gun-1v)	gp120(309-322)	KSITIGSGRAFHAI	L	V3 peptide	rat

Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware
References: [McKnight et al.(1995)]
NOTES:
- Dv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]

359	Fv	gp120(V3 tip, Gun-1v)	gp120(309-322)	KSITIGSGRAFHAI	L	V3 peptide	rat

Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware
References: [McKnight et al.(1995)]
NOTES:
- Fv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]

360	Gv	gp120(V3 tip, Gun-1v)	gp120(309-322)	KSITIGSGRAFHAI	L	V3 peptide	rat

Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware
References: [McKnight et al.(1995)]
NOTES:
- Gv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]

361	Hv	gp120(V3 tip, Gun-1v)	gp120(309-322)	KSITIGSGRAFHAI	L	V3 peptide	rat

Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware
References: [McKnight et al.(1995)]
NOTES:
- Hv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]

362	polyclonal	gp120(V3 304-318 LAI)	gp120(310-321)	RIHIGPGRAFYT		?	human(IgG, IgM)

Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware
References: [Langedijk et al.(1995)]
NOTES:
- Polylconal sera from six individuals tested for reactivity against a panel of peptides based on autologous sequences provide evidence for immunological escape mutations in the tip of the V3 loop [Langedijk et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
363	C311E	gp120 (V3 309-316 MN)	gp120(308-315)	RKRIHIGP	L	IIIB infection	chimpanzee(IgG1)

Donor: ?
References: [Warrier et al.(1996), Alsmadi & Tilley(1998)]
NOTES:
- C311E: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier et al.(1996)]
- C311E: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – C311E bound and directed lysis against all four strains [Alsmadi & Tilley(1998)]

364	5G11	gp120(V3 loop)	gp120	?		?	

Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA
References: [Moore & Sodroski(1996)]
NOTES:
- 5G11: Binds to conformation sensitive epitope in the V3 loop – reciprocal inhibition of other V3 loop MAbs – reciprocal enhancement of some C1-C5 MAbs (unusual for an anti-V3 MAb) and CD4 binding site MAbs – and enhances binding of V2 MAbs [Moore & Sodroski(1996)]

365	110.3	gp120(V3 308-328 BRU)	gp120(312-319)	QRGPGRAF	L	BRU infected cell lysates	murine($IgG_{1\kappa}$)

Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA
References: [Kinney Thomas et al.(1988), Evans et al.(1989), Langedijk et al.(1992), Pirofski et al.(1993), Connelly et al.(1994)]
NOTES:
- 110.3: Included as a control [Evans et al.(1989)]
- 110.3: MAb variable region sequenced – heavy chain: V 7138(40), D deletion, J_H4 – light chain: $V_\kappa21(47)$, $J_\kappa2$ [Pirofski et al.(1993)]
- 110.3: An anti-idiotypic MAb generated against 110.3 both mimics and binds to V3, suggesting that the V3 loop may associated with itself [Connelly et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
366	110.4	gp120(V3 308-328 BRU)	gp120(312-319)	QRGPGRAF	L	BRU infected cell lysates	murine($IgG_{1\kappa}$)

Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas

References: [Kinney Thomas et al.(1988), Thali et al.(1992b), Langedijk et al.(1992), Thali et al.(1993), Pirofski et al.(1993), Arendrup et al.(1993), Thali et al.(1994), Boudet et al.(1994), Connelly et al.(1994), McDougal et al.(1996), Valenzuela et al.(1998), Cao et al.(1997)]

NOTES:

- 110.4: 313 P/S substitution in the V3 region disrupts binding [Thali et al.(1992b)]
- 110.4: MAb variable region sequenced – heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J_H2 – light chain: $V_\kappa21$, $J_\kappa2$ [Pirofski et al.(1993)]
- 110.4: Primary isolates from different time points from one individual were not susceptible to neutralization by 110.4 [Arendrup et al.(1993)]
- 110.4: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali et al.(1994)]
- 110.4: An anti-idiotypic MAb generated against 110.3 also blocks binding of 110.4 [Connelly et al.(1994)]
- 110.4: Neutralizes HIV-1 LAI [McDougal et al.(1996)]
- 110.4: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11-20 is through inhibition of viral binding to the cell [Valenzuela et al.(1998)]
- 110.4: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao et al.(1997)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
367	110.5	gp120(V3 308-328 BRU)	gp120(312-319)	QRGPGRAF	L	BRU infected cell lysates	murine($IgG_{1\kappa}$)

Donor: E. Kinney-Thomas or Genetic Systems, Seattle WA

References: [Kinney Thomas et al.(1988), Moore et al.(1990), Cordell et al.(1991), Sattentau & Moore(1991), Langedijk et al.(1992), McKeating et al.(1992a), Pirofski et al.(1993), Moore et al.(1993b), Thali et al.(1993), Klasse et al.(1993a), Sattentau et al.(1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), McDougal et al.(1996), Jeffs et al.(1996), Binley et al.(1997), Ugolini et al.(1997), Parren et al.(1998)]

NOTES:

- 110.5: Did not induce dissociation of gp120, as sCD4 did – discrepancy with [Poignard et al.(1996a)], that was suggested to be due to MAb interference with detection, as the gp120-MAb complex was denatured in the Poignard study [Moore et al.(1990)]
- 110.5: Binding insensitive to gp120 reduction [Cordell et al.(1991)]
- 110.5: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 110.5: Variable region sequenced – heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J_H2 – light chain: $V_\kappa21$, $J_\kappa2$ [Pirofski et al.(1993)]
- 110.5: Thrombin cleavage of V3 loop between R-315 and A-316 abrogates binding – can inhibit C4 region antibody which has conformational requirements (G3-299) – binding to native gp120 100-300 fold greater than to denatured [Moore et al.(1993b)]
- 110.5: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 110.5 is not affected [Reitz Jr. et al.(1988), Klasse et al.(1993a)]
- 110.5: Pretreatment of HX10-infected H9 cells with sCD4 decreases signal from 110.5 at 37 degrees due to dissociation of gp120-gp41 [Sattentau et al.(1995)]
- 110.5: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strains – neutralizes cell-free Hx10 [Sattentau & Moore(1995)]
- 110.5: Reciprocal binding inhibition with other anti-V3 MAbs – enhances binding of some anti-V2 MAbs – binding enhanced by some CD4 binding site MAbs [Moore & Sodroski(1996)]
- 110.5: V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)]
- 110.5: Neutralizes HIV-1 LAI [McDougal et al.(1996)]
- 110.5: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)]
- 110.5: A high avidity antibody as assessed by urea elution
- 110.5: Viral binding inhibition by 110.5 was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 110.5: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
368	5023A	gp120(V3 311-317 BH10)	gp120(313-319)	RgPGRAF	L	15 mer synthetic BH10 V3 peptide	murine(IgG)

Donor: Paul Durda, Du Pont de Nemours and Co
References: [Langedijk et al.(1991), D'Souza et al.(1991), Back et al.(1993), Rovinski et al.(1995)]
NOTES:

- 5023A: Generation and Fine mapping of murine MAbs [Langedijk et al.(1991)]
- 5023A: Called 5023 – Langedijk also has an MAb called 5023B – strong cross-reactive neutralizing MAb [D'Souza et al.(1991)]
- 5023A: Called 5023 – Langedijk also has an MAb called 5023B – gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662-675 is ELDKWANLWNWFNI [Back et al.(1993)]
- 5023A: Called 5023 in this paper – Used to precipitate gp160 in immunoblots in a study examining the feasibility of using unprocessed gp160 glycoprotein as an immunogen [Rovinski et al.(1995)]

369	178.1	gp120(V3 305-309 BH10)	gp120(309-313)	KSiRI	L	yeast rgp160 IIIB	murine(IgG_{2a})

Donor: C. Thiriart, Smith Kline and MRC AIDS reagent project
References: [Thiriart et al.(1989), Back et al.(1993), Moore & Ho(1993), Cook et al.(1994)]
NOTES:

- 178.1: Reacts to gp120 and gp160 in RIPA EIA and immunoblot [Thiriart et al.(1989)]
- 178.1: Called 178.1.1 – conformational, does not bind well to denatured gp120 [Moore & Ho(1993)]
- 178.1: gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662-675 is ELDKWANLWNWFNI [Back et al.(1993)]
- 178.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* – binding of GalCer to gp120 inhibited but did not completely block MAb binding[Cook et al.(1994)]
- 178.1: UK Medical Research Council AIDS reagent: ARP331

370	5042A	gp120(V3 310-315 BH10)	gp120(312-317)	QrGPGR	L	15 mer synthetic BH10 V3 peptide	murine(IgG)

Donor: C. Thiriart, Smith Kline and MRC AIDS reagent project
References: [Langedijk et al.(1991)]
NOTES:

- 5042A: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
371	5025A	gp120(V3 313-317 BH10)	gp120(315-319)	pgRAF	L	15 mer synthetic BH10 V3 peptide	murine(IgG)

Donor: Paul Durda, Du Pont de Nemours and Co
References: [Langedijk et al.(1991), D'Souza et al.(1991)]
NOTES:
- 5025A: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]
- 5025: Called 5025 – strain specific weakly neutralizing [D'Souza et al.(1991)]

372	5020	gp120(V3 311-316 BH10)	gp120(313-318)	RGPGRA	N	15 mer synthetic BH10 V3 peptide	murine(IgG)

Donor: Paul Durda, Du Pont de Nemours and Co
References: [Langedijk et al.(1991)]
NOTES:
- 5020: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]

373	5042B	gp120(V3 310-315 BH10)	gp120(312-317)	QRGPGr	N	15 mer synthetic BH10 V3 peptide	murine(IgG)

Donor: Paul Durda, Du Pont de Nemours and Co
References: [Langedijk et al.(1991)]
NOTES:
- 5042B: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]

374	5025B	gp120(V3 310-316 BH10)	gp120(312-318)	QRGPGra	N	15 mer synthetic BH10 V3 peptide	murine(IgG)

Donor: Paul Durda, Du Pont de Nemours and Co
References: [Langedijk et al.(1991)]
NOTES:
- 5025B: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]

375	5023B	gp120(V3 309-316 BH10)	gp120(311-318)	IQRGPGra	N	15 mer synthetic BH10 V3 peptide	murine(IgG)

Donor: Paul Durda, Du Pont de Nemours and Co
References: [Langedijk et al.(1991)]
NOTES:
- 5023B: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
376	110.I	gp120(V3 316-322)	gp120(318-324)	AFVTIGK	L	recombinant gp120	murine

Donor: F. Traincard, Pasteur Institute, France

References: [Moore et al.(1993b), Moore et al.(1994c), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Wyatt et al.(1997), Parren et al.(1998)]

NOTES:

- 110.I: Binds to carboxy-terminal side of the V3 loop – inhibits binding of C4 region MAb G3-299 [Moore et al.(1993b)]
- 110.I: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains [Sattentau & Moore(1995)]
- 110.I: Reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and enhances binding of some anti-V2 MAbs – binding enhanced by some anti-CD4 binding site MAbs [Moore & Sodroski(1996)]
- 110.I: Epitope suggested to be RAFVTIGK – V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)]
- 110.I: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)]
- 110.I: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

377	110.J	gp120(V3 loop)	gp120	?		?	

Donor: F. Traincard, Pasteur Institute, France

References: [Thali et al.(1993), Moore & Sodroski(1996)]

NOTES:

- 110.J: Inhibits sCD4-inducible anti-CD4 binding site MAb 48d [Thali et al.(1993)]
- 110.J: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs [Moore & Sodroski(1996)]

378	G3-1472	gp120(V3 loop)	gp120	?		?	

Donor: M. Fung

References: [Moore & Sodroski(1996)]

NOTES:

- G3-1472: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs – binding inhibited by anti-C4 MAbs [Moore & Sodroski(1996)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
379	AG1121	gp120(V3 loop)	gp120	?	L	?	

Donor: AGMED, Inc, Bedford MA, commercial
References: [Sullivan et al.(1995), Cao et al.(1997)]
NOTES:

- AG1121: Also called 1121
- AG1121: Recognizes monomeric gp120 from T-cell adapted line HXBc2 and primary isolate 89.6 equally well, but 89.6 was three-fold less sensitive to neutralization by AG1121 than HXBc2 [Sullivan et al.(1995)]
- AG1121: Called 1121 – Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao et al.(1997)]

380	110.6	gp120(V3 BRU)	gp120(313-320)	RGPGRAFV	L (weak)	BRU infected cell lysates	murine($IgG_{1\lambda}$)

Donor: AGMED, Inc, Bedford MA, commercial
References: [Kinney Thomas et al.(1988), Pirofski et al.(1993), Langedijk et al.(1992)]
NOTES:

- 110.6: Variable region sequenced – heavy chain: V J558-146b.1α, D closest to DSP16.2, J_H3 – light chain: $V_\lambda1$, $J_\lambda1$ [Pirofski et al.(1993)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
381	BAT123	gp120(V3 308-322 HXB2)	gp120(308-324)	RIRIQRGPGRAFVTIGK	L	Inact IIIB	murine($IgG_{1\kappa}$)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Fung et al.(1987), Liou et al.(1989), Fung et al.(1990), Moore & Ho(1993), Safrit et al.(1993), Thali et al.(1993), Pirofski et al.(1993), Gauduin et al.(1995), Sattentau & Moore(1995), Poignard et al.(1996a), Andrus et al.(1998), Parren et al.(1998), Gauduin et al.(1998)]

NOTES:

- BAT123: Also called BAT-123 – CGP 47 439 is a BAT123 chimera that has a human IgG_1 Fc domain
- BAT123: Anti-idiotypic MAb, AB19-4i, stimulates anti-anti-ID which neutralizes MN and IIIB [Fung et al.(1990)]
- BAT123: Called BAT-123 – conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120 – does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- BAT123: Passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus [Safrit et al.(1993)]
- BAT123: Variable region sequenced – heavy chain: V 3660-SB32, D unknown, J_H3 – light chain: $V_\kappa21$, $J_\kappa2$ [Pirofski et al.(1993)]
- BAT123: Passive transfer of BAT123 to hu-PBL-SCID mice 1 hour prior to inoculation with HIV-1 LAI, or up to four hours post-exposure, could protect mice from infection – the protection, like the MAb, was specific for the viral strain LAI [Gauduin et al.(1995)]
- BAT123: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strain [Sattentau & Moore(1995)]
- BAT123: Epitope described as RGPGRAFVTIGK – V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus (BAT123 less so than the others), mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)]
- BAT123: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIV-1 LAI in hu-PBL-SCID mice, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus et al.(1998)]
- BAT123: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- BAT123: Post-exposure passive transfer of murine BAT123 can confer protection to hu-PBL-SCID mice challenged with HIV-1 LAI – this protection is not elicited by CGP 47 439, a BAT123 chimera that has a human IgG_1 Fc domain, suggesting that the protection is mediated by complement – the protective ability of BAT123 is lost when mice were treated with cobra venom factor, which inactivates serum complement – IgG_1 does not fix complement efficiently so an IgG_2 MAb might perform better [Gauduin et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
382	CGP 47 439	gp120(V3 tip)	gp120(308-324)	?	L	IIIB gp120	BAT123-human Ig chimera

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Liou et al.(1989), Safrit et al.(1993), Gunthard et al.(1994), Gauduin et al.(1998), Jacobson(1998)]
NOTES:

- CGP 47 439: passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus – BAT123-human Ig chimera [Safrit et al.(1993)]
- CGP 47 439: PhaseI/IIA clinical trial studying multidose tolerability, immunogenicity and pharmacokinetic responses – GP 47 439 was well tolerated, serum $t_{1/2}$ was 8-16 days, and a virus burden reduction was noted in some patients [Gunthard et al.(1994)]
- CGP 47 439: Post-exposure passive transfer of murine BAT123 can confer protection to hu-PBL-SCID mice challenged with HIV-1 LAI – this protection is not elicited by CGP 47 439, suggesting that the protection is mediated by complement – the protective ability of BAT123 is lost when mice were treated with cobra venom factor, which inactivates serum complement – in this circumstance complement activation provided a protective advantage [Gauduin et al.(1998)]
- CGP 47 439: Review of passive immunotherapy, summarizing [Gunthard et al.(1994)] in relation to other studies [Jacobson(1998)]

383	10F10	gp120(V3 MN)	gp120(308-322)	RKRIHIGPGRAFYTT	L	Peptide	murine(IgG_1)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Duarte et al.(1994)]
NOTES:

- 2C4: Putative epitope lies within IHIGPGRAFYT – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAFYTT) peptides, lower affinity for SF2 [Duarte et al.(1994)]

384	2C4	gp120(V3 MN)	gp120(308-322)	RKRIHIGPGRAFYTT	L (MN)	Peptide	murine(IgG_{2a})

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Duarte et al.(1994)]
NOTES:

- 2C4: Putative epitope lies within IHIGPGRAFYT – neutralizes MN, not IIIB and SF2 – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAFYTT) peptides, lower affinity for SF2 [Duarte et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
385	19b	gp120(V3)	gp120(310-322)	-I—-G–FY-T	L	HIV-1 infection	human(IgG_1)

Donor: James Robinson, University of Connecticut, Storrs

References: [Scott Jr et al.(1990), Moore et al.(1994b), Moore et al.(1994a), Sattentau(1995), Moore et al.(1995b), Moore et al.(1995a), Moore & Ho(1995), Gauduin et al.(1996), Wu et al.(1996), Trkola et al.(1996a), D'Souza et al.(1997), Binley et al.(1997), Fouts et al.(1997), Ugolini et al.(1997), Boots et al.(1997), Parren et al.(1997b), Mondor et al.(1998), Parren et al.(1998), Trkola et al.(1998)]

NOTES:

- 19b: V3 loop binding MAb that is more broadly clade cross-reactive than most (binds to 19/29 clade B and 10/12 clade E gp120s) [Moore et al.(1994b)]
- 19b: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore et al.(1994a)]
- 19b: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau et al.(1995)]
- 19b: Binds to some gp120s from clades A,B,C,E, and F – weakly neutralized some B and one C clade virus [Moore et al.(1995b)]
- 19b: Despite broad gp120 binding reactivity, not broadly neutralizing [Moore et al.(1995a)]
- 19b: Review: more broadly cross-reactive than anti-V3 tip MAb 447-D [Moore & Ho(1995)]
- 19b: Not as effective as IgG1b12 at neutralization *ex vivo* of virus direct from plasma of HIV-1 infected individuals [Gauduin et al.(1996)]
- 19b: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of 19b blocks this inhibition [Wu et al.(1996)]
- 19b: Inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)

385 cont.

NOTES:

- 19b: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – there were four sequences with variations the defined epitope among the 9 isolates tested [D'Souza et al.(1997)]
- 19b: A low avidity antibody as assessed by urea elution
- 19b: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 19b bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- 19b: Viral binding inhibition by 19b was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 19b: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – 19b has an epitope involving the tip of the V3 loop, with 5 or 6 essential amino acids distributed within a 12 amino acid stretch – the previously determined binding site was confirmed -I—-G–FY-T and some tolerated variants described, the I can be I,V, or L, the Y can be Y, F, or W – probably a β-turn is required for FY or FF binding, but WY in can bind with out the context of the turn [Boots et al.(1997)]
- 19b: Neutralizes TCLA strains but not primary isolates [Parren et al.(1997b)]
- 19b: Used as a control in this Hx10 binding and neutralizing MAb study because 19b does not bind to Hx10 [Mondor et al.(1998)]
- 19b: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- 19b: No detectable neutralizing activity among primary isolates with different co-receptor usage – some neutralization of TCLA strains [Trkola et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
386	G3-523	gp120(V3 308-322)	gp120(310-322)	RIQRGPGRAFVTIGK		?	murine

Donor: James Robinson, University of Connecticut, Storrs
References: [Matsushita et al.(1988), Jagodzinski et al.(1996)]
NOTES:
- G3-523: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits G3-523 binding [Jagodzinski et al.(1996)]

387	4G10	gp120(V3 308-322 LAI)	gp120(310-322)	RIQRGPGRAFVTGK		V3-loop HBcAg hybrid	murine

Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany
References: [von Brunn et al.(1993)]
NOTES:
- 4G10: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn et al.(1993)]
- 4G10: NIH AIDS Research and Reference Reagent Program: 2534

388	5F7	gp120(V3 308-322 LAI)	gp120(310-322)	RIQRGPGRAFVTGK		V3-loop HBcAg hybrid	murine

Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany
References: [von Brunn et al.(1993)]
NOTES:
- 5F7: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn et al.(1993)]
- 5F7: NIH AIDS Research and Reference Reagent Program: 2533

389	10/54	gp120(V3 311-321 HXB10)	gp120(313-323)	RGPGRAFVTIG	L (HXB10)	rgp120 BH10	rat(IgG_1)

Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany
References: [McKeating et al.(1992a), McKeating et al.(1993a), McKeating et al.(1993b)]
NOTES:
- 10/54: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)]
- 10/54: Studied in the context of a neutralization escape mutant [McKeating et al.(1993a)]

390	10/36e	gp120(V3 311-321 HXB10)	gp120(313-323)	RGPGRAFVTIG	L (HXB10)	rgp120 BH10	rat(IgG_{2a})

Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany
References: [McKeating et al.(1992a), McKeating et al.(1993b)]
NOTES:
- 10/36e: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
391	11/85b	gp120(V3 311-321 HXB10)	gp120(313-323)	RGPGRAFVTIG	L (HXB2)	rgp120 BH10	rat(IgG_{2b})

Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany
References: [McKeating et al.(1992a), McKeating et al.(1993b)]
NOTES:
- 11/85b: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)]

392	loop 2	gp120(V3)	gp120(311-322)	SISGPGRAFYTG	L	HIV-1 infection	human Fab

Donor: D. Burton, Scripps Research Institute, La Jolla, CA
References: [Barbas III et al.(1993), Moore et al.(1994b), Wu et al.(1996), Ditzel et al.(1997), Ugolini et al.(1997), Parren et al.(1997b), Parren & Burton(1997), Mondor et al.(1998), Parren et al.(1998)]
NOTES:
- loop2: Also known as Loop 2, IgG1 Loop 2 was a obtained by engineering Fab loop2 into an IgG1 molecule
- loop 2: Sequences of the heavy and light chain Fab variable regions were generated [Barbas III et al.(1993)]
- loop 2: Called Loop 2 – shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore et al.(1994b)]
- loop 2: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of loop 2 blocks this inhibition [Wu et al.(1996)]
- loop 2: Binds to gp120 from MN and SF2 but not LAI [Ditzel et al.(1997)]
- loop 2: Viral binding inhibition by loop 2 MAb or Fab was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- loop 2: Epitope is probably GPGRAF – binds to 10/17 US clade B monomeric gp120s – IgG1 form can neutralize MN and 2 primary isolates tested [Parren & Burton(1997)]
- loop 2: Neutralizes TCLA strains but not primary isolates [Parren et al.(1997b)]
- loop 2: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 $>$ 3B3 $>$ b12 = DO8i $>$ b11 $>$ b3 $>$ b14 $>$ b13 $>$ DO142-10 $>$ DA48 $>$ L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 $>$ b12 $>$ DO142-10 $>$ Loop 2 $>$ b11 $>$ L17 $>$ b6 $>$ DO8i $>$ b14 $>$ DA48 $>$ b3 $>$ b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope – binding affinity of divalent IgG1 loop 2 is only 2-fold greater than monovalent Fab loop 2, suggesting the IgG1 form may bind with only one arm [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
393	41.1	gp120(V3 dis HXB10)	gp120	DISCONTINUOUS	L (HXB2)	rgp120 BH10	rat(IgG_{2a})

Donor: J. Cordell, Institute for Cancer Research, Sutton, Surrey, UK

References: [McKeating et al.(1992a), McKeating et al.(1993b), Klasse et al.(1993a), McLain & Dimmock(1994), Armstrong & Dimmock(1996), Armstrong et al.(1996), Jeffs et al.(1996), Ugolini et al.(1997)]

NOTES:

- 41.1: Also called ICR41.1i and ICR41
- 41.1: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 41.1 is not affected [Reitz Jr. et al.(1988), Klasse et al.(1993a)]
- 41.1: Called ICR41.1i – Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – neutralization mediated by 3 molecules of IgG per virion – most efficient at neutralization of the three MAbs studied – acts with multi-hit kinetics [McLain & Dimmock(1994)]
- 41.1: Called ICR41.1i – IgG_{2c}? – Neutralization was affected if the Ab was added after the virus bound to the host cells at 24 degrees C or below [Armstrong & Dimmock(1996)]
- 41.1: Called ICR41.1i – Neutralization occurs by blocking a post-fusion internalization event, in contrast to MAb F58 [Armstrong et al.(1996)]
- 41.1: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)]
- 41.1: Viral binding inhibition by 41.1 was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
394	DO142-10	gp120(V3 MN)	gp120	SISGPGRAFYTG	L	HIV-1 infection	human Fab(IgG_1)

Donor: J. Cordell, Institute for Cancer Research, Sutton, Surrey, UK

References: [Ditzel et al.(1997), Parren et al.(1997b), Parren & Burton(1997), Parren et al.(1998)]

NOTES:

- DO142-10: Phage expression libraries panned against MN peptide were used to select Fab DO142-10 – Fab binds MN gp120, but not a primary isolate rec gp120 [Ditzel et al.(1997)]
- DO142-10: Neutralizes TCLA strains but not primary isolates [Parren et al.(1997b)]
- DO142-10: Binds to gp120 MN and an MN V3 peptide with equal affinity, but binds a consensus B peptide and JRCSF less well, and to IIIB gp120 not at all [Parren & Burton(1997)]
- DO142-10: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different that FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
395	257-D	gp120(V3 MN)	gp120(309-313)	KRIHI	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Gorny et al.(1991), D'Souza et al.(1991), Karwowska et al.(1992b), Gorny et al.(1993), Cavacini et al.(1993a), Spear et al.(1993), D'Souza et al.(1994), VanCott et al.(1994), D'Souza et al.(1995), Zolla-Pazner et al.(1995), Schutten et al.(1995a), Schutten et al.(1995b), Fontenot et al.(1995), Wisnewski et al.(1996), Schutten et al.(1996), Schutten et al.(1997), Stamatatos et al.(1997), Hill et al.(1997), LaCasse et al.(1998), Yang et al.(1998), Gorny et al.(1998)]

NOTES:

- 257-D: Also called 257, 257-2-D-IV and 257-D-IV
- 257-D: Called 257-2-D-IV – potent neutralizing MAb [D'Souza et al.(1991)]
- 257-D: Reacts with MN, NY5, CDC4 and SF2, does not cross-react with RF, WM52, or HXB2 [Karwowska et al.(1992b)]
- 257-D: Neutralizes MN – binds SF2: KSIYI – specificity: MN, SF2, NY5, RF. [Gorny et al.(1993)]
- 257-D: Additive MN or SF2 neutralization when combined with CD4 binding site MAb F105 – does not neutralize RF [Cavacini et al.(1993a)]
- 257-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG – complement mediated virolysis of MN, but not in the presence of sCD4 [Spear et al.(1993)]
- 257-D: Included a multi-lab study for antibody characterization and assay comparison – best NAb against MN, but not IIIB [D'Souza et al.(1994)]
- 257-D: Potent MN neutralization, slow dissociation constant [VanCott et al.(1994)]
- 257-D: Called 257-D-IV – could neutralize MN and closely related JRCSF, but not 2 B subtype and 1 D subtype primary isolates in a multi-laboratory study involving 11 labs [D'Souza et al.(1995)]
- 257-D: In serotyping study using flow-cytometry, bound only to virus with KRIHI [Zolla-Pazner et al.(1995)]
- 257-D: Only inhibition of SI phenotype virus, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten et al.(1995a)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)

395 cont.

NOTES:

- 257-D: Comparable affinity for SI and NSI viruses, in contrast to MAb MN215 [Schutten et al.(1995b)]
- 257-D: 257-D is V_H5 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- 257-D: IIIB neutralizing MAbs *in vitro* fail to neutralize in a mouse model *in vivo* [Schutten et al.(1996)]
- 257-D: Neutralized (>90%) an SI-env chimeric virus and enhanced (>200%) an NSI-env chimeric virus [Schutten et al.(1997)]
- 257-D: Binds less extensively than MAb 391-95D on the surface of HIV-1 isolates SF162 and SF128A – neutralizes less potently than 391-95D – stronger neutralization of primary macrophage targets than PBMC [Stamatatos et al.(1997)]
- 257-D: Called 257 – gp120 can inhibit MIP-1α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill et al.(1997)]
- 257-D: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by anti-V3 MAbs. The primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse et al.(1998)]
- 257-D: A neutralization assay was developed based on heminested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang et al.(1998)]
- 257-D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 257-D has a slow dissociation, thus the highest affinity among V3 MAbs [Gorny et al.(1998)]
- 257-D: UK Medical Research Council AIDS reagent: ARP3023
- 257-D: NIH AIDS Research and Reference Reagent Program: 1510

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
396	4117C	gp120(V3)	gp120(311-317)	IXIGPGR	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Tilley et al.(1991a), Tilley et al.(1992), di Marzo Veronese et al.(1993), Pinter et al.(1993a), Pinter et al.(1993b), Alsmadi & Tilley(1998)]

NOTES:

- 4117C: Potent neutralizing activity against MN, SF-2, and NY-5 – synergy with CD4BS MAb 1125H [Tilley et al.(1991a)]
- 4117C: Neutralizes SF2 and MN synergistically combined with anti-CD4 binding site discontinuous MAb [Pinter et al.(1993a), Tilley et al.(1992)]
- 4117C: Binds V3 loop – does not immunoprecipitate soluble gp120, does react with gp120 on intact virions [Pinter et al.(1993b)]
- 4117C: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against MN and SF2, but not IIIB and RF [Alsmadi & Tilley(1998)]

397	41148D	gp120(V3 MN)	gp120(309-315)	KRIHIGP	L	HIV-1 infection	human(IgG1)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Pinter et al.(1993b), Alsmadi & Tilley(1998)]

NOTES:

- 41148D: Neutralizes less potently than 4117C, reacts with MN, IIIB, SF2 [Pinter et al.(1993b)]
- 41148D: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against strains IIIB, MN, SF-2, comparable to 4117C, however 41148D is 10x less efficient at neutralization, showing ADCC and neutralization don't always correlate [Alsmadi & Tilley(1998)]

398	453-D	gp120(V3 MN)	gp120(311-317)	IHIGPGR	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Gorny et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)]

NOTES:

- 453-D: Neutralizes MN – binds SF2: IYIGPGR – specificity: MN, SF2, NY5, RF [Gorny et al.(1993)]
- 453-D: Moderate homologous neutralization, moderately slow dissociation rate [VanCott et al.(1994)]
- 453-D : Called 453, epitope described as KRIHIGPGR – the tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot et al.(1995)]

399	504-D	gp120(V3)	gp120(311-317)	IHIGPGR	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Gorny et al.(1993)]

NOTES:

- 504-D – Neutralizes MN – binds SF2: IYIGPGR [Gorny et al.(1993)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
400	418-D	gp120(V3)	gp120(312-318)	HIGPGRA	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Karwowska et al.(1992b), Gorny et al.(1993)]
NOTES:
- 418-D: MN strain specific, does not cross-react with SF2, NY5, RF, CDC4 WM52 or HXB2 [Karwowska et al.(1992b)]
- 418-D: Neutralizes MN, does not bind to SF2 or HXB2 [Gorny et al.(1993)]

401	311-11-D	gp120(V3)	gp120(309-315)	KRIHIGP	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Gorny et al.(1993), Spear et al.(1993), Gorny et al.(1998)]
NOTES:
- 311-11-D: Also called 311-11D
- 311-11-D: Neutralizes MN – binds SF2: KSIYIGP [Gorny et al.(1993)]
- 311-11-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear et al.(1993)]

402	391/95-D	gp120(V3)	gp120(308-322)	RKRIHIGPGRAFYTT	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Gorny et al.(1993), Fontenot et al.(1995), Seligman et al.(1996), Stamatatos et al.(1997)]
NOTES:
- 391/95-D: Also called 391-95D
- 391/95-D: Neutralizes MN – binds to SF2, not IIIB [Gorny et al.(1993)]
- 391/95-D: Competition ELISAs with serial deletions estimated the epitope to be KRIHIGPGRAFY – unconstrained peptide had higher affinity than cyclic [Seligman et al.(1996)]
- 391/95-D: Called 391-95D – binds more extensively than MAb 257-D on the surface of HIV-1 isolates SF162 and SF128A – neutralizes more potently than 257-D – stronger neutralization of primary macrophage targets than PBMC – binding post-gp120-sCD4 association is related to anti-V3 Abs neutralizing capacity [Stamatatos et al.(1997)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
403	412-D	gp120(V3 MN)	gp120(308-322)	RKRIHIGPGRAFYTT	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Gorny et al.(1993), Spear et al.(1993), VanCott et al.(1994), Fontenot et al.(1995), Gorny et al.(1998)]

NOTES:

- 412-D: Also called 412-10D and 412
- 412-D: Neutralizes MN, does not bind SF2 or HXB2 – not reactive with hexa or heptapeptides by Pepscan [Gorny et al.(1993)]
- 412-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear et al.(1993)]
- 412-D: Called 412-10D – relatively rapid dissociation and weak homologous neutralization [VanCott et al.(1994)]
- 412-D: Called 412 – The tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot et al.(1995)]
- 412-D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 412-D has a relatively fast dissociation, thus low affinity among V3 MAbs [Gorny et al.(1998)]

404	MN215	gp120(V3 MN)	gp120(310-324)	RIHIGPGRAFYTTKN	NEUTRALIZING;	HIV-1 infection	human(IgG_1)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Schutten et al.(1995b)]

NOTES:

- MN215: Minimum epitope for MAB using the Dutch consensus is AFYTTGE, different than defined for MN – generated by EBV transformation of PBMC – displayed higher affinity for NSI than for SI glycoproteins – amino acids HIGP were essential for binding [Schutten et al.(1995b)]

405	SP.BAL114	gp120(V3 BAL)	gp120(310-319)	SIHIGPGRAF	L	?	murine?($IgG_{2a\kappa}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Arendrup et al.(1995)]

NOTES:

- Authors suggest that during *in vivo* immunoselection of escape virus, the V3 domain gains increasing resemblance to that of lab strains [Arendrup et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
406	SP.SF2:104	gp120(V3 SF2)	gp120(310-319)	SIYIGPGRAF	L	HIV-1 infection	($IgG_{2a\kappa}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Arendrup et al.(1993), Arendrup et al.(1995)]
NOTES:
- SP.SF2:104: Anti-V3 antibody that could neutralize primary virus isolated from a time point of neutralization resistance of autologous virus [Arendrup et al.(1993)]
- SP.SF2:104: Authors suggest that during *in vivo* immunoselection of escape virus, the V3 domain gains increasing resemblance to lab strains [Arendrup et al.(1995)]

407	A47/B1	gp120(V3 307-316 IIIB)	gp120(311-320)	IQRGPGRAFV	L	IIIB gp120	murine(IgG)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Akerblom et al.(1990)]

408	G44/H7	gp120(V3 307-316 IIIB)	gp120(311-320)	IQRGPGRAFV	L	IIIB gp120	murine(IgG)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Akerblom et al.(1990)]

409	D59/A2	gp120(V3 307-316 IIIB)	gp120(311-320)	IQRGPGRAFV	L	IIIB gp120	murine(IgG)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Akerblom et al.(1990)]

410	IIIB-34 V3	gp120(V3 308-316 IIIB)	gp120(311-319)	IQRGPGRAF	L	Peptide	murine(IgG_1)

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Laman et al.(1992), Laman et al.(1993)]
NOTES:
- IIIB-34 V3: Neutralizes IIIB but not MN – QXGPG are critical amino acids for binding by Pepscan analysis [Laman et al.(1992)]
- IIIB-34 V3: Called IIIB-V3-34 – IIIB strain specific neutralization – binding is reduced somewhat by DTT or SDS-DTT, enhanced by NP40, but binds to native and denatured gp120 [Laman et al.(1993)]
- IIIB-34 V3: UK Medical Research Council AIDS reagent: ARP3047

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
411	IIIB-13 V3	gp120(V3 308-316 IIIB)	gp120(311-319)	IQRGPGRAF	L	Peptide	murine(IgG_1)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Laman et al.(1992), Laman et al.(1993), D'Souza et al.(1994), Watkins et al.(1993)]

NOTES:

- IIIB-13 V3: Also known as 1044-13 and as IIIB-V3-13 (J. P. Moore, per. comm.)
- IIIB-13 V3: Neutralizes IIIB but not MN [Laman et al.(1992)]
- IIIB-13 V3: Included in a panel of antibodies used in a multi-lab study for antibody characterization and assay comparison, some neutralization of strains other than IIIB [D'Souza et al.(1994)]
- IIIB-13 V3: Called IIIB-V3-13 – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – IIIB-V3-13 neutralization was only slightly reduced by this mutation [Watkins et al.(1993)]
- IIIB-13 V3: UK Medical Research Council AIDS reagent: ARP3046
- IIIB-13 V3: NIH AIDS Research and Reference Reagent Program: 1727

412	M77	gp120(V3 IIIB)	gp120(309-322)	IRIQRGPGRAFVTI	L	HIV-1 infection	human(IgG)

Donor: Advanced BioScience Laboratories, Rockville, MD, commercial

References: [Pal et al.(1992), di Marzo Veronese et al.(1992), di Marzo Veronese et al.(1993), Watkins et al.(1993), Cook et al.(1994), Devico et al.(1995), Denisova et al.(1995), Denisova et al.(1995), Watkins et al.(1996)]

NOTES:

- M77: IIIB-specific MAb, immunoprecipitates deglycosylated form [di Marzo Veronese et al.(1992)]
- M77: Antibody binding to viral isolates from IIIB infected lab worker followed through time – A to T substitution resulted in the loss of neutralization and native gp120 binding, but not peptide binding [di Marzo Veronese et al.(1993)]
- M77: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook et al.(1994)]
- M77: Reacted with both reduced and non-reduced covalently cross-linked gp120-CD4 complex [Devico et al.(1995)]
- M77: Conformational rearrangements upon binding of M77 to gp120 generates novel epitopes called metatopes [Denisova et al.(1995)]
- M77: Stated to be a murine MAb – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – M77 neutralization was only slightly reduced by this mutation [Watkins et al.(1993)]
- M77: Used M77 bound to gp120 as an immunogen – analysis of polyclonal and monoclonal (62 MAbs were generated) response suggests the M77-gp120 immunogen generated MAbs to more linear epitopes than gp120 alone or gp120 bound to CD4 [Denisova et al.(1996)]
- M77: Native M77 is highly strain specific, and V3 binding is primarily dependent on its heavy chain – a light chain switched Fab version of M77 could recognize HIV-1 strains that had substitutions on the left side of the V3 loop – R in GPGR is likely to be critical for binding [Watkins et al.(1996)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
413	268-D	gp120(V3 MN)	gp120(312-317)	HIGPGR	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Gorny et al.(1991), D'Souza et al.(1991), Karwowska et al.(1992b), Gorny et al.(1993), Spear et al.(1993), VanCott et al.(1994), Zolla-Pazner et al.(1995), Fontenot et al.(1995), McKeating et al.(1996), Wisnewski et al.(1996), Stamatatos et al.(1997), LaCasse et al.(1998)]

NOTES:

- 268-D: Also called 268-11-D-IV and 268D
- 268-D: Called 268-11-D-IV – strain specific weakly neutralizing [D'Souza et al.(1991)]
- 268-D: Reacts with MN, NY5, CDC4, RF and SF2, does not cross-react with WM52 or HXB2 [Karwowska et al.(1992b)]
- 268-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny et al.(1993)]
- 268-D: Mediated deposition of complement component C3 on HIV infected cells, but not in the presence of sCD4 [Spear et al.(1993)]
- 268-D: Moderate dissociation rate and homologous neutralization titer [VanCott et al.(1994)]
- 268-D: Serotyping study using flow-cytometry, if H of HIGPGR was substituted in virus, 268-D did not bind [Zolla-Pazner et al.(1995)]
- 268-D: Failed to neutralize HXB2 and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]
- 268-D: 268-D is V_H4 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- 268-D: Poor reactivity against HIV-1 isolates SF162 and SF128A and no neutralization, in contrast to MAbs 391/95-D and 257-D [Stamatatos et al.(1997)]
- 268-D: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by anti-V3 MAbs. The primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse et al.(1998)]
- 268-D: UK Medical Research Council AIDS reagent: ARP3024
- 268-D: NIH AIDS Research and Reference Reagent Program: 1511

414	polyclonal	gp120(V3 MN)	gp120(313-320)	IGPGRAFY	L	gp120-*B. abortus* complex (SF2 or MN)	murine(IgG2a)

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Golding et al.(1995)]

NOTES:

- Ab is evoked even in mice depleted of CD4+ cells

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
415	0.5β	gp120(V3 316-330 HXB2)	gp120(313-326)	RGPGRAFVTIGKIG	L (IIIB)	IIIB Env	murine(IgG$_{1\kappa}$)

Donor: Shuzo Matsushita or Toshio Hattori of Kumamoto University

References: [Matsushita et al.(1988), Skinner et al.(1988b), Skinner et al.(1988a), Reitz Jr. et al.(1988), Nara et al.(1990), D'Souza et al.(1991), Matsushita et al.(1992), Emini et al.(1992), Maeda et al.(1992), McKeating et al.(1992a), Sperlagh et al.(1993), di Marzo Veronese et al.(1993), Moore et al.(1993b), Klasse et al.(1993a), Watkins et al.(1993), Cook et al.(1994), Thali et al.(1994), Okada et al.(1994), Boudet et al.(1994), Broder et al.(1994), Zvi et al.(1995b), Zvi et al.(1995a), Jagodzinski et al.(1996), Warrier et al.(1996), McDougal et al.(1996), Jeffs et al.(1996), Huang et al.(1997), Zvi et al.(1997), Wyatt et al.(1997), Faiman & Horovitz(1997)]

NOTES:

- 0.5β: Also called 0.5 beta and 0.5beta
- 0.5β: Type-specific neutralization of IIIB – does not neutralize MN or RF [Matsushita et al.(1988), Skinner et al.(1988b)]
- 0.5β: Emergence of virus resistant to MAb 0.5β and autologous sera neutralization in IIIB infected chimps [Nara et al.(1990)]
- 0.5β: Potent neutralizing activity [D'Souza et al.(1991)]
- 0.5β: Chimeric mouse-human MAb Cβ1 was constructed by combining the human Cγ1 and Cκ constant regions with the 0.5β murine MAb – ADCC and neutralizing activity[Matsushita et al.(1992)]
- 0.5β: sCD4 causes loss of IIIB type-specificity, allowing binding and neutralization of MN, in contrast to MAb μ5.5 [Maeda et al.(1992)]
- 0.5β: Monoclonal anti-idiotype antibodies that mimic the 0.5β epitope were generated [Sperlagh et al.(1993)]
- 0.5β: Neutralization of virus carrying an A to T substitution (contrast with MAb M77) [di Marzo Veronese et al.(1993)]
- 0.5β: Binding to native gp120 100-300 fold greater than to denatured [Moore et al.(1993b)]
- 0.5β: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to some antiserum and conformationally sensitive neutralizing MAbs – neutralization efficiency of 0.5β is not affected [Reitz Jr. et al.(1988), Klasse et al.(1993a)]
- 0.5β: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – of the MAbs tested , 0.5β neutralization was the most profoundly affected by this mutation [Watkins et al.(1993)]
- 0.5β: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook et al.(1994)]
- 0.5β: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali et al.(1994)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)

415 cont.

NOTES:

- 0.5β: Binding domain aa 310-319: RGPGRAFVTIGKIG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: 306 R/T,309 R/T and 313 R/G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5β [Okada et al.(1994)]
- 0.5β: Type-specific neutralization of IIIB – does not neutralize SF2 [Broder et al.(1994)]
- 0.5β: The interactions of the peptide RKSIRIQRGPGRAFVT 0.5β were studied by NMR, and hydrophobic interactions between the two Is and the V form the base of a 12 amino acid loop with GPGR at the apex[Zvi et al.(1995b)]
- 0.5β: NMR of 0.5β bound NNTRKSIRIQRGPGRAFVTIGKIG suggests that the bound amino acids are in the region SIRIQRGPGRAFVT [Zvi et al.(1995a)]
- 0.5β: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits 0.5β binding – 0.5β epitope described as GPGRAFVTIG [Jagodzinski et al.(1996)]
- 0.5β: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier et al.(1996)]
- 0.5β: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)]
- 0.5β: Relative to the native peptide, an O-linked α-galactosamine modified V3 peptide enhanced binding to 0.5 β, while an N-linked β-glucosamine modified peptide showed reduced binding [Huang et al.(1997)]
- 0.5β: The structure of a 17 amino acid V3 peptide bound to the FAb was studied using NMR [Zvi et al.(1997)]
- 0.5β: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)]
- 0.5β: The Fv fragment was purified and the temperature dependence and effect of mutations was studied [Faiman & Horovitz(1997)]
- 0.5β: UK Medical Research Council AIDS reagent: ARP3025
- 0.5β: NIH AIDS Research and Reference Reagent Program: 1591

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
416	924	gp120(V3 309-318 IIIB)	gp120(308-316)	RKSIRIQRGPG		vaccinia-gp160 IIIB	murine($IgG_{1\kappa}$)

Donor: ?

References: [Chesebro & Wehrly(1988), Pincus et al.(1991), Pincus & McClure(1993), Pincus et al.(1993), Cook et al.(1994), Pincus et al.(1996), Pincus et al.(1998)]

NOTES:

- 924: HIV IIIB strain specific [Chesebro & Wehrly(1988)]
- 924: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIB strain-specific [Pincus et al.(1991)]
- 924: MAb was coupled to ricin A chain (RAC) – immunotoxin efficacy was not significantly decreased by sCD4, although the efficacy of gp41 MAb immunotoxins in vitro increased 30-fold by sCD4 [Pincus & McClure(1993)]
- 924: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – MAb 924 was used as a control – infected lab workers and a vaccinia gp160 vaccine had strong V3 MAb response, but alum absorbed rec gp160 did not generate anti-V3 response [Pincus et al.(1993)]
- 924: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook et al.(1994)]
- 924: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
417	907	gp120(V3 309-318)	gp120(308-316)	RKSIRIQRGPG	L	vaccinia-gp160 IIIB	murine($IgG_{1\kappa}$)

Donor: ?

References: [Chesebro & Wehrly(1988), Pincus et al.(1989), Pincus et al.(1991), Pincus et al.(1996)]

NOTES:

- 907: Strain specific binding, and neutralization of only the LAV strain [Chesebro & Wehrly(1988)]
- 907: Coupled to ricin A chain (RAC), MAb 907 inhibited protein synthesis and cell growth in HIV-infected cells [Pincus et al.(1989)]
- 907: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIB strain-specific [Pincus et al.(1991)]
- 907: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
418	Cβ1	gp120(V3 316-330 HXB2)	gp120(313-326)	RGPGRAFVTIGKIG	L	IIIB Env	human (IgG_1) 0.5β chimera
		Donor: ? **References:** [Emini et al.(1992)] **NOTES:** • Cβ1: passive transfer to chimpanzees confers protection against challenge with homologous cell-free virus – mouse 0.5β human IgG_1 chimera [Emini et al.(1992)]					
419	386-D	gp120(V3 MN)	gp120(312-317)	HIGPGR	L	HIV-1 infection	human($IgG_{1\lambda}$)
		Donor: ? **References:** [Karwowska et al.(1992b), Gorny et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)] **NOTES:** • 386-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny et al.(1993)] • 386-D: Slow dissociation rate, potent homologous neutralization [VanCott et al.(1994)]					
420	5021	gp120(V3)	gp120(312-318)	QrGPGRa	L	15 mer BH10 V3 peptide	murine(IgG)
		Donor: ? **References:** [Durda et al.(1988), Durda et al.(1990), Langedijk et al.(1991), Moore et al.(1993b)] **NOTES:** • 5021: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)] • 5021: Binding to native gp120 100-300 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]					
421	5042	gp120(V3)	gp120(312-318)	QRGPGRA	L	peptide	murine
		Donor: ? **References:** [Durda et al.(1988), Durda et al.(1990), Moore et al.(1993b)] **NOTES:** • 5042: Binding to native gp120 100-300 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]					
422	F58/D1	gp120(V3)	gp120(311-318)	IXXGPGRA	L	virus derived gp120	human
		Donor: ? **References:** [Akerblom et al.(1990), Broliden et al.(1991), Moore et al.(1993b)] **NOTES:** • F58/D1: Binding to native gp120 1-3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
423	P1/D12	gp120(V3)	gp120(311-318)	IXXGPGRA	L	virus derived IIIB gp120	murine(IgG)

Donor: ?
References: [Akerblom et al.(1990), Moore et al.(1993b)]
NOTES:

- P1/D12: Binding to native gp120 1-3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]

424	P4/D10	gp120(V3)	gp120(311-318)	IXXGPGRA	L	virus derived IIIB gp120	murine($IgG_{1\kappa}$)

Donor: ?
References: [Akerblom et al.(1990), Broliden et al.(1990), Broliden et al.(1991), Marks et al.(1992), Moore et al.(1993b), Arendrup et al.(1993), Hinkula et al.(1994), Jacobson(1998)]
NOTES:

- P4/D10: Neutralizing and ADCC activity [Broliden et al.(1990)]
- P4/D10: Variable domain sequenced and is identical to F58/H3 [Marks et al.(1992)]
- P4/D10: Binding to native gp120 3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]
- P4/D10: Primary isolates from different time points from one individual were not susceptible to neutralization by P4/D10 [Arendrup et al.(1993)]
- P4/D10: Used for passive immunotherapy in four late-stage HIV-infected patients – the serum level of p24 did not decrease in any of these four – see also MAb F58/H3 [Hinkula et al.(1994)]
- P4/D10: Review of passive immunotherapy, summarizing [Hinkula et al.(1994)] in relation to other studies [Jacobson(1998)]

425	419-D	gp120(V3)	gp120(311-317)	IHIGPGR	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: ?
References: [Karwowska et al.(1992b), Gorny et al.(1993), Spear et al.(1993), Fontenot et al.(1995)]
NOTES:

- 419-D: MN, NY5 and SF2 strain specific, does not cross-react with RF, CDC4, WM52 or HXB2 [Karwowska et al.(1992b)]
- 419-D: Neutralizes MN – binds SF2: IYIGPGR [Gorny et al.(1993)]
- 419-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear et al.(1993)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
426	537-D	gp120(V3)	gp120(313-317)	IGPGR	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: ?

References: [Karwowska et al.(1992b), Gorny et al.(1992), Gorny et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)]

NOTES:

- 537-D: Reacts with MN, NY5, CDC4, RF, WM52 and SF2, but does not cross-react with HXB2 [Karwowska et al.(1992b)]
- 537-D: MN type specific neutralization observed – binds SF2, also IGPGR [Gorny et al.(1992), Gorny et al.(1993)]
- 537-D: Moderate homologous neutralization, relatively rapid dissociation constant [VanCott et al.(1994)]

427	NM-01	gp120(V3 MN)	gp120(314-317)	GPGR	L	IIIB MN	murine(IgG)

Donor: M. Terada

References: [Ohno et al.(1991), Yoshida et al.(1997), Smith et al.(1998)]

NOTES:

- NM-01: Resistance mutation selected by propagation of molecular cloned isolate in the presence of NM-01 [Yoshida et al.(1997)]
- NM-01: The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and NM-01 was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
428	447-52D	gp120(V3 MN)	gp120(314-317)	GPXR	L	HIV-1 infection	human($IgG_{3\lambda}$)

Donor: Dr. Susan Zolla-Pazner, NYU Med Center NY, NY, or Cellular Products Inc, Buffalo, NY, USA

References: [Gorny et al.(1992), Buchbinder et al.(1992), Karwowska et al.(1992b), Gorny et al.(1993), Keller et al.(1993), Cavacini et al.(1993a), Spear et al.(1993), Conley et al.(1994a), Laal et al.(1994), VanCott et al.(1994), Gorny et al.(1994), Moore et al.(1994a), Sattentau(1995), Fontenot et al.(1995), Saarloos et al.(1995), Zolla-Pazner et al.(1995), Zolla-Pazner & Sharpe(1995), Moore et al.(1995a), Moore & Ho(1995), Forthal et al.(1995), Jagodzinski et al.(1996), Trkola et al.(1996a), Sattentau(1996), D'Souza et al.(1997), Binley et al.(1997), Fouts et al.(1997), Hioe et al.(1997), Boots et al.(1997), Parren et al.(1997b), Hill et al.(1997), Inouye et al.(1998), Mondor et al.(1998), Smith et al.(1998), Parren et al.(1998), Connor et al.(1998), Gorny et al.(1998)]

NOTES:

- 447-52D: Also called 447/52-DII, 447-52-D, 447d, 447-52-D, and 447-D (per. comm. S. Zolla-Pazner)
- 447-52D: Requires GPXR at the tip of the V3 loop – neutralizes a broad array of B clade lab isolates [Gorny et al.(1992)]
- 447-52D: 60-fold increase in neutralization potency when combined 1:1 with human MAb 588-D [Buchbinder et al.(1992)]
- 447-52D: Reacts with MN, NY5, CDC4, SF2, RF, WM52, and HXB2 [Karwowska et al.(1992b)]
- 447-52D: Neutralizes MN and IIIB: GPGR, and binds SF2: GPGR [Gorny et al.(1993)]
- 447-52D: Peptide phage library showed that any of the residues ADGLMNQRS in the X position tolerated in peptides that react well with the antibody [Keller et al.(1993)]

428 cont.

- 447-52D: Additive neutralization of MN and SF2 when combined with CD4 binding site MAb F105 – supra-additive neutralization of RF [Cavacini et al.(1993a)]
- 447-52D: Complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear et al.(1993)]
- 447-52D: Requires GPxR at the tip of the V3 loop, common in B clade – neutralized primary isolates [Conley et al.(1994a)]
- 447-52D: Neutralization synergy in combination with CD4 binding domain MAbs [Laal et al.(1994)]
- 447-52D: GPGQ in MAL resulted in enhanced dissociation – GPGQ in CM234 or K14T did not bind – binding affected by identity of amino acids flanking GPGR core [VanCott et al.(1994)]
- 447-52D: Mild oxidation of carbohydrate moieties does not alter binding [Gorny et al.(1994)]
- 447-52D: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore et al.(1994a)]
- 447-52D: Called 447d – Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau et al.(1995)]
- 447-52D: Called 447 – The tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot et al.(1995)]
- 447-52D: Ab-mediated activation of complement on HIV+ cells is higher than Ab independent activation – what has been termed “Ab independent” in fact results in part from IgM in normal human serum that is HIV-cross-reactive [Saarloos et al.(1995)]
- 447-52D: Serotyping study using flow-cytometry – bound only to GPGR V3 loop tips [Zolla-Pazner et al.(1995)]
- 447-52D: Neutralization of primary and prototype laboratory HIV-1 isolates using a resting cell assay enhances sensitivity [Zolla-Pazner & Sharpe(1995)]
- 447-52D: Binding affected by identity of amino acids flanking GPGR core – poor breadth of primary virus neutralization [Moore et al.(1995a)]
- 447-52D: Review: the V3 loop motif GPGR is not common outside subtype B isolates, MAb 19b is more cross-reactive [Moore & Ho(1995)]
- 447-52D: Neutralizing (- complement), no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]
- 447-52D: Called 447-52-D – The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits binding [Jagodzinski et al.(1996)]
- 447-52D: Neutralizes JR-FL – strongly inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 447-52D: Review: called 447-52-D – only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]
- 447-52D: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – many of these isolates had the GPGR motif at the apex of the V3 loop [D’Souza et al.(1997)]
- 447-52D: An antibody with “intermediate” avidity as assessed by urea elution

428 cont.

- 447-52D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 447-52D bound monomer, oligomer, and neutralized JRFL [Fouts et al.(1997)]
- 447-52D: Tested using a resting cell neutralization assay [Hioe et al.(1997)]
- 447-52D: Viral binding inhibition by 447-D was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 447-52D: Neutralizes TCLA strains but not primary isolates [Parren et al.(1997b)]
- 447-52D: Called 447 – gp120 can inhibit MIP-1α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill et al.(1997)]
- 447-52D: Called 447-D – 447-D resistance took longer to acquire in virus with the M184V substituted RT, and had the form (AAC N to TAC Y) at position 5 of the V3 loop, rather than the GPGR to GPGR resistance found with wildtype RT [Inouye et al.(1998)]
- 447-52D: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – 447-52D has an epitope involving the tip of the V3 loop, that was previously studied with this method [Keller et al.(1993)] – in Keller et al., with no competition, LxGPxR was the most common six-mer, 38% of the peptides – after competition with a gp120 IIIB ligand (QRGPGR)i, RGPxR was the most common and one peptide had the sequence QRGPGR, showing type specific mimotyopes can be enriched by strain specific ligand competition protocols [Boots et al.(1997)]
- 447-52D: Inhibits binding of Hx10 to both CD4 positive and negative HeLa cells [Mondor et al.(1998)]
- 447-52D: Called 447-52-D – The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 447-52D was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith et al.(1998)]
- 447-52D: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- 447-52D: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D [Connor et al.(1998)]
- 447-52D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 1324E was comparable to 447-52D [Gorny et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
429	59.1	gp120(308-313 MN)	gp120(314-319)	GPGRAF	L	cyclic V3 MN peptide	murine(IgG_1)

Donor: Mary White-Scharf and A. Profy, Repligen Corporation

References: [D'Souza et al.(1991), White-Scharf et al.(1993), Potts et al.(1993), Ghiara et al.(1993), Bou-Habib et al.(1994), D'Souza et al.(1994), Seligman et al.(1996), Ghiara et al.(1997), Smith et al.(1998)]

NOTES:

- 59.1: Called R/V3-59.1 – potent neutralizing MAb [D'Souza et al.(1991)]
- 59.1: Epitope defined by peptide reactivity and binding affinity with amino acid substitutions – GPGRAF [White-Scharf et al.(1993)]
- 59.1: Synergistic neutralization of MN when combined with sCD4 or the CD4BS MAb F105 [Potts et al.(1993)]
- 59.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 Fab fragment – contact residues IGPGRAF [Ghiara et al.(1993)]
- 59.1: Greater affinity for T-cell tropic strain T-CSF than the primary isolate JR-CSF, from which T-CSF was derived [Bou-Habib et al.(1994)]
- 59.1: Multi-lab study for antibody characterization and assay comparison – neutralizes MN and IIIB [D'Souza et al.(1994)]
- 59.1: Competition ELISAs with serial deletions produced longer estimate of epitope length than x-ray crystallography or Alanine substitution, RIHIGPGRAFYTT, suggesting significance of non-contact residues [Seligman et al.(1996)]
- 59.1: A conformationally restricted analog of the tip of the V3 loop was constructed and bound with Fab 59.1 – crystal stucture shows interactions between 59.1 and an MN peptide and 59.1 and the modified peptide are similar, but NMR studies reveal that the modified peptide is more ordered in solution, retaining the Fab bound form [Ghiara et al.(1997)]
- 59.1: The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 59.1 was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
430	50.1	gp120(V3 MN)	gp120(310-314)	RIHIG	L	V3 MN peptide	murine(IgG_1)

Donor: Mary White-Scharf, Repligen Corporation, Cambridge, MA

References: [D'Souza et al.(1991), White-Scharf et al.(1993), Potts et al.(1993), Ghiara et al.(1993), Rini et al.(1993), Bou-Habib et al.(1994), VanCott et al.(1994), Robert-Guroff et al.(1994), Moore et al.(1994b), VanCott et al.(1995), Fontenot et al.(1995), Seligman et al.(1996), Berman et al.(1997), LaCasse et al.(1998)]

NOTES:

- 50.1: Called R/V3-50.1 – potent neutralizing MAb [D'Souza et al.(1991)]
- 50.1: Epitope defined by peptide reactivity and changes affinity with amino acid substitutions – epitope RIHIGP [White-Scharf et al.(1993)]
- 50.1: No synergistic neutralization of MN when combined with CD4BS MAb F105 – isotype stated to be IgG_{2a} [Potts et al.(1993)]
- 50.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 and 50.1 Fab fragments – epitope KRIHIGP [Ghiara et al.(1993)]
- 50.1: Crystal structure of V3 loop bound to 50.1 – light chain binds just to the left of GPG, heavy chain binds further to the left [Rini et al.(1993)]
- 50.1: No neutralization of primary isolate JR-CSF – greater affinity for and neutralization of T cell tropic strain T-CSF, derived from JR-CSF [Bou-Habib et al.(1994)]
- 50.1: Potent MN neutralization, slow dissociation rate [VanCott et al.(1994)]
- 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FACS signal, Ab affinity, and viral neutralization [Robert-Guroff et al.(1994)]
- 50.1: Shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore et al.(1994b)]
- 50.1: Used to monitor HIV-1 Env expression in infected H9 cells [VanCott et al.(1995)]
- 50.1: Competition ELISAs with serial deletions produced comparable estimate of epitope length to crystal structure and alanine substitution – KRIHIGP [Seligman et al.(1996)]
- 50.1: Binds to 6/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)]
- 50.1: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by anti-V3 MAbs. The primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse et al.(1998)]
- 50.1: NIH AIDS Research and Reference Reagent Program: 1289

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
431	58.2	gp120(V3 MN)	gp120(312-319)	HIGPGRAF	L	MN V3 peptide	murine(IgG_1)

Donor: Mary White-Scharf, Repligen Corporation, Cambridge, MA
References: [White-Scharf et al.(1993), Potts et al.(1993), Moore et al.(1994b), Seligman et al.(1996)]
NOTES:
- 58.2: Epitope defined by peptide reactivity and changes in affinity with amino acid substitutions – 4/7 primarily isolates were neutralized [White-Scharf et al.(1993)]
- 58.2: Did not synergistically neutralize MN in combination with MAb F105 – there was synergistic neutralization when combined with sCD4 [Potts et al.(1993)]
- 58.2: Modest cross-reactivity among B clade gp120s, little outside B clade – core epitope as I-IHIG [Moore et al.(1994b)]
- 58.2: Competition ELISAs with serial deletions produced longer estimates of epitope length, RIHIGPGRAFY, than Alanine substitution, suggesting significance of non-contact residues [Seligman et al.(1996)]

432	Nea 9301	gp120(V3 IIIB)	gp120(312-327)	RIQRGPGRAFVTIGKI			murine

Donor: Dupont, commercial
References: [Wagner et al.(1996)]
NOTES:
- 694/98-D:

433	694/98-D	gp120(V3 IIIB)	gp120(316-319)	GRAF	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Drs. S. Zolla-Pazner and M. Gorny, NYU Med Center NY, NY
References: [Gorny et al.(1992), Gorny et al.(1993), Cavacini et al.(1993a), Spear et al.(1993), Gorny et al.(1994), Laal et al.(1994), VanCott et al.(1994), Cook et al.(1994), VanCott et al.(1995), Zolla-Pazner et al.(1995), Forthal et al.(1995), Li et al.(1997), Zolla-Pazner et al.(1997), Smith et al.(1998), Li et al.(1998), Andrus et al.(1998)]
NOTES:
- 694/98-D: Also called 694/98
- 694/98-D: MAb first described [Skinner et al.(1988b)]
- 694/98-D: Type-specific lab isolate neutralization was observed – binds with 1-3 fold greater affinity to gp120 than to peptides [Gorny et al.(1992)]
- 694/98-D: Neutralizes MN and IIIB (GRAF) – binds SF2 (GRAF) – binding reactivity: MN, IIIB, SF2, NY5, RF, CDC4, WM52 [Gorny et al.(1993)]
- 694/98-D: Called 694-D – complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear et al.(1993)]

433 cont.

- 694/98-D: 50% neutralization of HIV-IIIB at a concentration of 0.15μg/ml [Gorny et al.(1994)]
- 694/98-D: Potent neutralization of IIIB – no neutralization synergy in combination with CD4 binding domain MAbs [Laal et al.(1994)]
- 694/98-D: GRVY did not alter peptide binding – GRVI and GQAW enhanced dissociation – GQVF and GQAL did not bind [VanCott et al.(1994)]
- 694/98-D: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – V3 MAbs can inhibit gp120 binding to GalCer *in vitro* – binding of GalCer to gp120 inhibited but did not completely block MAb binding[Cook et al.(1994)]
- 694/98-D: Human HIV-1 infected sera and MAb 694/98 have high reactivity to MN and RF infected H9 cells, but Genentech rec gp120 IIIB vaccine recipients do not [VanCott et al.(1995)]
- 694/98-D: Serotyping study using flow-cytometry – bound GRAX bearing virus in 10/11 cases – somewhat conformation dependent [Zolla-Pazner et al.(1995)]
- 694/98-D: ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]
- 694/98-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – could only achieve 50% neutralization alone – all Ab combinations tested showed synergistic neutralization – 694/98-D has synergistic response with MAbs F105, 15e, b12, 2F5, 17b, 2G12, and 48d, and with HIVIG [Li et al.(1997)]
- 694/98-D: Used to study pre- and post-exposure prophylaxis Hu-PBL-SCID mice infected by an intraperitoneal injection of HIV-1 LAI – MAb half life in plasma in mice is 9 days – 2 hours post-694/98-D mice were challenged with LAI, and at an Ab concentration of 1.32 mg/Kg, 50% of the mice were infected, and one of the infected mice carried the resistant form GRTF rather than GRAF (critical amino acids for binding are GRA) – post-exposure prophylaxis was effective if delivered 15 min post-exposure, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus et al.(1998)]
- 694/98-D: The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 694/98-D was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith et al.(1998)]
- 694/98-D: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) [Li et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
434	9205	gp120(V3 IIIB)	gp120(317-319)	RAF (core reactivity)	L	IIIB V3 Peptide	murine(IgG_1)

Donor: NEN, Boston MA, commercial

References: [Durda et al.(1990), Trujillo et al.(1993), Allaway et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)]

NOTES:

- 9205: Called NEA-9205, epitope RIQRGPGRAFVTIGK – reacts with three human brain proteins of 35, 55, 110 kDa molecular weight – similar to 9284 [Trujillo et al.(1993)]
- 9205: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)]
- 9205: Neutralizes IIIB but not MN – significantly slower dissociation constant for IIIB than MN [VanCott et al.(1994)]

435	N11-20	gp120(V3 317-325)	gp120(314-322)	GPGRAFVTI	L (LAI)	unk	murine($IgG_{1\kappa}$)

Donor: J. C. Mazie, Hybridolab, Institut Pasteur

References: [Valenzuela et al.(1998)]

NOTES:

- N11-20: Also called 110-H
- N11-20: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11-20 is through inhibition of virus binding to the cell [Valenzuela et al.(1998)]

436	902	gp120(V3 IIIB)	gp120(315-326)	PGRAFVTIGKIG	L	vaccinia-gp160 IIIB	murine($IgG_{1\kappa}$)

Donor: Bruce Chesebro, Rocky Mountain National Laboratory, Montana

References: [Chesebro & Wehrly(1988), Laman et al.(1993), Broder et al.(1994), Earl et al.(1994), Sakaida et al.(1997)]

NOTES:

- 902: Strain specific neutralization of HIV [Chesebro & Wehrly(1988)]
- 902: Epitope may be partially masked or altered in the oligomeric molecule [Broder et al.(1994)]
- 902: Used as a control in a study of the influence of oligomeric structure of Env in determining the repertoire of the Ab response [Earl et al.(1994)]
- 902: V3-BH10 peptide with loop-structure inhibits IL-2 induced T-cell proliferation, thought to be due to altering intracellular signaling, and MAb 908 can block the peptide inhibition [Sakaida et al.(1997)]
- 902: NIH AIDS Research and Reference Reagent Program: 522

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
437	IIIB-V3-01	gp120(V3 IIIB)	gp120(322-330)	IGKIGNMRQ	N	IIIB carboxy-terminus V3-loop peptide	murine(IgG_1)
		Donor: Jon Laman **References:** [Laman et al.(1993)] **NOTES:** • IIIB-V3-01: Specific for carboxy-terminal flank of the IIIB V3 loop – epitope is hidden native gp120, exposed on denaturation [Laman et al.(1993)] • IIIB-V3-01: UK Medical Research Council AIDS reagent: ARP3046 • IIIB-V3-01: NIH AIDS Research and Reference Reagent Program: 1726					
438	9305	gp120(V3)	gp120		L		murine
		Donor: Du Pont, Wilmington DE **References:** [McDougal et al.(1996)]					
439	D/6D1	gp120(V4 351-382 LAI)	gp120(350-431)	ASKLREQFGNNKTIIFK-QSSGGDPEIVTHSFN	N	Baculovirus-expressed rgp120 LAI	murine(IgG_1)
		Donor: Du Pont, Wilmington DE **References:** [Bristow et al.(1994)] **NOTES:** • D/6D1: V4 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]					
440	4D7/4	gp120(V4 361-380 LAI)	gp120(364-384)	IFKQSSGGDPEIVTHSF-NCGG		Env glycopro	murine(IgG)
		Donor: S. Ranjbar, NIBSC, UK **References:** [Moore et al.(1994c)] **NOTES:** • 4D7/4: C3 region – the relative affinity for denatured/native gp120 is >10 [Moore et al.(1994c)] • 4D7/4: UK Medical Research Council AIDS reagent: ARP3051					
441	36.1(ARP 329)	gp120(V4 362-381 LAI)	gp120(365-385)	FKQSSGGDPEIVTHSFN-CGGE		Env glycopro	murine(IgG)
		Donor: S. Ranjbar, NIBSC, UK **References:** [Thiriart et al.(1989), Moore et al.(1994c)] **NOTES:** • 36.1: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P impair binding [Moore et al.(1994c)] • 36.1: UK Medical Research Council AIDS reagent: ARP329					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
442	C12	gp120(V4 362-381 LAI)	gp120(365-385)	FKQSSGGDPEIVTHSFN-CGGE		mis-folded LAI rgp160	murine(IgG_1)

Donor: George Lewis
References: [Moore & Ho(1993), Moore et al.(1994c), Abacioglu et al.(1994), Moore et al.(1994d)]
NOTES:
- C12: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- C12: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P, and 384 Y/E impair binding – also binds GEFFYCNSTQLFNS, gp120(380-393 LAI) [Moore et al.(1994c)]
- C12: C3 region – epitope boundaries mapped by peptide scanning, core FNCGG [Abacioglu et al.(1994)]

443	110.D	gp120(C3 380-393 LAI)	gp120(384-397)	GEFFYCNSTQLFNS	N	Env glycopro	murine(IgG)

Donor: F. Traincard, Pasteur Institute, France
References: [Moore et al.(1994c), Valenzuela et al.(1998)]
NOTES:
- 110.D: The relative affinity for denatured/native gp120 is >50 [Moore et al.(1994c)]

444	B32	gp120(380-393 LAI)	gp120(384-397)	GEFFYCNSTQLFNS		mis-folded LAI rgp160	murine(IgG_1)

Donor: F. Traincard, Pasteur Institute, France
References: [Moore et al.(1994c), Abacioglu et al.(1994)]
NOTES:
- B32: The relative affinity for denatured/native gp120 is >100 – mutations 380 G/F, 381 G/P, 382 F/L, 384 Y/E, and 386 N/R impair binding [Moore et al.(1994c)]
- B32: C3 region – epitope boundaries mapped by peptide scanning – FFY(core) [Abacioglu et al.(1994)]

445	B2C	gp120(C3 HIV2ROD)	gp120	HYQ(core)	L	Peptide	murine

Donor: F. Traincard, Pasteur Institute, France
References: [Matsushita et al.(1995)]
NOTES:
- B2C: Viral neutralization was type-specific for HIV-2 ROD [Matsushita et al.(1995)]

446	2H1B	gp120(C3 370-376 HIV2ROD)	gp120(361-367)	RNISFKA	N	Peptide	murine

Donor: F. Traincard, Pasteur Institute, France
References: [Matsushita et al.(1995)]
NOTES:
- 2H1B: Binds in WB, but binds poorly to Env on the cell surface [Matsushita et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
447	2F19C	gp120(C3 HIV2ROD)	gp120	APGK(core)	N	Peptide	murine
		Donor: F. Traincard, Pasteur Institute, France **References:** [Matsushita et al.(1995)] **NOTES:** • 2F19C: Binds in WB, but binds poorly to Env on the cell surface [Matsushita et al.(1995)]					
448	B15	gp120(V4 395-400 BH10)	gp120(394-399)	WFNSTW		mis-folded LAI rgp160	murine(IgG_{2b})
		Donor: George Lewis **References:** [Moore & Ho(1993), Moore et al.(1993b), Abacioglu et al.(1994)] **NOTES:** • B15: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • B15: Binds native BH10 gp120 with 5 fold less affinity than denatured – does not bind native or denatured MN gp120 [Moore et al.(1993b)] • B15: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
449	B34	gp120(V4 395-400 BH10)	gp120(394-399)	WFNSTW		mis-folded LAI rgp160	murine(IgG_{2b})
		Donor: George Lewis **References:** [Abacioglu et al.(1994)] **NOTES:** • B34: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
450	7F11	gp120(397-439 IIIB)	gp120(396-440)	?		purified gp120	murine
		Donor: George Lewis **References:** [Lasky et al.(1987), Nilsen et al.(1996)] **NOTES:** • 7F11: There is another MAb with this name that binds to integrase [Nilsen et al.(1996)]					
451	5C2E5	gp120(C4 406-415 IIIB)	gp120(423-432)	QFINMWQEVK		purified gp120	murine
		Donor: T. Gregory and R. Ward, Genentech, San Francisco **References:** [Lasky et al.(1987), Cordell et al.(1991)] **NOTES:** • 5C2E5: Blocks the gp120-CD4 interaction [Lasky et al.(1987)] • 5C2E5: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell et al.(1991)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
452	G3-211	gp120(C4 423-437 IIIB)	gp120(424-438)	IINMWQKVGKAMYAP	L	virus derived IIIB gp120	murine(IgG_1)

Donor: T. Gregory and R. Ward, Genentech, San Francisco
References: [Sun et al.(1989)]
NOTES:
- G3-211, 42, 299, 508, 519, 536, 537: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun et al.(1989)]

453	G3-537	gp120(C4 423-437 IIIB)	gp120(424-438)	IINMWQKVGKAMYAP	L	virus derived IIIB gp120	murine(IgG_1)

Donor: T. Gregory and R. Ward, Genentech, San Francisco
References: [Sun et al.(1989), Ho et al.(1991b), McKeating et al.(1992b)]
NOTES:
- G3-537, 211, 299, 508, 519, 536, 42: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun et al.(1989)]
- G3-537: Weakly neutralizing – binds to a linear binding domain of gp120, NMWQEVGKAMYAPPISG [McKeating et al.(1992b)]

454	polyclonal	gp120(CD4BS)	gp120(426-437)	NMWQEVGKAMYA	L	oral immunization – peptide plus cholera toxin adjuvant	murine(IgA)

Donor: T. Gregory and R. Ward, Genentech, San Francisco
References: [Bukawa et al.(1995)]
NOTES:
- Polyclonal secretory IgA antibody raised by mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to the V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa et al.(1995)]

455	MO86/C3	gp120(C4 429-443)	gp120(430-444)	EVGKAMYAPPISGQI		rIIIB Env 286-467	human(IgM)

Donor: T. Gregory and R. Ward, Genentech, San Francisco
References: [Ohlin et al.(1992)]
NOTES:
- MO86: Generated through *in vitro* “immunization” of uninfected-donor lymphocytes [Ohlin et al.(1992)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
456	G3-42	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun et al.(1989), Moore et al.(1993b), Thali et al.(1993), Sattentau & Moore(1995), Jagodzinski et al.(1996), Moore & Sodroski(1996), Poignard et al.(1996a), Trkola et al.(1996a), Binley et al.(1997)]

NOTES:

- G3-42: Neutralization of IIIB but not RF [Sun et al.(1989)]
- G3-42: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – G3-42, G3-299 have lower affinity than G3-508, G3-519, and G3-536 – bound native gp120, not denatured – poor peptide binding, epitope spans V3-C4 regions – 433A/L, 435Y/H and 430V/S substitutions impaired binding, V3 loop insertion abolished binding [Moore et al.(1993b)]
- G3-42: Inhibits binding of CD4 inducible MAb 48d [Thali et al.(1993)]
- G3-42: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-42: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS potently inhibits G3-42 binding – G3-42 epitope described as KVGKAMYAPP [Jagodzinski et al.(1996)]
- G3-42: Inhibits binding of many anti-V3, -CD4 binding site, and -C4 region MAbs – enhances binding of some anti-V2 region MAbs [Moore & Sodroski(1996)]
- G3-42: Epitope described as KQIINMWQKVGKAMYAPPIS – binding resulted in slight gp120 dissociation from virus and exposure of the gp41 epitope for MAb 50-69 [Poignard et al.(1996a)]
- G3-42: Called G3 42 – Does not inhibit gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study – described as V3-C4 discontinuous epitope [Trkola et al.(1996a)]
- G3-42: A low avidity antibody as assessed by urea elution

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
457	G3-299	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

Donor: M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun et al.(1989), Moore et al.(1993b), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Ditzel et al.(1997), Wyatt et al.(1997), Parren et al.(1998)]

NOTES:

- G3-299: Best neutralization of IIIB in panel of 7 MAbs that bind overlapping epitope [Sun et al.(1989)]
- G3-299: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – G3-42, G3-299 lower affinity than G3-508, G3-519, and G3-536 – bound native gp120, not denatured – poor peptide binding, epitope spans V3-C4 regions – 433A/L, 435Y/H and 430V/S substitutions impaired binding, V3 loop cleavage or insertion abolished binding [Moore et al.(1993b)]
- G3-299: Binds with higher affinity to monomer than to oligomer, slow association rate, although faster than other C4 MAbs tested, with more potent neutralization of lab strain [Sattentau & Moore(1995)]
- G3-299: Discontinuous V3-C4 epitope, binding enhanced by a few anti-C1, anti-CD4 binding site, and V2 MAbs – binding reciprocally inhibited by anti-V3 MAbs – G3-229 enhances the binding of some anti-V2 MAbs [Moore & Sodroski(1996)]
- G3-299: Epitope described as KQIINMWQKVGKAMYAPPIS – binding resulted in slight gp120 dissociation from virus and exposure of the gp41 epitope for MAb 50-69 [Poignard et al.(1996a)]
- G3-299: A low avidity antibody as assessed by urea elution
- G3-299: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)]
- G3-299: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
458	G3-508	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

Donor: M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun et al.(1989), Thali et al.(1993), Moore et al.(1993b), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Trkola et al.(1996a), Binley et al.(1997), Parren et al.(1998), Binley et al.(1998)]

NOTES:

- G3-508: Neutralization of IIIB and RF [Sun et al.(1989)]
- G3-508: Inhibits binding of CD4 inducible MAb 48d [Thali et al.(1993)]
- G3-508: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 10 fold greater affinity than native – 433A/L, 435Y/H and 430V/S substitutions impaired binding [Moore et al.(1993b)]
- G3-508: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-508: Inhibits binding of some V3, C4 and CD4 binding site MAbs, enhances binding of V2 region MAbs [Moore & Sodroski(1996)]
- G3-508: Binding resulted in slight gp120 dissociation from virus and exposure of the gp41 epitope for MAb 50-69 [Poignard et al.(1996a)]
- G3-508: Also called G3 508 – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- G3-508: A low avidity antibody as assessed by urea elution
- G3-508: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- G3-508: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
459	G3-519	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun et al.(1989), Moore & Ho(1993), Moore et al.(1993b), D'Souza et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Wyatt et al.(1997), Parren et al.(1998)]

NOTES:

- G3-519: Best neutralization of RF in panel of 7 MAbs that bind overlapping epitope [Sun et al.(1989)]
- G3-519: Neutralizes IIIB, is reactive with SF-2 gp120, mild inhibition of HIV-1+ sera binding to IIIB gp120 [Moore & Ho(1993)]
- G3-519: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 5 fold greater affinity than native – 433A/L, 435Y/H, 438P/R and 430V/S substitutions impaired binding [Moore et al.(1993b)]
- G3-519: Included in a multi-lab study for antibody characterization, and binding and neutralization assay comparison, also binds IIIB: IINMWQKVGKAMYAPP [D'Souza et al.(1994)]
- G3-519: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-519: Non-reciprocal enhanced binding in the presence of the C5 MAb 1C1 and the C1 MAb 135/9 – reciprocal enhanced binding with some V2 MAbs. Inhibited binding the presence of some C4, V3 and CD4 binding site MAbs [Moore & Sodroski(1996)]
- G3-519: Epitope described as KVGKAMYAPP – binding resulted in slight gp120 dissociation from virus but no significant exposure of the gp41 epitope for MAb 50-69 [Poignard et al.(1996a)]
- G3-519: A low avidity antibody as assessed by urea elution
- G3-519: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)]
- G3-519: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
460	G3-536	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun et al.(1989), Ho et al.(1991b), Cordell et al.(1991), McKeating et al.(1992b), Moore & Ho(1993), Moore et al.(1993b), Gorny et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Parren et al.(1998)]

NOTES:

- G3-536: Weak neutralization of IIIB and RF – cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – epitope:IINMWQKVGKAMYAP [Sun et al.(1989)]
- G3-536: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell et al.(1991)]
- G3-536: Weakly neutralizing – binds to a linear determinant in the CD4 binding domain of gp120 [McKeating et al.(1992b)]
- G3-536: Neutralizes IIIB, is reactive with SF-2 gp120, mild inhibition of HIV-1+ sera binding to IIIB gp120 [Moore & Ho(1993)]
- G3-536: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 15 fold greater affinity than native – 433A/L, 435Y/H, 438P/R, and 430V/S substitutions impaired binding [Moore et al.(1993b)]
- G3-536: Enhances binding of anti-V2 MAb 697-D [Gorny et al.(1994)]
- G3-536: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-536: Inhibits binding of some V3, C4 and CD4 binding site MAbs, enhances binding of V2 region MAbs [Moore & Sodroski(1996)]
- G3-536: Epitope described as KVGKAMYAPP [Poignard et al.(1996a)]
- G3-536: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
461	ICR38.1a	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	rBH10 gp120	rat(IgG_{2b})

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Cordell et al.(1991), McKeating et al.(1992b), McKeating et al.(1992a), McKeating et al.(1992), McKeating et al.(1993b), McKeating et al.(1993a), Moore et al.(1993b), Jeffs et al.(1996)]

NOTES:

- ICR38.1a: Also called 38.1a
- ICR38.1a: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with MAbs G3-536, 5C2E5, and ICR38.8f [McKeating et al.(1992b), Cordell et al.(1991)]
- ICR38.1a: Unable to exert a synergistic effect in combination with V3 directed MAbs, in contrast to MAb 39.13g, that binds to a conformational epitope involved in CD4 binding [McKeating et al.(1992a)]
- ICR38.1a: Studied in the context of a neutralization escape mutant [McKeating et al.(1993a)]
- ICR38.1a: Unreactive with solid-phase decapeptide, competed in solution phase assay – ICR 38.1a and ICR 38.8f were initially reported to be independent MAbs, but are actually subclones of the same MAb [Moore et al.(1993b)]
- ICR38.1a: Called 38.1a – 10 to 20 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]
- ICR38.1a: UK Medical Research Council AIDS reagent: ARP388/ARP389

462	ICR38.8f	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	rBH10 gp120	rat(IgG_{2b})

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Cordell et al.(1991)]

NOTES:

- ICR38.8f: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with ICR38.1a, 5C2E5, and G3-536 [Cordell et al.(1991)]
- ICR38.8f:ICR 38.1a and ICR 38.8f were initially reported to be independent MAbs, but are actually subclones of the same MAb [Moore et al.(1993b)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
463	G45-60	gp120(C4 429-438 BRU)	gp120(432-441)	GKAMYAPPIS	L	virus derived IIIB gp120	murine(IgG_1)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Sun et al.(1989), Moore et al.(1993b), Gorny et al.(1994), Moore & Sodroski(1996), Jagodzinski et al.(1996)]
NOTES:

- G45-60: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPI, decapeptide flanking peptides also bound – bound equivalently to native and denatured gp120 – 433A/L and 435Y/H (not 430V/S) substitutions impaired binding [Moore et al.(1993b)]
- G45-60: Enhances binding of anti-V2 MAb 697-D [Gorny et al.(1994)]
- G45-60: Non-reciprocal enhancement of G45-60 binding by some C1 and C5 antibodies – reciprocal enhancement of some V2 region MAbs – reciprocal inhibition with many MAbs that bind to the V3, C4 and CD4 binding site regions [Moore & Sodroski(1996)]
- G45-60: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus CRDS inhibits G45-60 binding [Jagodzinski et al.(1996)]

464	1662	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	N	poliovirus-antigen chimera	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [McKeating et al.(1992b)]
NOTES:

- 1662: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]

465	1663	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	N	poliovirus-antigen chimera	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [McKeating et al.(1992b)]
NOTES:

- 1663: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]

466	1664	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	N	poliovirus-antigen chimera	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [McKeating et al.(1992b)]
NOTES:

- 1664: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
467	1697	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	N	poliovirus-antigen chimera	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [McKeating et al.(1992b)]
NOTES:
- 1697: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]

468	1794	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	N	poliovirus env chimera	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [McKeating et al.(1992b)]
NOTES:
- 1794: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]

469	1804	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	N	poliovirus env chimera	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [McKeating et al.(1992b)]
NOTES:
- 1804: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]

470	1807	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	N	poliovirus env chimera	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [McKeating et al.(1992b)]
NOTES:
- 1807: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]

471	1808	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	N	poliovirus env chimera	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [McKeating et al.(1992b)]
NOTES:
- 1808: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
472	1795	gp120(CD4BS 425-441 IIIB)	gp120(426-442)	0PISG	L	poliovirus env chimera	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [McKeating et al.(1992b)]
NOTES:
- 1795: CD4 binding site – weakly neutralizing – binding inhibited by WQEVGKAMYA, GKAM may be involved [McKeating et al.(1992b)]

473	13H8	gp120(C4 412-453)	gp120(432-441)	GKAMYAPPIS	L	rgp120 MN	murine(IgG)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Nakamura et al.(1992), Nakamura et al.(1993), Jeffs et al.(1996)]
NOTES:
- 13H8: Cross blocks 5C2 in IIIB-rsgp160 ELISA – reactive with diverse strains in rgp120 ELISA [Nakamura et al.(1992)]
- 13H8: Bound diverse strains, neutralizing activity against MN [Nakamura et al.(1993)]
- 13H8: Binds V3 and C4 peptides (J. P. Moore, per. comm.)
- 13H8: 3 and 4.5 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120, respectively [Jeffs et al.(1996)]

474	1024	gp120(C4)	gp120				

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Berman et al.(1997)]
NOTES:
- 1024: Binds to 1/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)]

475	polyclonal	gp120(V5 LAI)	gp120(462-469)	NNNNGSEI		HIV-1 infection augmented by gp160 vaccine	human

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Loomis-Price et al.(1997)]
NOTES:
- HIV-1+ positive individuals were given a gp160 vaccine as immunotherapy, and this region was the most reactive new epitope as measured by a modified Pepscan technique which improved sensitivity – 4/14 showed vaccine-induced reactivity [Loomis-Price et al.(1997)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
476	M91	gp120(V5 C5 451-470 LAI)	gp120(463-472)	SNNESEIFRL	N	451 Env	rat(IgG_{2a})

Donor: Fulvia di Marzo Veronese

References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996), Ditzel et al.(1997), Binley et al.(1998)]

NOTES:

- M91: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)]
- M91: The relative affinity for denatured/native gp120 is 24 – mutation in position 470 P/L impairs binding [Moore et al.(1994c)]
- M91: 470 P/L impairs binding, but not 475 D/V, in contrast to CRA1 – some C2 mutations can enhance binding [Moore et al.(1994d)]
- M91: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – M91 binding was enhanced by 1C1, but 1C1 binding was inhibited by M91 – non-reciprocal binding enhancement of C1 and V2 antibodies – non-reciprocal binding inhibition of CD4 binding site antibodies [Moore & Sodroski(1996)]
- M91: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

477	CRA1(ARP 323)	gp120(V5-C5 451-470 LAI)	gp120(463-472)	SNNESEIFRL	N	Env glycopro	murine(IgG)

Donor: M. Page, NIBSC, UK

References: [Moore & Ho(1993), Moore et al.(1994d), Moore et al.(1994c), Moore & Sodroski(1996), Trkola et al.(1996a)]

NOTES:

- CRA1: Also called CRA-1
- CRA1: Bound preferentially to denatured IIIB and SF2 gp120 [Moore & Ho(1993)]
- CRA1: Some C5 mutations abrogate binding 470 P/L or G, 475 M/S, some C2 mutations enhance binding [Moore et al.(1994d)]
- CRA1: The relative affinity for denatured/native gp120 is 24 – C5 mutations 470 P/L or G, 475 M/S impairs binding to the native gp120 – only mutation 470 P/L impairs binding to denatured [Moore et al.(1994c)]
- CRA1: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – reciprocal binding inhibition with anti-C5 antibodies 1C1 and M91 – non-reciprocal binding enhancement some C1 and V2 antibodies – non-reciprocal binding inhibition of some CD4 binding site antibodies [Moore & Sodroski(1996)]
- CRA1: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- CRA1: UK Medical Research Council AIDS reagent: ARP323

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
478	9301	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY-KVVK		Env glycopro	murine(IgG)

Donor: Dupont, commercial
References: [Skinner et al.(1988b), Moore & Ho(1993), Moore et al.(1994c), Moore et al.(1994d), Wagner et al.(1996)]
NOTES:
- 9301: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- 9301: The relative affinity for denatured/native gp120 is 19 [Moore et al.(1994d)]
- 9301: Wagner et al. claim that Nea 9301 is anti-V3 – might they have meant MAb 9305? [Wagner et al.(1996)]

481	H11	gp120(C5 472-477 HXB2)	gp120(474-479?)	GGDMRD?			murine

References: [Pincus & McClure(1993), Pincus et al.(1996)]
NOTES:
- H11: Binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)]

482	M38	gp120(C5 490-508)	gp120(487-506)	KYKVVKEIPLGVAPTKA-KRR	N	IIIB immunization	murine

References: [Beretta et al.(1987), Grassi et al.(1991), Lopalco et al.(1993), DeSantis et al.(1994), Beretta & Dalgleish(1994)]
NOTES:
- M38: Binds to gp120 and to a 80 kd human protein expressed on a small fraction of mononuclear cells in the lymph nodes [Beretta et al.(1987)]
- M38: Binds to the carboxy terminus of gp120, in a gp41 binding region, and also to denatured human HLAs (antigenic homology) [Lopalco et al.(1993)]
- M38: Infected individuals have HLA class I-gp120 cross-reactive antibodies [DeSantis et al.(1994)]

483	1C1	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY-KVVK		Env glycopro	murine (IgG)

Donor: Repligen Inc, Cambridge, MA, commercial
References: [Moore et al.(1994c), Moore et al.(1994d), VanCott et al.(1995), Moore & Sodroski(1996)]
NOTES:
- 1C1: The relative affinity for denatured/native gp120 is 15 [Moore et al.(1994c)]
- 1C1: C2 and V3 regions substitutions can influence binding [Moore et al.(1994d)]
- 1C1: Linear epitope not exposed on conformationally intact gp120 [VanCott et al.(1995)]
- 1C1: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – M91 binding was enhanced by 1C1, but 1C1 binding was inhibited by M91 – non-reciprocal binding enhancement of some C1 and V2 antibodies – non-reciprocal binding inhibition of some CD4 binding site antibodies [Moore & Sodroski(1996)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
484	B221	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY-KVVK		Baculovirus-expressed misfolded rgp160 IIIB:NL43, MicroGenSys	murine($IgG_{1\kappa}$)

Donor: Rod Daniels
References: [Moore & Ho(1993), Bristow et al.(1994), Moore et al.(1994c)]
NOTES:

- B221: Called 221 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- B221: MAbs generated in the context of a study of the humoral immune response to rgp120 and rgp160 – boundaries described as 443-462 of LAI [Bristow et al.(1994)]
- B221: The relative affinity for denatured/native gp120 is 12 – mutation 477 D/V impairs binding [Moore et al.(1994c)]
- B221: Called 221 – C2 and V3 substitutions influence binding [Moore et al.(1994d)]
- B221: UK Medical Research Council AIDS reagent: ARP301

485	660-178	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY-KVVK		Env glycopro	murine(IgG)

Donor: G. Robey, Abbott Labs
References: [Moore et al.(1994c), Moore et al.(1994d)]
NOTES:

- 660-178: The relative affinity for denatured/native gp120 is >100 [Moore et al.(1994c)]
- 660-178: ΔV1/V2 and ΔV1/V2/V3 reduce binding – C2 and C5 mutations enhance binding [Moore et al.(1994d)]

486	8C6/1	gp120(V5-C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY-KVVK		Env glycopro	murine(IgG)

Donor: S. Ranjbar, NIBSC, UK
References: [Moore et al.(1994c)]
NOTES:

- 8C6/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>30 fold) – mutation 485 K/V impairs binding [Moore et al.(1994c)]
- 8C6/1: UK Medical Research Council AIDS reagent: ARP3052

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
487	5F4/1	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY-KVVK		Peptide	murine
		Donor: S. Ranjbar, NIBSC, UK **References:** [Moore et al.(1994c)] **NOTES:** • 5F4/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>10 fold) – mutation 485 K/V impairs binding [Moore et al.(1994c)]					
488	3F5	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY-KVVK		Env	murine(IgG)
		Donor: S. Nigida, NCI, USA **References:** [Moore et al.(1994c)] **NOTES:** • 3F5: The relative affinity for denatured/native gp120 is 100 [Moore et al.(1994c)]					
489	MO101/ V3,C4	gp120(V3 314-323 + C5 494-503)	gp120	GRAFVTIGKI + LGVA-PTKAKR		pB1 (IIIB Env 286-467)	human(IgM)
		Donor: S. Nigida, NCI, USA **References:** [Ohlin et al.(1992)] **NOTES:** • MO101: generated through *in vitro* “immunization” of uninfected-donor lymphocytes: reacts with peptides from the V3 and C4 regions [Ohlin et al.(1992)]					
490	9201	gp120(C5 475-486 LAI)	gp120(473-484)	GGGDMRDNWRSE?	N		murine
		Donor: Du Pont **References:** [McDougal et al.(1996)] **NOTES:** • 9201: Does not neutralize LAI [McDougal et al.(1996)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
491	W2	gp120(C5 472-491 LAI)	gp120(474-493)	GGDMRDNWRSELYKYK-VVKI		Env	murine(IgG)

Donor: D. Weiner, U. Penn., USA
References: [Moore et al.(1994c)]
NOTES:
- W2: The relative affinity for denatured/native gp120 is 30 – mutation 485 K/V impairs binding [Moore et al.(1994c)]

479	42F	gp120(C5 491-500 HXB2)	gp120(483-492)	IEPLGVAPTK	N	HIV-1 infection	human($IgG_1\lambda$)

References: [Alsmadi et al.(1997), Alsmadi & Tilley(1998)]
NOTES:
- 42F: 42F and 43F were isolated from a long term non-progressor by EBV transformation of PBMC – samples were taken 14 months apart – both MAbs stained diverse strains of infected cells and directed ADCC – were more potent for ADCC if the cell was infected with HIV-1, rather than just presenting absorbed gp120 [Alsmadi et al.(1997)]
- 42F: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against strains IIIB, MN, SF-2, and RF, but not a clone of MN [Alsmadi & Tilley(1998)]

480	43F	gp120(C5 491-500 HXB2)	gp120(483-492)	IEPLGVAPTK	N	HIV-1 infection	human($IgG_1\lambda$)

References: [Alsmadi et al.(1997)]
NOTES:
- 42F: 42F and 43F were isolated from a long term non-progressor by EBV transformation of PBMC – samples were taken 14 months apart – both MAbs stained diverse strains of infected cells and directed ADCC – were more potent for ADCC if the cell was infected with HIV-1, rather than just presenting absorbed gp120 [Alsmadi et al.(1997)]

492	Chim 1	gp120(C5 492-498 HXB2)	gp120(489-495)	KVVKEIP?			humanized chimpanzee

Donor: D. Weiner, U. Penn., USA
References: [Pincus & McClure(1993), Pincus et al.(1996)]
NOTES:
- Chim 1: Also called C-1
- Chim 1: binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)]

493	RV110026	gp120(C5 491-500 LAI)	gp120(493-502)	IEPLGVAPTK		Peptide	human

Donor: Commercial, Olympus Inc
References: [Moore et al.(1994c), Moore et al.(1994d)]
NOTES:
- RV110026: Preferentially binds SDS-DTT denatured gp120 (15 fold using R1/87 as capture reagent) [Moore et al.(1994c)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
494	110.1	gp120(C5 491-500 LAI)	gp120(493-502)	IEPLGVAPTK	N	BRU infected cell lysates	murine($IgG_{1\kappa}$)

Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas
References: [Gosting et al.(1987), Linsley et al.(1988), Kinney Thomas et al.(1988), Pincus et al.(1991), Moore et al.(1994c), Cook et al.(1994), McDougal et al.(1996), Binley et al.(1997), Valenzuela et al.(1998)]
NOTES:

- 110.1: There is another antibody with this ID that binds to gp120, but at aa 200-217 [Pincus et al.(1996)]
- 110.1: Referred to as 110-1 – does not inhibit CD4-gp120 binding or neutralize HIV-1 strains [Linsley et al.(1988)]
- 110.1: Difference in the epitope: mapped to aa 421-429 (KQIINMWQE), the T1 sequence – poor efficacy as an immunotoxin when linked to RAC [Pincus et al.(1991)]
- 110.1: The relative affinity for denatured/native gp120 is 0.7 [Moore et al.(1994c)]
- 110.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the carboxy-terminus of gp120 inhibit gp120 binding to GalCer but not as potently as anti-V3 MAbs – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]
- 110.1: Does not neutralize HIV-1 LAI [McDougal et al.(1996)]
- 110.1: A high avidity antibody as assessed by urea elution
- 110.1: Does effect LAI viral binding or entry into CEM cells [Valenzuela et al.(1998)]

495	GV1G2	gp120(494-499 IIIB)	gp120(496-501)	LGVAPT		gp120 complexed with MAb M77	murine

Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas
References: [Denisova et al.(1996)]
NOTES:

- GV1G2: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV12F6 and GV3H1 are homologous to GV1G2 and were generated in the same experiment [Denisova et al.(1996)]

496	722-D	gp120(C term 503-509)	gp120(505-511)	RRVVQRE	N	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas
References: [Laal et al.(1994), Forthal et al.(1995)]
NOTES:

- 722-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal et al.(1994)]
- 722-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
497	670-D	gp120(C term 503-509)	gp120(500-506)	PTKAKRR?	N	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas
References: [Zolla-Pazner et al.(1995), Forthal et al.(1995), Hill et al.(1997), Gorny et al.(1998)]
NOTES:
- 670-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner et al.(1995)]
- 670-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity, numbering provided suggests epitope is RRVVQRE [Forthal et al.(1995)]
- 257-D: Called 257 – gp120 can inhibit MIP-1α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill et al.(1997)]

498	450-D	gp120(C term 475-486 or 503-509 BH10)	gp120(500-506)	PTKAKRR (or RRVVQ-RE, or MRDNWRSELYKY depending on reference)	N	HIV-1 infection?	human($IgG_{1\lambda}$)

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY
References: [Durda et al.(1988), Karwowska et al.(1992a), Karwowska et al.(1992b), Spear et al.(1993), Laal et al.(1994), Gorny et al.(1994), Cook et al.(1994), Forthal et al.(1995), Manca et al.(1995), Li et al.(1997)]
NOTES:
- 450-D: Also called 450-D-3 and 450D
- 450-D: Bound to MN, SF-2 and IIIB, but was not neutralizing [Karwowska et al.(1992a)]
- 450-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear et al.(1993)]
- 450-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal et al.(1994)]
- 450-D: Epitope is defined as PTKAKRR [Gorny et al.(1994)]
- 450-D: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the carboxy-terminus of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]
- 450-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]
- 450-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca et al.(1995)]
- 450-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 6 μg/ml [Li et al.(1997)]

499	750-D	gp120(C term 503-509)	gp120(500-506)	PTKAKRR	N	HIV-1 infection	human($IgG_{3\lambda}$)

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY
References: [Forthal et al.(1995)]
NOTES:
- 750-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
500	120-1	gp120(C term 503-532)	gp120	?	N	Peptide	murine(IgM_{κ})
		Donor: ?					
		References: [Chanh et al.(1986), Dalgleish et al.(1988)]					
501	polyclonal	gp120(C term 508-516)	gp120(505-513)	RRVVQREKR		HIV-1 infection	human
		Donor: ?					
		References: [Palker et al.(1987), Loomis-Price et al.(1997)]					
		NOTES:					
		• Most HIV-1+ individuals have an antibody response to this epitope – in this study, reactivity to RRVVQREKR was used as a positive control for HIV-1+ gp160 vaccine recipients [Loomis-Price et al.(1997)]					
502	858-D	gp120(C term 510-516)	gp120(507-513)	VVQREKR	N	HIV-1 infection	human(IgG)
		Donor: ?					
		References: [Zolla-Pazner et al.(1995), Forthal et al.(1995)]					
		NOTES:					
		• 858-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner et al.(1995)]					
		• 858-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]					
504	989-D	gp120(C term)	gp120(507-513)	VVQREKR		HIV-1 infection	human(IgG)
		Donor: ?					
		References: [Zolla-Pazner et al.(1995)]					
		NOTES:					
		• 989-D: In serotyping study using flow-cytometry, showed B clade specificity, but only reacted with 7/11 B clade virus [Zolla-Pazner et al.(1995)]					
503	1131-A	gp120(C term 510-516 LAI)	gp120(507-513)	VVQREKR	N	HIV-1 infection	human($IgG_3\lambda$)
		Donor: ?					
		References: [Bandres et al.(1998)]					
		NOTES:					
		• 1131-A: A very high affinity antibody used in studies that demonstrate that CXCR4 can bind to gp120 in the absence of CD4-gp120 interactions, and that this binding can be enhanced by Env deglycosylation [Bandres et al.(1998)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
505	D7324	gp120(C term)	gp120	?		Peptide from the C-term	sheep

Donor: Aalto BioReagents Ltd, Dublin, Ireland
References: [Moore(1990), Sattentau & Moore(1991), Moore et al.(1993a), Moore et al.(1993b), Wyatt et al.(1995), Trkola et al.(1996a), Ditzel et al.(1997), Ugolini et al.(1997), Mondor et al.(1998), Binley et al.(1998)]
NOTES:

- D7324: Binding unaltered by gp120 binding to sCD4, in contrast to 110.5, 9284, 50-69 and 98-6 [Sattentau & Moore(1991)]
- D7324: Binds to the last 15 amino acids in gp120 – used for antigen capture ELISA [Wyatt et al.(1995)]
- D7324: Epitope in C5 – Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- D7324: Used to capture gp120 onto solid phase for epitope mapping [Moore et al.(1993a), Moore et al.(1993b), Ditzel et al.(1997), Binley et al.(1998)]

506	23A	gp120(C term)	gp120	?	N	?	

Donor: J. Robinson, Tulane University, LA
References: [Thali et al.(1992a), Thali et al.(1993), Wu et al.(1996), Trkola et al.(1996a), Fouts et al.(1997)]
NOTES:

- 23A: Called 2.3A – Did not block ability of gp120-sCD4 complexes to inhibit MIP-1α binding – binds to gp41-binding domain [Wu et al.(1996)]
- 23A: C5 binding MAb – does not inhibit gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 23A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – 23A bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]

507	8F101	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine(IgG)

Donor: J. Robinson, Tulane University, LA
References: [Devico et al.(1995)]
NOTES:

- 8F101: MAbs specifically reactive to crosslinked gp120 and CD4 were derived (8F101, 8F102) – conformation dependent – competition studies indicate the epitope is immunogenic in infected humans [Devico et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
508	8F102	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine(IgG)

Donor: J. Robinson, Tulane University, LA
References: [Devico et al.(1995)]
NOTES:

- 8F102: MAbs specifically reactive to crosslinked gp120 and CD4 were derived (8F101, 8F102) – conformation dependent – competition studies indicate the epitope is immunogenic in infected humans [Devico et al.(1995)]

509	CG-10	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	L	CD4/gp120 IIIB complex	murine(IgG_1)

Donor: Jonathan Gershoni, Tel Aviv University, Isreal
References: [Gershoni et al.(1993), Wu et al.(1996), Lee et al.(1997), Rizzuto et al.(1998), Sullivan et al.(1998)]
NOTES:

- CG-10: Also called CG10
- CG-10: Reacts exclusively with sCD4-gp120 complex, not with sCD4 or gp120 alone [Gershoni et al.(1993)]
- CG-10: Called CG10 – MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4, and MAb CG10 does not block this inhibition [Wu et al.(1996)]
- CG-10: Called CG10 – Promotes envelope mediated fusion between both T-cell and macrophage tropic viruses and CD4+ lines
- CG-10: Called CG10 – disrupts gp120-CCR5 interaction and competes with MAb 17b –binds near the conserved bridging sheet of gp120 – mutations in positions K/D 121, T/D 123, K/D 207, K/D 421, Q/L 422, Y/S 435, M/A 434, K/A 432 and I/S 423 result in a $\geq$ 70% reduction in CG10 binding [Rizzuto et al.(1998)]
- CG-10: Called CG10 – CD4BS MAb 15e competes with CG-10 binding, probably due to the disruption of CD4-gp120 by 15e – CD4i MAbs 17b and 48d compete and the binding sites may overlap – MAb A32 enhances binding of 17b, 48d and CG10 – MAbs C11, 2G12 and 212A do not affect CG10 binding – CG-10 can bind gp120 with V1/V2 and V3 deleted – HXBc2 mutations Δ 119-205, 314 G/W, 432 K/A, 183,184 PI/SG decrease CG-10 recognition, HXBc2 mutations Δ 298-327 (V3), 384 Y/E, 298 R/G, 435 Y/S enhance recognition – the CD4 contribution to the CG10 epitope maps to the CD4 CDR2-like loop – CG10 can neutralize HIV-1 in the presence of sCD4 even though it does not do so in the context of cell surface CD4 binding to gp120 [Sullivan et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
510	CG-4	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	N	CD4/gp120 complex	murine(IgG_1)

Donor: Jonathan Gershoni, Tel Aviv University, Isreal
References: [Gershoni et al.(1993)]
NOTES:
- CG-4: Reacts with gp120 and sCD4-gp120 complex, not with sCD4 [Gershoni et al.(1993)]
- CG-4: Called CG4

511	CG-9	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	L	CD4/gp120 complex	murine(IgG_1)

References: [Gershoni et al.(1993)]
NOTES:
- CG-9: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 [Gershoni et al.(1993)]

512	CG-25	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	L	CD4/gp120 complex	murine(IgG_1)

References: [Gershoni et al.(1993)]
NOTES:
- CG-25: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 [Gershoni et al.(1993)]

513	CG-76	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	L	CD4/gp120 complex	murine(IgG_1)

References: [Gershoni et al.(1993)]
NOTES:
- CG-76: Reacts equally well with sCD4-gp120 and sCD4, but not with purified gp120 [Gershoni et al.(1993)]

514	ID6	gp120(1-193 BH10)	gp120	UNDEFINED AMINO TERMINUS	L	?	murine(IgG_1)

References: [Ugen et al.(1993), Cook et al.(1994)]
NOTES:
- ID6: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]
- ID6: NIH AIDS Research and Reference Reagent Program: 2343

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
515	AD3	gp120(1-193 BH10)	gp120	UNDEFINED AMINO TERMINUS	L	?	murine(IgG_1)

References: [Ugen et al.(1993), Cook et al.(1994)]
NOTES:
- AD3: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]
- AD3: NIH AIDS Research and Reference Reagent Program: 2342

516	522-149	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	Env glycopro	

Donor: G. Robey, Abbott Inc.
References: [Moore & Sodroski(1996), Trkola et al.(1996a), Binley et al.(1998)]
NOTES:
- 522-149: Binding is enhanced by C5 antibodies M91 and 1C1 – mutual binding-inhibition with anti-C1 antibody 133/290 – binding is destroyed by a W/L (position 61, LAI) gp120 amino acid substitution – other C1 antibodies enhance binding to gp120 [Moore & Sodroski(1996)]
- 522-149: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 522-149: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

517	MAG 45	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994), Moore & Sodroski(1996), Wyatt et al.(1997)]
NOTES:
- MAG 45: Also called #45
- MAG 45: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)]
- MAG 45: Reciprocal binding inhibition with anti-C1-C5 and anti-C1-C4 discontinuous MAbs – binding enhanced by anti-V3 5G11 – inhibits binding of anti-CD4 binding site MAbs [Moore & Sodroski(1996)]
- MAG 45: Called #45 – binds to efficiently sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-50, are deleted [Wyatt et al.(1997)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
518	MAG 95	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994)]
NOTES:
- MAG 95: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
519	MAG 97	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994)]
NOTES:
- MAG 97: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
520	MAG 104	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994)]
NOTES:
- MAG 104: Only observed amino acid substitution that reduces binding: 88 N/P and 106 E/A – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
521	M90	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	451 Env	(IgG_1)

Donor: Fulvia di Marzo Veronese

References: [di Marzo Veronese et al.(1992), Devico et al.(1995), Moore & Sodroski(1996), Ditzel et al.(1997), Wyatt et al.(1997), Binley et al.(1998)]

NOTES:

- M90: Reactive only with native gp120, so binds to a discontinuous epitope – reacts with multiple strains [di Marzo Veronese et al.(1992)]
- M90: Reacted with both non-reduced (but not denatured) covalently cross-linked gp120-CD4 complex [Devico et al.(1995)]
- M90: Reciprocal inhibition of binding of other anti-C1 MAbs – inhibits CD4 binding site MAbs – enhances binding of V2 MAbs G3-4 and SC258 [Moore & Sodroski(1996)]
- M90: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-82, are deleted [Wyatt et al.(1997)]
- M90: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

522	p7	gp120(C1 dis HXBc2)	gp120(dis)	DISCONTINUOUS		HIV infection	human Fab(IgG_1)

Donor: Fulvia di Marzo Veronese

References: [Ditzel et al.(1997), Parren et al.(1997b)]

NOTES:

- p7: gp120 immobilized on solid phase by capture with sCD4 was used for selection of Fabs – three novel N-term Fabs were obtained that bind to similar epitopes, p7, p20, and p35 – a C1 W/S substitution at position 45 abolished binding, a Y/D at position 45 reduced binding, and C5 region substitutions 475 M/S and 493 P/K enhanced binding – compete with MAbs M85, M90 and 212A, but not M91 and G3-299 [Ditzel et al.(1997)]
- p7: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)]

523	L19	gp120(C1 dis HXBc2)	gp120(dis)	DISCONTINUOUS		HIV infection	human Fab (IgG_1)

Donor: Fulvia di Marzo Veronese

References: [Ditzel et al.(1997)]

NOTES:

- L19: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for the selection of Fabs – six N-term Fabs, L19 L34, L35, L52, L59, and L69, were obtained that have a similar epitope to Fab p7 [Ditzel et al.(1997)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
524	L100	gp120(C1-C2 dis HXBc2)	gp120(dis)	DISCONTINUOUS		HIV infection	human Fab(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [Ditzel et al.(1997), Parren et al.(1997b), Parren & Burton(1997)]
NOTES:
- L100: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)]
- L100: gp120 immobilized on solid phase by capture with sCD4 and then masked with Fab p7 allowed selection of a new Fab, L100, with a novel specificity for C1 and C2 – gp120 C1 substitutions 69 W/L and 76 P/Y abolish L100 binding, and C2 substitutions 252 R/W, 256 S/Y, 262 N/T and 267 E/L abolish or strongly inhibit L100 binding – inhibits binding of MAbs M90 and G3-299, but not M85, 212A, and M91 [Ditzel et al.(1997), Parren & Burton(1997)]

525	L17	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS			human Fab

Donor: Fulvia di Marzo Veronese
References: [Ditzel et al.(1997), Parren et al.(1998)]
NOTES:
- L17: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

526	684-238	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L	IIIB gp120 from infected cells	murine

Donor: Gerry Robey, Abbott Laboratories
References: [Moore et al.(1993a), Thali et al.(1993), Gorny et al.(1994), Ditzel et al.(1995), Moore & Sodroski(1996), Ditzel et al.(1997)]
NOTES:
- 684-238: Also called 52-684-238 and 52-684
- 684-238: Specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – neutralizes BH10 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177FY/AT, 179/180LD/DL, 183/184PI/SG, and 192-194YSL/GSS [Moore et al.(1993a)]
- 684-238: Weakly neutralizing, IC_{50} = 84 μg/ml [Gorny et al.(1994)]
- 684-238: Does not compete with IgG1b12, reciprocal inhibition with MAbs L39, L40, and L78 [Ditzel et al.(1995)]
- 684-238: Limited reciprocal enhancement of binding with anti-V3 and C4 region antibodies – reciprocal inhibition with V2 region antibodies [Moore & Sodroski(1996)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
527	CRA-3	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	N	rBH10 gp120	murine(IgG_{2a})

Donor: Mark Page, NIBSC AIDS reagent project, Potters Bar, Herts, UK

References: [Moore & Ho(1993), Moore et al.(1993a), Thali et al.(1993), Shotton et al.(1995), Moore & Sodroski(1996), Ditzel et al.(1997)]

NOTES:

- CRA-3: Conformational, does not bind well to denatured gp120 [Moore & Ho(1993)]
- CRA-3: specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS – epitope probably involves stem of V1/V2 loop structure [Moore et al.(1993a)]
- CRA-3: Many MAbs enhance binding, including some anti-C5, C1, V4, and C4 MAbs – enhances binding of only a small number of anti-V3 loop MAbs [Moore & Sodroski(1996)]
- CRA-3: Called CRA3 – Same competition group as CRA6 [Shotton et al.(1995)]
- CRA-3: UK Medical Research Council AIDS reagent: ARP324

528	CRA-6	gp120(V1V2 dis)	gp120(dis)	DISCONTINUOUS	N	?	murine

Donor: Mark Page, NIBSC AIDS reagent project, Potters Bar, Herts, UK

References: [Shotton et al.(1995)]

NOTES:

- CRA-6: Called CRA6 – same competition group as CRA-3 [Shotton et al.(1995)]

529	CRA-4	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L (HXB2)	rBH10 gp120	murine(IgG_1)

Donor: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325

References: [McKeating et al.(1993b), Moore & Ho(1993), Moore et al.(1993a), Thali et al.(1993), Shotton et al.(1995), Moore & Sodroski(1996)]

NOTES:

- CRA-4: Also called CRA4
- CRA-4: Changes at residues 191/192/193 (YSL/GSS) within V2, 435 (Y/H) in C4, abrogate binding – type-specific neutralization [McKeating et al.(1993b)]
- CRA-4: Conformational, does not bind well to denatured gp120 [Moore & Ho(1993)]
- CRA-4: Specific for BH10 and HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)]
- CRA-4: Cross-competes with MAbs 11/68b, 62c, 66c, 66a – similar to 66c and 66a – non-reciprocal inhibition by MAbs 12b, 60b and CRA-6 [Shotton et al.(1995)]
- CRA-4: The only MAbs that enhanced binding were anti-V3 MAb 5G11 and anti-C1 MAb 135/9 binding – reciprocal inhibition of anti-V2 MAbs [Moore & Sodroski(1996)]
- CRA-4: UK Medical Research Council AIDS reagent: ARP325

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
530	66a	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L (HXB2)	rBH10 gp120	murine(IgG_1)

Donor: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325
References: [Shotton et al.(1995)]
NOTES:
- 66a: Substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184 PI/SG, and 191-193 YSL/GSS abrogate binding – same competition group as CRA4 [Shotton et al.(1995)]
- 66a: UK Medical Research Council AIDS reagent: ARP3074

531	66c	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L (HXB2)	rBH10 gp120	murine(IgG_1)

Donor: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325
References: [Shotton et al.(1995)]
NOTES:
- 66c: Substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184 PI/SG, and 191-193 YSL/GSS abrogate binding – same competition group as CRA4 [Shotton et al.(1995)]

532	11/68b	gp120(V1V2 dis)	gp120(dis)	DISCONTINUOUS	L (HXB2)	rBH10 gp120	rat(IgG_1)

Donor: Shotton and Dean
References: [McKeating et al.(1993b), Shotton et al.(1995)]
NOTES:
- 11/68b: Changes at residues 183/184 (PI/SG) within V2, 435 (Y/H) in C4, abrogate binding [McKeating et al.(1993b)]
- 11/68b: 435 (Y/H) in C4 does not abrogate binding (John Moore, per comm, 1996)
- 11/68b: Cross-competes with MAbs 62c, 66c, 66a, and CRA-4 – similar to MAb 62c – HXB2 neutralization escape mutant had a D/N substitution at residue 185 – non-reciprocal inhibition of binding of CRA-3 and CRA-6 [Shotton et al.(1995)]
- 11/68b: UK Medical Research Council AIDS reagent: ARP3041

533	62c	gp120(V1V2 dis)	gp120(dis)	DISCONTINUOUS	N	rBH10 gp120	rat(IgG_1)

Donor: Shotton and Dean
References: [Shotton et al.(1995)]
NOTES:
- 62c: Cross-competes with MAbs 11/68b, 66c, 66a, and CRA-4 – same cross-competition group as MAb 11/68b – non-reciprocal inhibition of binding of CRA-3 and CRA-6 – substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184 PI/SG, and 191-193 YSL/GSS abrogate binding – binds but does not neutralize Hx10 [Shotton et al.(1995)]
- 62c: UK Medical Research Council AIDS reagent: ARP3075

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
534	SC258	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L	IIIB gp120 from infected cells	murine

Donor: Gerry Robey, Abbott Laboratories
References: [Moore et al.(1993a), Thali et al.(1993), Gorny et al.(1994), Yoshiyama et al.(1994), Moore et al.(1994b), Ditzel et al.(1995), Moore & Sodroski(1996), Trkola et al.(1996a), Ditzel et al.(1997)]
NOTES:

- SC258: Also called 52-581-SC258 – binds to BH10, MN, and RF gp120 – neutralizes BH10 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)]
- SC258: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity – 177 Y/H inhibits SC258 neutralization [Yoshiyama et al.(1994)]
- SC258: Very poor reactivity with gp120 molecules outside of clade B [Moore et al.(1994b)]
- SC258: Does not compete with IgG1b12 – reciprocal inhibition with MAbs L39, L40, and L78 [Ditzel et al.(1995)]
- SC258: Several MAbs binding to various gp120 epitopes enhance binding, but the only MAb that SC258 enhanced binding of was anti-CD4 binding site MAb F91 – reciprocal inhibition with V2 region antibodies [Moore & Sodroski(1996)]
- SC258: Does not inhibit gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study – listed as not neutralizing [Trkola et al.(1996a)]

535	110-B	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	N	BRU infected cell lysates	murine

Donor: Hybridolabs, Institute Pasteur, Paris, France
References: [Moore et al.(1993a)]
NOTES:

- 110-B: specific for BH10, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 168 K/L, 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)]

536	L15	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	P (weak)	HIV infection	human(IgG_1)

Donor: Hybridolabs, Institute Pasteur, Paris, France
References: [Ditzel et al.(1997), Parren et al.(1997b)]
NOTES:

- L15: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – 2 anti-V2 Fabs were obtained with very similar epitopes, L15 and L17 – deletions in V1 and V2 abolished binding, and rodent anti-V2 MAbs SC258, CRA3, G3-G4,G3-136, BAT-085, and 52-684 all compete with L15 [Ditzel et al.(1997)]
- L15: Does not neutralize TCLA strains but neutralizes some primary isolates weakly [Parren et al.(1997b)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
537	L39	gp120(V2-CD4BS dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Hybridolabs, Institute Pasteur, Paris, France
References: [Ditzel et al.(1995)]
NOTES:

- L39: This Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L39 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684-238 – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]

538	L40	gp120(V2-CD4BS dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Hybridolabs, Institute Pasteur, Paris, France
References: [Ditzel et al.(1995)]
NOTES:

- L40: This Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L40 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding only partially affected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684-238 – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]

539	L78	gp120(V2-CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Hybridolabs, Institute Pasteur, Paris, France
References: [Ditzel et al.(1995)]
NOTES:

- L78: Substitutions at V2: (152/153 GE/SM, 183/184 PI/SG, 191/193 YL/GS), 262 N/T, V3 (314 G/W), CD4BS (257 T/R, 368 D/R, 370 E/R) inhibit binding, and some C4 and C5 substitutions enhance binding – this Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – Fab neutralizes MN and LAI – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684-238 – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
540	L25	gp120(V2-CD4BS dis)	gp120(dis)	DISCONTINUOUS	L (weak)	HIV-1 infection	human(IgG_1)

Donor: Hybridolabs, Institute Pasteur, Paris, France

References: [Ditzel et al.(1995), Ditzel et al.(1997), Parren et al.(1997b)]

NOTES:

- L25: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – a single anti-V2-CD4 BS Fab was obtained with with sensitivity to substitutions in the V2 and CD4 BS regions – rodent anti-V2 MAb SC258 competes with L25 [Ditzel et al.(1997)]
- L25: Neutralizes TCLA strains weakly, but not primary isolates [Parren et al.(1997b)]

541	C11	gp120(C1-C5 dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human

Donor: J. Robinson, Tulane University, LA

References: [Robinson et al.(1992), Moore et al.(1994d), Moore & Sodroski(1996), Trkola et al.(1996a), Wu et al.(1996), Binley et al.(1997), Fouts et al.(1997), Wyatt et al.(1997), Parren et al.(1997b), Sullivan et al.(1998)]

NOTES:

- C11: Also called c11
- C11: Mutations that inhibit binding: C1 (45 W/S, 88 N/P) – V5 (463 N/D) – and C5 (491 I/F,493 P/K and 495 G/K) and enhance binding: C1 (36 V/L) – V1-V2 (152/153 GE/SM) – andΔV1/V2/V3 [Moore et al.(1994d)]
- C11: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore & Sodroski(1996)]
- C11: Did not block ability of gp120-sCD4 complexes to inhibit MIP-1α binding – binds to gp41-binding domain [Wu et al.(1996)]
- C11: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- C11: A low avidity antibody as assessed by urea elution
- C11: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – C11 bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- C11: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – partial reexposure if sCD4 was bound – does not bind to HXBc2 gp120 if the 19 C-term amino acids are deleted [Wyatt et al.(1997)]
- C11: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)]
- C11: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 [Sullivan et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
542	212A	gp120(C1-C5 dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human

Donor: J. Robinson, Tulane University, LA

References: [Robinson et al.(1992), Moore et al.(1994d), Moore & Sodroski(1996), Binley et al.(1997), Fouts et al.(1997), Ditzel et al.(1997), Wyatt et al.(1997), Parren et al.(1997b), Sullivan et al.(1998), Binley et al.(1998)]

NOTES:

- 212A: Mutations that inhibit binding: C1 (45 W/S) and V5 (463 N/D) – and enhance binding: V2 (179/180 LD/DL) and C5 (495 G/K) [Moore et al.(1994d)]
- 212A: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore & Sodroski(1996)]
- 212A: A low avidity antibody as assessed by urea elution
- 212A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 212A bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- 212A: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids are deleted [Wyatt et al.(1997)]
- 212A: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)]
- 212A: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 [Sullivan et al.(1998)]
- 212A: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

543	L81	gp120(C1-C5 dis)	gp120(dis)	DISCONTINUOUS	N	HIV infection	human(IgG_1)

Donor: J. Robinson, Tulane University, LA

References: [Ditzel et al.(1997), Parren et al.(1997b)]

NOTES:

- L81: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – L81 binding is abolished by C1 substitution 45 W/S, C5 substitution 491 I/F, and C3 substitution L/A [Ditzel et al.(1997)]
- L81: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
544	2G12	gp120(C2-C3-V4 dis)	gp120(dis)	DISCONTINUOUS	L P	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Herman Katinger, Inst. Appl. Microbiol. or Polymun Scientific Inc., Vienna, Austria, MRC AIDS reagent project

References: [Buchacher et al.(1994), Trkola et al.(1995), Moore & Ho(1995), McKeating et al.(1996), McKeating(1996), Trkola et al.(1996b), Moore & Sodroski(1996), Poignard et al.(1996b), Trkola et al.(1996a), Sattentau(1996), D'Souza et al.(1997), Mo et al.(1997), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Moore & Trkola(1997), Mascola et al.(1997), Ugolini et al.(1997), Burton & Montefiori(1997), Parren et al.(1997b), Andrus et al.(1998), Wyatt et al.(1998), Mondor et al.(1998), Parren et al.(1998), Sullivan et al.(1998), Connor et al.(1998), Binley et al.(1998), Trkola et al.(1998), Fouts et al.(1998), Takefman et al.(1998), Li et al.(1998), Wyatt & Sodroski(1998)]

NOTES:

- 2G12: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells [Buchacher et al.(1994)]
- 2G12: Highly potent Cross-clade neutralizing activity [Trkola et al.(1995)]
- 2G12: Conformationally sensitive epitope destroyed by mutations altering the N-linked glycosylation sites near the base of the V3 loop and the amino-terminal flank of the V4 loop [Trkola et al.(1996b)]
- 2G12: Binding weakly enhanced by some anti-C1, -C4, -V3, and CD4 binding site MAbs – unusual in that 2G12 binding neither enhanced or inhibited the binding of other MAbs included in the study [Moore & Sodroski(1996)]
- 2G12: Review: binding site is distinct from CD4BS MAbs epitope and is unique among known gp120 MAbs, human or rodent [Moore & Ho(1995)]
- 2G12: Review: exceptional capacity to neutralize primary isolates in terms of both breadth and potency – one of three MAbs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard et al.(1996b)]
- 2G12: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 2G12: Neutralizes primary isolates, HXB2, and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]
- 2G12: Review: Only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]
- 2G12: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 μg per ml for 90% viral inhibition – neutralized 6 of 9 primary isolates [D'Souza et al.(1997)]
- 2G12: A JRCSF variant that was selected for IgG1b12 resistance remained sensitive to MAbs 2G12 and 2F5, for combination therapy [Mo et al.(1997)]
- 2G12: A low avidity antibody as assessed by urea elution
- 2G12: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 2G12 bound monomer, and weakly bound oligomer and neutralized JRFL [Fouts et al.(1997)]

544 cont.

- 2G12: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 2G12 was a strong neutralizer of SHIV-vpu+ – all Ab combinations tested showed synergistic neutralization – 2G12 has synergistic response with MAbs 694/98-D (anti-V3), 2F5, F105, and b12 [Li et al.(1997)]
- 2G12: Review: MABs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MAbs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs' epitopes [Moore & Trkola(1997)]
- 2G12: Using concentrations of Abs achievable *in vivo*, the triple combination of 2F5, 2G12 and HIVIG was found to be synergistic to have the greatest breadth and magnitude of response against 15 clade B primary isolates [Mascola et al.(1997)]
- 2G12: Viral binding inhibition by 2G12 was strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 2G12: Review that discusses this MAb – reacts with residues at the base of the V3 loop and V4, and most of the changes that reduce binding are glycosylation sites – it is not clear whether the binding site is peptidic or direct carbohydrate [Burton & Montefiori(1997)]
- 2G12: Neutralizes TCLA strains and primary isolates [Parren et al.(1997b)]
- 2G12: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIV-1 LAI in hu-PBL-SCID mice, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus et al.(1998)]
- 2G12: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- 2G12: Summary of the implications of the crystal structure of gp120 combined with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by 2G12 is unknown, but dependent on proper glycosylation and 2G12 is predicted to be oriented towards the target cell when bound, so neutralization may be due to steric hindrance – mutations in positions N 295, T 297, S 334, N 386, N 392 and N 397 HXBc2 (IIIB) decrease 2G12 binding, and the binding region is 25 angstroms from the CD4 binding site – probably the Ab binds in part to carbohydrates, which may account for both its broad reactivity and the scarcity of Abs in the same competition group [Wyatt et al.(1998)]
- 2G12: Enhances Hx10 binding to CD4 positive or negative HeLa cells, but inhibited binding to CD4+ T-cell line A3.01 – neutralizes Hx10 infection of the HeLa cells [Mondor et al.(1998)]
- 2G12: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D [Connor et al.(1998)]
- 2G12: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 [Sullivan et al.(1998)]

544 cont.

- 2G12: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – MAb 2G12 was the only exception to this, showing reduced binding efficiency [Binley et al.(1998)]
- 2G12: A wide range of neutralizing titers was observed that was independent of co-receptor usage [Trkola et al.(1998)]
- 2G12: Points out that 2G12 and 2F5, potent neutralizing antibodies, were identified by screening for cell surface (oligomeric envelope) reactivity [Fouts et al.(1998)]
- 2G12: Induces Complement-mediated lysis in MN but not primary isolates – primary isolates are refractive to CML [Takefman et al.(1998)]
- 2G12: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) [Li et al.(1998)]
- 2G12: Discussed in a review of the antigenic and receptor binding-domains of gp120 in relation to the structure of the molecule – antibodies are discussed by category (anti-V2, anti-V3, CD4i, CD4BS...), however as 2G12 binds to a rarely immunogenic region, and it is dependent on glycosylation, it was discussed individually [Wyatt & Sodroski(1998)]
- 2G12: UK Medical Research council AIDS reagent: ARP3030
- 2G12: NIH AIDS Research and Reference Reagent Program: 1476

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
545	SUMMARY CD4BS	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			

Donor: Herman Katinger, Inst. Appl. Microbiol. or Polymun Scientific Inc., Vienna, Austria, MRC AIDS reagent project
References: [Thali et al.(1993), Moore & Sodroski(1996)]
NOTES:
- Shared components of MAb epitopes and the discontinuous CD4 binding regions included Thr 257, Asp 368, Glu 370, Lys 421 through Trp 427 and Asp 457 [Thali et al.(1993)]
- Anti-CD4 binding site antibodies (CD4BS) competitively inhibit CD4 binding to monomeric gp120, and they differ in precise dependence on gp120 residues, but generally require Asp-368 and Glu-370 [Moore & Sodroski(1996)]

546 2G6 gp120(CD4BS dis) gp120(dis) DISCONTINUOUS
Donor: Herman Katinger, Inst. Appl. Microbiol. or Polymun Scientific Inc., Vienna, Austria
References: [Fouts et al.(1998)]
NOTES:
- 2G6: Binds to JRFL oligomer with an affinity comparable to IgG1b12, but does not neutralize the virus, so binding of oligomer is not always predictive of neutralization – conclusions of this paper contrast with [Parren et al.(1998)] – authors propose a model where 205-46-9 and 2G6 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts the neutralizing effect [Fouts et al.(1998)]

547 588-D gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L HIV-1 infection human(IgG$_{1\kappa}$)
Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY
References: [Karwowska et al.(1992a), Buchbinder et al.(1992), Moore & Ho(1993), Jeffs et al.(1996)]
NOTES:
- 588-D: Also called 588
- 588-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska et al.(1992a)]
- 588-D: 4-fold increase in neutralization potency for 588-D when combined 1:1 with human MAb 447-D [Buchbinder et al.(1992)]
- 588-D: Weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- 588-D: Called 588 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
548	10/46c	gp120(CD4BS dis)	?	?		rgp120	rat

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY
References: [Cordell et al.(1991), Jeffs et al.(1996)]
NOTES:
- 10/46c: Increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]

549	TH9	gp120(CD4BS)	?	?	L	?	human($IgG_{1\kappa}$)

Donor: Michael Fung, Tanox Biosystem, USA
References: [D'Souza et al.(1995), Yang et al.(1998)]
NOTES:
- TH9: Found to neutralize MN, but not JRCSF, two B subtype primary isolates, or a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs[D'Souza et al.(1995)]
- TH9: A neutralization assay was developed based on heminested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang et al.(1998)]

550	BM12	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human

Donor: Michael Fung, Tanox Biosystem, USA
References: [Kessler 2nd et al.(1995)]
NOTES:
- BM12: Broad cross-clade neutralization of primary isolates – additive effect in combination with MAb 2F5 [Kessler 2nd et al.(1995)]

551	654-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG_{κ})

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY
References: [Karwowska et al.(1993), Laal et al.(1994), Gorny et al.(1994), Stamatatos & Cheng-Mayer(1995), Li et al.(1997), Stamatatos et al.(1997), Gorny et al.(1998)]
NOTES:
- 654-D: Also called 654-30D and 654/30D
- 654-D: Dissociation constant gp120 IIIB 0.008 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D – reported to be human($IgG_{1\lambda}$) [Laal et al.(1994)]
- 654-D: Mild oxidation of carbohydrate moieties inhibits binding [Gorny et al.(1994)]
- 654-D: Binds to HIV-1 SF128A and SF162 [Stamatatos & Cheng-Mayer(1995)]
- 654-D: Called 654-30D – One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env [Li et al.(1997)]
- 654-D: Anti-CD4 BS MAb 654-30D and IgG1b12 have comparable binding affinities, neither mediates gp120-virion dissociation, but IgG1b12 can neutralize SF128A and SF162 and 654-D cannot – 654-D actually enhances infection by both viruses in primary macrophages [Stamatatos et al.(1997)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
552	S1-1	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY
References: [Lake et al.(1992), Moran et al.(1993), Wisnewski et al.(1996)]
NOTES:
- S1-1: Neutralizes IIIB and MN without complement, and neutralizes RF and a clinical isolate with complement – binds to native but not denatured gp120 – inhibits sCD4-gp120 binding [Lake et al.(1992)]
- S1-1: Heavy (V_HI) and light (V_λIII) chain sequenced – no enhancing activity – similar germline sequence to MAb 86, but very different activity [Moran et al.(1993)]
- S1-1: S1-1 is V_H1 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]

553	559/64-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY
References: [Karwowska et al.(1992a), McKeating et al.(1992), Spear et al.(1993), Forthal et al.(1995), Jeffs et al.(1996), Hioe et al.(1997)]
NOTES:
- 559/64-D: Also called 559
- 559/64-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska et al.(1992a)]
- 559/64-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear et al.(1993)]
- 559/64-D: Neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]
- 559/64-D: Called 559 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]
- 559/64-D: Used in the development of resting cell neutralization assay [Hioe et al.(1997)]

554	428	gp120(CD4BS dis)		?		HIV-1 infection	human

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY
References: [Karwowska et al.(1992a), Jeffs et al.(1996)]
NOTES:
- 428: Slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]

555	558-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY
References: [McKeating et al.(1992)]
NOTES:
- 558-D: Blocks gp120-CD4 binding – binds a panel of mutants all except for 256 S/Y and 262 N/T, which are probably conformationally disruptive [McKeating et al.(1992)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
556	448-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY

References: [Karwowska et al.(1992a), McKeating et al.(1992), Spear et al.(1993), Laal et al.(1994), Forthal et al.(1995), Manca et al.(1995), Li et al.(1997), Wyatt et al.(1998)]

NOTES:

- 448-D: Also called 448D
- 448-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska et al.(1992a)]
- 448-D: Called 448D – blocks gp120-CD4 binding – substitutions at gp120 residues 88, 113, 117, 257, 368 and 370 reduce binding – epitope similar to rat MAbs 39.13g and 39.3b [McKeating et al.(1992)]
- 448-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear et al.(1993)]
- 448-D: Dissociation constant gp120 IIIB 0.029 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D [Laal et al.(1994)]
- 448-D: Neutralizing activity, positive ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]
- 448-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca et al.(1995)]
- 448-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env [Li et al.(1997)]
- 448-D: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)]

557	729-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY

References: [Laal et al.(1994), D'Souza et al.(1997), Li et al.(1997), Parren et al.(1997b)]

NOTES:

- 729-D: Also called 729-30D
- 729-D: Dissociation constant gp120 IIIB 0.025 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D [Laal et al.(1994)]
- 729-D: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – reported here to have a λ light chain, but originally reported in [Laal et al.(1994)] to be $IgG_{1\kappa}$ [D'Souza et al.(1997)]
- 729-D: Called 720-30D – one of 14 human MAbs tested for ability to neutralize chimeric SHIV-vpu+, which expressed HIV-1 IIIB env [Li et al.(1997)]
- 729-D: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
558	HF1.7	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	purified anti-Leu-3a MAb	murine(IgM)

Donor: ?
References: [Chanh et al.(1987)]
NOTES:
- HF1.7: An anti-Id antibody, stimulated by anti-CD4 MAb Leu-3a, binds a recombinant gp160, suggesting HF1.7 mimics CD4 [Chanh et al.(1987)]

559	D20	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: ?
References: [Broder et al.(1994), Richardson Jr et al.(1996), Otteken et al.(1996), Earl et al.(1997)]
NOTES:
- D20: Binding completely blocked by pooled human sera [Broder et al.(1994)]
- D20: Human sera blocked binding in oligomeric ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 [Richardson Jr et al.(1996)]
- D20: Pulse label experiments of 4 MAbs (D20, D27, T20, and T22) binding to noncleavable gp160 revealed that these anti-CD4 MAbs bound with a delay, and that the epitope formed with a $t_{1/2}$ of about 10 minutes [Otteken et al.(1996)]
- D20: Used for comparison in a study of gp41 antibodies – D20 binds to a greater extent to cell surface expressed Env than any of 38 conformation dependent anti-gp41 MAbs [Earl et al.(1997)]

560	D60	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: ?
References: [Richardson Jr et al.(1996)]

561	50-61A	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG_κ)

Donor: ?
References: [Fevrier et al.(1995)]
NOTES:
- 50-61A: Neutralizes lab strains LAI and SF2 – competes with sera from 45 seropositive subjects – binding affinity 2.4×10^{-10} M [Fevrier et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
562	48-16	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(IgG_{κ})

Donor: ?
References: [Fevrier et al.(1995)]
NOTES:
- 48-16: Broadly cross-reactive, reacts outside the CD4 binding site and V3 region – competes with sera from 45 seropositive subjects – binding affinity $2 - 5 \times 10^{-9}$ M [Fevrier et al.(1995)]

563	L41	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: ?
References: [Ditzel et al.(1995)]
NOTES:
- L41: Substitutions at 133 D/R, 256 S/Y, 257 T/R, 368 D/R or D/T, 370 E/Q or E/R, 384 Y/E, and 421 K/L reduce binding – paradoxically, this Fab was retrieved from the library after masking with known anti-CD4BS MAbs – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]

564	L28	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: ?
References: [Ditzel et al.(1995)]
NOTES:
- L28: Substitutions at 257 T/R, 368 D/R, 370 E/R and 370 E/Q, 475 M/S 102 E/L and 463 N/D reduce binding – binding was enhanced by removal of the V3 loop and by substitutions 45 W/S, 298 R/G, 381 E/P, 382 F/L, 420 I/R, 435 Y/H or Y/R – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]

565	L33	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: ?
References: [Ditzel et al.(1995)]
NOTES:
- L33: binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]

566	L42	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: ?
References: [Ditzel et al.(1995)]
NOTES:
- L42: Substitutions at 257 T/R, 368 D/R, 370 E/R, 266 A/E and 477 D/V reduce binding – binding was significantly enhanced by 381 E/P and 382 F/L – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
567	L52	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: ?
References: [Ditzel et al.(1995)]
NOTES:
- L52: Binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
568	GP13	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG_1)

References: [Schutten et al.(1993), Back et al.(1993), Bagley et al.(1994), Schutten et al.(1995a), Schutten et al.(1995b), Bolmstedt et al.(1996), Wisnewski et al.(1996), Schutten et al.(1996), Schutten et al.(1997)]
NOTES:
- GP13: Neutralized a broad range of HIV-1 strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D), 384(Y/E) [Schutten et al.(1993)]
- GP13: Mutations in a neutralization resistant isolate obtained by passage of the IIIB isolate in chimpanzees reduced neutralization, but the escape was not as clear as seen with anti-V3 MAbs [Back et al.(1993)]
- GP13: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten et al.(1995a)]
- GP13: Neutralizes T-cell adapted viruses but not the SI strain 16.2, despite high binding affinity [Schutten et al.(1995b)]
- GP13: Sera was obtained from guinea pigs vaccinated either with gp160, or with gp160 lacking N-linked glycans at N406, N448, and N463 – these sera could block equally well both the CD4 BS MAb GP13 and the V3 MAb F58/H3 [Bolmstedt et al.(1996)]
- GP13: GP13 is V_H5 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- GP13: IIIB neutralizing MAbs *in vitro* fail to neutralize in a mouse model *in vivo* [Schutten et al.(1996)]
- GP13: Neutralized (50%) an SI-env chimeric virus and enhanced (>5 fold) an NSI-env chimeric virus [Schutten et al.(1997)]
- GP13: UK Medical Research council AIDS reagent: ARP3054

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
569	GP44	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG1)

References: [Schutten et al.(1993), Bagley et al.(1994), Wisnewski et al.(1996)]

NOTES:

- GP44: Exhibited a more restricted pattern of neutralizing activity than GP13 and GP68 – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D) [Schutten et al.(1993)]
- GP44: GP44 is V_H1 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]

570	L72	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			murine

Donor: Dr. Hariharam, IDEC Pharmaceuticals Corp La Jolla, CA

References: [Ditzel et al.(1997)]

NOTES:

- L72: Used to bind gp120 to solid phase to select MAbs from a phage selection library [Ditzel et al.(1997)]

571	GP68	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG1)

Donor: Dr. Hariharam, IDEC Pharmaceuticals Corp La Jolla, CA

References: [Schutten et al.(1993), Klasse et al.(1993a), Bagley et al.(1994), Schutten et al.(1995a)]

NOTES:

- GP68: Neutralized a broad range of HIV-1 lab strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 117(K/W), 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q), 384(Y/E), 435(Y/H) [Schutten et al.(1993)]
- GP68: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – GP68 required markedly higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)]
- GP68: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten et al.(1995a)]
- GP68: GP68 is V_H1 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- GP68: UK Medical Research Council AIDS reagent: ARP3055

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
572	ICR 39.13g	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	rgp120 BH10	rat(IgG_{2b})

Donor: Jackie Cordell and C. Dean

References: [Cordell et al.(1991), McKeating et al.(1992a), McKeating et al.(1992), McKeating et al.(1993b), Moore & Ho(1993), Thali et al.(1993), Klasse et al.(1993a), McLain & Dimmock(1994), Beretta & Dalgleish(1994), McKeating et al.(1996), Armstrong & Dimmock(1996), Klasse & Sattentau(1996)]

NOTES:

- ICR 39.13g: also known as ICR39.13g and 39.13g
- ICR 39.13g: Cross-competes with MAbs ICR 39.3b and 15e [Cordell et al.(1991)]
- ICR 39.13g: Binds to a conformational epitope involved in CD4 binding – exerts a synergistic effect in combination with V3 directed MAbs [McKeating et al.(1992a)]
- ICR 39.13g: Neutralization activity against HXB10, RF, SF-2 and MN strains of HIV-1 [McKeating et al.(1993b)]
- ICR 39.13g: Conformational, does not bind denatured gp120 – weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- ICR 39.13g: Strongly inhibits CD4 inducible MAb 48d [Thali et al.(1993)]
- ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)]
- ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)]
- ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]
- ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)]
- ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)]
- ICR 39.13g: UK Medical Research Council AIDS reagent: ARP390

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
573	ICR 39.3b	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	rgp120 BH10	rat(IgG_{2b})

Donor: J. Cordell and C. Dean

References: [Cordell et al.(1991), McKeating et al.(1992), Moore et al.(1993b), McLain & Dimmock(1994), Armstrong & Dimmock(1996), Jeffs et al.(1996), Wyatt et al.(1998)]

NOTES:

- ICR 39.3b: also known as 39.3, 39.3b and ICR39.3b
- ICR 39.3b: Cross-competes with MAbs ICR 39.13g and 15e [Cordell et al.(1991)]
- ICR 39.3b: Conformational, does not bind to denatured IIIB [Moore & Ho(1993)]
- ICR 39.3b: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively [McLain & Dimmock(1994)]
- ICR 39.3b: Neutralizes only if the antibody is added prior to the attachment of the virus to the cell, in contrast to 39.13g [Armstrong & Dimmock(1996)]
- ICR 39.3b: Called 39.3b – increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]
- ICR 39.3b: Called 39.3 – summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)]
- ICR 39.3b: UK Medical Research Council AIDS reagent: ARP391

574	15e	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: J. Robinson, Tulane University, LA, and David Ho, ADARC, NY, NY

References: [Robinson et al.(1990), Thali et al.(1991), Cordell et al.(1991), Ho et al.(1991b), Koup et al.(1991), Ho et al.(1992), Wyatt et al.(1992), Thali et al.(1992a), Takeda et al.(1992), Moore & Ho(1993), Thali et al.(1993), Wyatt et al.(1993), Bagley et al.(1994), Thali et al.(1994), Cook et al.(1994), Moore et al.(1994b), Moore et al.(1994a), Sattentau & Moore(1995), Lee et al.(1995), McKeating et al.(1996), Moore & Sodroski(1996), Poignard et al.(1996a), Trkola et al.(1996a), McDougal et al.(1996), Wisnewski et al.(1996), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Wyatt et al.(1997), Berman et al.(1997), Parren et al.(1997b), Wyatt et al.(1998), Parren et al.(1998), Sullivan et al.(1998), Binley et al.(1998), Trkola et al.(1998), Fouts et al.(1998)]

NOTES:

- 15e: Also called 1.5e, 1.5E and 15E – original paradigm for this type of antibody
- 15e: Broadly neutralizing, binds multiple strains, competes with CD4 for gp120 binding, DTT reduction of env abrogates binding – more potent blocking of gp120-sCD4 binding than MAbs G3-536 and G3-537 [Ho et al.(1991b)]
- 15e: Cross-competes with MAbs ICR 39.13g and ICR 39.3b [Cordell et al.(1991)]
- 15e: Binds to gp120 of HIV-1 IIIB, but not RF – mediates ADCC – deletion of the V3 loop from gp120 does not alter ADCC activity [Koup et al.(1991)]
- 15e: gp120 mutants that affect 15e epitope binding: 113, 257, 368, 370, 421, 427, 475 – four of these coincide with amino acids important for the CD4 binding domain [Ho et al.(1992)]

574 cont.

- 15e: Precipitation of Δ 297-329 env glycoprotein, with a deleted V3 loop, is much more efficient that precipitation of wild type [Wyatt et al.(1992)]
- 15e: Amino acid substitutions in HXB2 that strongly inhibit binding, similar to [Ho et al.(1992)], some additional, 88, 102, 117, 113, 257, 368, 370, 421, 427, 457, 470, 480 [Thali et al.(1992a)]
- 15e: Called N70-1.5e – does not enhance infection of HIV-1 IIIB and MN [Thali et al.(1992a)]
- 15e: Conformational, does not bind denatured gp120 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- 15e: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is greater than binding to wildtype gp120 [Wyatt et al.(1993)]
- 15e: Called 15E – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – 15E neutralization was not affected by this mutation [Watkins et al.(1993)]
- 15e: Heavy chain is V_HIV, V2-1 – light chain is V_κI, Hum01/012. Compared to 21h and F105 [Bagley et al.(1994)]
- 15e: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 21h and 17b) [Thali et al.(1994)]
- 15e: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – anti-CD4 MAbs moderately inhibit gp120 binding to GalCer, possibly through steric hindrance – binding of GalCer to gp120 inhibited but did not completely block 15e binding [Cook et al.(1994)]
- 15e: Cross-reactive with gp120 proteins from clades B and D, less so with A and C, and not reactive with clade E and F [Moore et al.(1994b)]
- 15e: Binds with higher affinity to monomer than to oligomer, moderate association rate [Sattentau & Moore(1995)]
- 15e: The V4 and V5 domains are essential for 1.5e binding, in contrast to the V1, V2, and V3 loops [Lee et al.(1995)]
- 15e: Called 1.5e – Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]
- 15e: gp120 binding enhanced by anti-V3 MAb 5G11 and anti-V2 MAb G3-136 – binding inhibited by other CD4 binding site MAbs, antibodies that bind to gp120 only when CD4 is bound, and CD4-IgG [Moore & Sodroski(1996)]
- 15e: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Poignard et al.(1996a)]
- 15e: Inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 15e: Neutralizes HIV-1 LAI less potently than V3 specific MAbs [McDougal et al.(1996)]
- 15e: 15e is V_H4 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- 15e: A low avidity antibody as assessed by urea elution
- 15e: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 15e bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]

574 cont.

- 15e: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 15e could only achieve 50% neutralization, but could act synergistically with anti-V3 MAb 694/98-D to achieve 90% [Li et al.(1997)]
- 15e: Does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-93, are deleted [Wyatt et al.(1997)]
- 15e: Called 1.5E – Binds to 7/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)]
- 15e: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- 15e: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)]
- 15e: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- 15e: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MAbs 15e, F91 and IgG1b12 bound better to the deleted protein than to wild type [Binley et al.(1998)]
- 15e: Competes with CG-10 binding, a MAb raised against a gp120 CD4 complex, this was probably due to the disruption of CD4-gp120 by 15e [Sullivan et al.(1998)]
- 15e: No detectable neutralizing activity among primary isolates with different co-receptor usage – some neutralization of TCLA strains [Trkola et al.(1998)]
- 15e: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer [Fouts et al.(1998)]
- 15e: UK Medical Research Council AIDS reagent: ARP3016

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
575	1125H	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L (MN)	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Shermaine Tilley, Public Health Research Institute, USA

References: [Tilley et al.(1991b), Tilley et al.(1991a), Thali et al.(1992a), Wyatt et al.(1992), Pinter et al.(1993b), D'Souza et al.(1995), Warrier et al.(1996), Pincus et al.(1996), Wyatt et al.(1998), Alsmadi & Tilley(1998), Yang et al.(1998)]

NOTES:

- 1125H: Also called 1125h
- 1125H: Binding to gp120 inhibited by CD4 – epitope is destroyed by reduction, but not by removal of N-linked sugars – potent neutralization of MN, RF, SF-2 and IIIB – neutralization synergy with anti-V3 MAb 4117C [Tilley et al.(1991a)]
- 1125H: Amino acid substitutions in HXB2 that strongly inhibit binding: 88, 102, 117, 113, 257, 368, 370, 421, 427, 457, 470, 480 [Thali et al.(1992a)]
- 1125H: Binding to soluble gp120 enhanced by the presence of an anti-V3 HuMAb, 41148D [Pinter et al.(1993b)]
- 1125H: Precipitation of Δ 297-329 env glycoprotein, which has a deleted V3 loop, is much more efficient that precipitation of wild type [Wyatt et al.(1992)]
- 1125H: Neutralization was MN specific – failed to neutralize JRCSF, and 2 B subtype and 1 D subtype primary isolates in a multi-laboratory study involving 11 labs [D'Souza et al.(1995)]
- 1125H: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier et al.(1996)]
- 1125H: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]
- 1125H: Called 1125h – summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)]
- 1125H: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against all four strains [Alsmadi & Tilley(1998)]
- 1125H: A neutralization assay was developed based on heminested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
576	5145A	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG1)

Donor: Shermaine Tilley, Public Health Research Institute, USA

References: [Pinter et al.(1993a), Warrier et al.(1996), Pincus et al.(1996), Alsmadi & Tilley(1998)]

NOTES:

- 5145A: Potent and broadly cross-reactive neutralization of lab strains [Pinter et al.(1993a)]
- 5145A: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier et al.(1996)]
- 5145A: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]
- 5145A: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against all four strains [Alsmadi & Tilley(1998)]

577	21h	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG_1)

Donor: J. Robinson, Tulane University, LA

References: [Ho et al.(1991b), Thali et al.(1992a), Ho et al.(1992), Wyatt et al.(1993), Moore & Ho(1993), Moore et al.(1994b), Moore et al.(1994a), Bagley et al.(1994), Thali et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Wisnewski et al.(1996), McKeating et al.(1996), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Ugolini et al.(1997), Wyatt et al.(1997), Parren et al.(1997b), Wyatt et al.(1998), Parren et al.(1998), Fouts et al.(1998)]

NOTES:

- 21h: Also called 2.1H
- 21h: Amino acid substitutions in HXB2 that inhibit binding, some shared with CD4 binding inhibition, 88, 113, 257, 368, 370, 421, 470, 480 [Thali et al.(1992a)]
- 21h: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is greater than binding to wildtype gp120 [Wyatt et al.(1993)]
- 21h: Conformational, does not bind denatured gp120 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- 21h: Has strong cross-reactivity with gp120 monomers from most subtypes, A-F, with the least reactivity to clade E [Moore et al.(1994b)]
- 21h: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore et al.(1994a)]
- 21h: Heavy chain is V_HIII, VDP-35 – light chain is V_λIIIa, Hum318. Compared to 15e and F105 [Bagley et al.(1994)]
- 21h: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 15e and 17b) [Thali et al.(1994)]
- 21h: Binds with higher affinity to monomer than to oligomer, moderate association rate [Sattentau & Moore(1995)]
- 21h: Anti-CD4 binding site MAb – reciprocal inhibition by anti-C1, -C4 and other anti-CD4 binding site antibodies – enhanced by some anti-V2 MAbs and anti-V3 MAb 5G11 – enhances binding of some anti-V3 and -V2 MAbs [Moore & Sodroski(1996)]

577 cont.

- 21h: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Poignard et al.(1996a)]
- 21h: 21h is V_H3 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- 21h: Called 2.1H – Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]
- 21h: A low avidity antibody as assessed by urea elution
- 21h: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 21h bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- 21h: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 67 μg/ml [Li et al.(1997)]
- 21h: Viral binding inhibition by 21h strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 21h: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding – major deletions in C1 and C5 and deletions of the V1V2 and V3 loops do not diminish binding [Wyatt et al.(1997)]
- 21h: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- 21h: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)]
- 21h: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- 21h: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer – conclusions of this paper contrast with [Parren et al.(1998)] [Fouts et al.(1998)]
- 21h: UK Medical Research Council AIDS reagent: ARP3017

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
578	F105	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: Marshall Posner, Boston MA

References: [Posner et al.(1991), Thali et al.(1991), Thali et al.(1992a), Marasco et al.(1992), Wyatt et al.(1992), Posner et al.(1992b), Posner et al.(1992a), Moore & Ho(1993), Posner et al.(1993), Cavacini et al.(1993a), Cavacini et al.(1993b), Wyatt et al.(1993), Montefiori et al.(1993), Potts et al.(1993), Klasse et al.(1993a), Pincus et al.(1993), Watkins et al.(1993), Bagley et al.(1994), Thali et al.(1994), Cook et al.(1994), Cavacini et al.(1994b), Cavacini et al.(1994a), Earl et al.(1994), Chen et al.(1994a), Turbica et al.(1995), Posner et al.(1995), Cavacini et al.(1995), Sullivan et al.(1995), Khouri et al.(1995), Jagodzinski et al.(1996), Wolfe et al.(1996), McDougal et al.(1996), Wisnewski et al.(1996), Pincus et al.(1996), Litwin et al.(1996), Chen et al.(1996), Parren et al.(1997b), D'Souza et al.(1997), Li et al.(1997), Cao et al.(1997), Wyatt et al.(1997), Wyatt et al.(1998), Cavacini et al.(1998), Li et al.(1998)]

NOTES:

- F105: First description of F105, binds topographically near the CD4-binding site – inhibits binding of free, infectious virions to uninfected HT-H9 cells, but does not react with virus adsorbed to uninfected HT-H9 cells – soluble rCD4 pre-bound to infected cells inhibits F105 binding – F105 inhibits infection of HT-H9 cells in standard neutralization assays with HIV-1 and MN strains [Posner et al.(1991)]
- F105: Neutralization escape mutants result from changes in amino acids in four discontinuous regions: C2, 256-262; C3, 386,370; C4, 421
- F105: Amino acid substitutions that impair F105 neutralization inhibit gp120-CD4 interaction [Thali et al.(1992a)]
- F105: MAb cDNA sequence – V_H4 V71-4 rearranged with a D_H D-D fusion product of dlr4 and da4, and with J_{H5} – V_κ is from the *Humvk325* germline gene joined with Jκ2 [Marasco et al.(1992)]
- F105: Precipitation of Δ 297-329 env glycoprotein, with has a deleted V3 loop, is much more efficient than precipitation of wild type [Wyatt et al.(1992)]
- F105: F105 mediates ADCC against SF2 through the CD16+ population of PBMC – does not mediate complement-dependent cytotoxicity [Posner et al.(1992b)]
- F105: Significant enhancement of F105 binding to RF infected cells preincubated with V3-specific MAbs V3-2 and V3-1 [Posner et al.(1992a)]
- F105: Called F-105 – neutralizes IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- F105: F105 binds to and neutralizes selected lab strains and 3/9 HIV-1 primary isolates – synergistic enhancement of neutralization by seropositive sera [Posner et al.(1993)]
- F105: No neutralization of primary isolates observed (John Moore, pers comm)
- F105: Additive MN or SF2 neutralization when combined with anti-V3 MAbs 447-52D and 257-D [Cavacini et al.(1993a)]
- F105: Serum from all asymptomatic HIV-1 positive people tested block F105 binding, but only from 27% of symptomatic individuals [Cavacini et al.(1993b)]
- F105: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is 2.4- and 13-fold greater, respectively, than binding to wildtype gp120 [Wyatt et al.(1993)]
- F105: Study of synergism between F105 and sera from vaccinated volunteers with V3-loop specific neutralization activity – 2/3 sera demonstrated neutralization synergy, and 3/3 binding/fusion-inhibition synergy [Montefiori et al.(1993)]

578 cont.

- F105: Study of synergism of neutralization and binding comparing F105 and sCD4 with the V3 MAbs: 50.1, 59.1, 83.1, and 58.2 – synergy was observed, and the data suggest that binding of one ligand (F105) can increase the binding of the second (*e. g.* V3 loop MAbs) due to conformational changes [Potts et al.(1993)]
- F105: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – required > 81 fold higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)]
- F105: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – F105 was used as a control – infected lab workers and some of the gp160 vaccinees had a MAb response that could inhibit gp120-CD4 binding, at lower titers than the infected lab workers [Pincus et al.(1993)]
- F105: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – F105 neutralization was not affected by this mutation [Watkins et al.(1993)]
- F105: Comparison of MAb F105 sequences with those of MAbs 21h and 15e [Bagley et al.(1994)]
- F105: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MABs 48d, 21h, 15e and 17b) [Thali et al.(1994)]
- F105: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – anti-CD4 MAbs moderately inhibit gp120 binding to GalCer, possibly through steric hindrance – binding of GalCer to gp120 inhibited but did not completely block F105 binding[Cook et al.(1994)]
- F105: Administered intravenously to four cynomologus monkeys, plasma pharmacokinetics and biological activity tested [Cavacini et al.(1994b)]
- F105: Fab fragments show reduced capacity to neutralize IIIB, MN, and RF compared to intact IgG_1, suggesting bivalent interaction may be important in binding and neutralization [Cavacini et al.(1994a)]
- F105: Used as a positive control for CD4 BS antibodies in a study of the influence of oligomeric structure of Env in determining the repertoire of the Ab response [Earl et al.(1994)]
- F105: A human CD4+ T lymphocyte line was transduced to express Fab fragments of F105 – heavy and light chains are joined by an inter-chain linker – in the transduced cells infected with HIV-1, the Fab binds intracellularly to the envelope protein and inhibits HIV-1 production – secreted Fab fragments neutralize cell-free HIV-1 – combined intra- and extracellular binding activities of the expressed Fab make transduced cells resistant to HIV-1 infection and also can protect surrounding lymphocytes by secreting neutralizing antibodies [Marasco et al.(1993), Chen et al.(1994a)]
- F105: An immunoassay for titrating CD4BS serum antibody was developed using a gp120-coated solid phase and competition with MAb F105 – 109/110 French HIV-1+ sera and 51/56 HIV-1+ African sera had detectable CD4BS Abs using this assay, demonstrating CD4 binding site conservation among diverse subtypes – CD4BS Abs were detected soon after seroconversion and persisted – 0/21 HIV-2+ sera reacted, indicating that the HIV-1 and HIV-2 CD4BS Abs are not cross-reactive [Turbica et al.(1995)]
- F105: Eight patient phase Ia trial for use as an immunotherapeutic – no clinical or biochemical side effects observed, plasma levels $\geq$ of 10 μg/ml maintained for 21 days [Posner et al.(1995)]
- F105: Efficient neutralization of T-cell adapted lines HXBc2 and MN, no neutralization of primary isolates 89.6, ADA and YU2 – even some enhancement of infection of ADA and YU2 [Sullivan et al.(1995)]
- F105: Biotinylated F105 was used for competition studies with Ab derived from pregnant HIV-1+ women – a correlation between maternal anti-CD4 BS Abs overlapping the F105 binding site and lack of HIV-1 transmission to infants was noted[Khouri et al.(1995)]

578 cont.

- F105: Changing heavy chain from IgG_1 to IgG_3 increased neutralization efficiency [Cavacini et al.(1995)]
- F105: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop results in less potent inhibition of F105 binding by CRDS – binding site of F105 described as 256-257 ST, 368-370 DPE, 421 K, and 470-484 PGGGDMRDNWRSELY [Jagodzinski et al.(1996)]
- F105: Phase I study – MAb clearance in plasma has a 13 day half-life [Wolfe et al.(1996)]
- F105: Neutralizes HIV-1 LAI less potently than V3 specific MAbs [McDougal et al.(1996)]
- F105: F105 is V_H4 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- F105: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]
- F105: Binding of F105 to oligomeric gp120 occurs despite the fact it cannot neutralize primary isolates [Litwin et al.(1996)]
- F105: Intracellular co-expression of heavy and light chains of the Fab105 fragment MAb F105 was enhanced by inclusion of an internal ribosome entry site (IRES) sequence – the Fab105 IRES expression cassette was cloned into an adeno-associated virus (AAV) shuttle vector, and transduced into human lymphocytes which were able to produce and secrete the Fab105 fragments while maintaining normal growth – several primary HIV-1 patient isolates were effectively blocked [Chen et al.(1996)]
- F105: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- F105: In a multilaboratory blinded study, failed to neutralize any of nine B clade primary isolates [D'Souza et al.(1997)]
- F105: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – F105 could only achieve 50% neutralization alone – all Ab combinations tested showed synergistic neutralization – F105 has synergistic response with MAbs 694/98-D (anti-V3), 48d, 2F5, and 2G12, and also with HIVIG [Li et al.(1997)]
- F105: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to a CD4BS MAb F105 or sCD4 [Cao et al.(1997)]
- F105: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-93, are deleted [Wyatt et al.(1997)]
- F105: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)]
- F105: Phase I dose escalation study, single dose of 100 or 500 mg/m^2 was given to 4 HIV+ patients – sustained levels, no immune response against F105, no toxicity, infused Ab retained function – there was no evidence of anti-HIV-1 activity and virus was not diminished at day 1 or 7, by culture or plasma RNA [Cavacini et al.(1998)]
- F105: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) [Li et al.(1998)]
- F105: NIH AIDS Research and Reference Reagent Program: 857

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
579	IgG1b12	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L P	HIV-1 infection	human($IgG_1\kappa$)

Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA

References: [Burton et al.(1991), Barbas III et al.(1992), Roben et al.(1994), Burton et al.(1994), Moore et al.(1994b), Sattentau(1995), Moore et al.(1995a), Moore & Ho(1995), Parren et al.(1995), Trkola et al.(1995), Ditzel et al.(1995), Sullivan et al.(1995), Yang et al.(1997), Moore & Sodroski(1996), Gauduin et al.(1996), Poignard et al.(1996b), Poignard et al.(1996a), Trkola et al.(1996a), Sattentau(1996), McKeating(1996), D'Souza et al.(1997), Schutten et al.(1997), Mo et al.(1997), Fouts et al.(1997), Li et al.(1997), Kessler II et al.(1997), Moore & Trkola(1997), Stamatatos et al.(1997), Valenzuela et al.(1998), Ditzel et al.(1997), Ugolini et al.(1997), Wyatt et al.(1997), Wyatt et al.(1998), Burton & Montefiori(1997), Boots et al.(1997), Parren et al.(1997b), Parren et al.(1997a), Parren & Burton(1997), Mondor et al.(1998), Parren et al.(1998), Connor et al.(1998), Binley et al.(1998), Fouts et al.(1998), Takefman et al.(1998)]

NOTES:

- IgG1b12: Fab b12, Fab 3B3 and MAb IgG1b12 (Also called IgG1-b12, IgG1 b12, IgGB12, and b4/12and b12) – Fab 3B3 was derived from Fab b12 by random mutagenesis and selected for increased affinity to sgp120
- IgG1b12: The original Fab fragment was derived from a combinatorial phage library from bone marrow of an HIV-1 positive individual [Burton et al.(1991)]
- IgG1b12: Anti-CD4 binding site Fab, potent neutralizing activity, greater affinity for a subpopulation of gp120 molecules suggested to be in a mature confirmation – mutations in gp120 that abrogate binding: 368 D/R or D/T, 370 E/R, and 477 D/V, of clone HXBc2 of LAI – sensitive to V1 and V2 substitutions [Roben et al.(1994)]
- IgG1b12: Very potent neutralization, of primary and lab strains, at concentrations that could be achieved by passive immunization – reduced binding with A,C, and D clade viruses relative to B clade, poor reactivity with E clade – isolates that were refractive to neutralization by sera from HIV-1+ donors could be neutralized by IgG1 b12 [Burton et al.(1994)]
- IgG1b12: Cross-reactive with some gp120s, (but not all), from clades A-D – not reactive with gp120 from clades E or F [Moore et al.(1994b)]
- IgG1b12: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau et al.(1995)]
- IgG1b12: Anti-CD4 binding site MAb – very potent neutralization of a number of primary isolates [Moore et al.(1995a)]
- IgG1b12: Complete protection against HIV-1 infection was achieved in hu-PBL-SCID mice by passive immunization with physiologically relevant doses – pharmacokinetics showed serum half-life of 30.2 $+/-$ 1.3 hours for Fab b12 and 7.4 $+/-$ 0.7 days for IgG1 b12 in mice, but IgG1 half-lives in human are generally between 21-23 days [Parren et al.(1995), Parren & Burton(1997)]
- IgG1b12: Called BM12 – broad cross-clade neutralization of primary isolates – additive neutralization in combination with MAb 2F5 [Kessler 2nd et al.(1995)]
- IgG1b12: Review: unusual properties for anti-CD4 BS MAb: sensitive to V2 substitutions, preferential recognition of the oligomer on the cell surface [Moore & Ho(1995)]

579 cont.

- IgG1b12: Could potently neutralize primary isolates from within clade B, but showed a slight reduction in efficacy outside of clade B [Trkola et al.(1995)]
- IgG1b12: Because of Fab b12's reduction in binding when the V2 loop is deleted and when aa 183/184 PI/SG substitutions are made [Roben et al.(1994)], competition studies were done with Fab L78 anti-V2 MAbs SC258 and 684-238
- IgG1b12: Fab b12 showed potent neutralization of T-cell-line-adapted strains, but much reduced neutralization of 3 primary isolates – 2 of the 3 primary isolates also had reduced binding affinity, but the third was as efficiently immunoprecipitated as HXBc2 [Sullivan et al.(1995)]
- IgG1b12: Saturation mutagenesis of the complementarity-determining region and optimization strategies were used to create very high affinity versions of this Fab – increased affinity was dominated by a slowing of the off rate [Yang et al.(1997)]
- IgG1b12: Potent neutralizing *ex vivo* of virus taken directly from plasma of HIV-1 infected individuals – little correlation between neutralization sensitivity of passaged virus and plasma derived virus – more effective than MAb 19b [Gauduin et al.(1996)]
- IgG1b12: Review: Unique among anti-CD4BS MAbs in terms of being potent against both lab adapted virus and primary isolates – one of three MAbs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard et al.(1996b)]
- IgG1b12: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Poignard et al.(1996a)]
- IgG1b12: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- IgG1b12: Review: Only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]
- IgG1b12: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 μg per ml for 90% viral inhibition – IgG1b12 failed to neutralize only 1/9 primary isolates, although there was some variation between test sites [D'Souza et al.(1997)]
- IgG1b12: Inhibited some SI- and NSI-env chimeric viruses but enhanced one NSI-env chimeric virus 3 fold [Schutten et al.(1997)]
- IgG1b12: JRCSF was cultured in the presence of IgG1b12 until a 100-fold resistance to neutralization was selected – resistance was due to three changes: V2 substitution D182N and C3 substitution P365L conferred resistance, and V2 D164N was also required for a viable virus – IgG1b12 resistant virus remained sensitive to MAbs 2G12 and 2F5 [Mo et al.(1997)]
- IgG1b12: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – IgG1b12 bound monomer, oligomer, and neutralized JRFL [Fouts et al.(1997)]
- IgG1b12: b12 was used in its IgG_1 form – of 14 human MAbs, the most potent neutralizer of SHIV-vpu+, which expressed HIV-1 IIIB env – all Ab combinations tested showed synergistic neutralization – b12 has a synergistic response with MAbs 694/98-D (anti-V3), 2F5, and 2G12 [Li et al.(1997)]

579 cont.

- IgG1b12: 35 primary isolates were tested and all were neutralized by IgG1b12 (including 4, UG270, RW92/026, ZB20, and 301727 which been had reported as not neutralized by IgG1b12 [Trkola et al.(1995)]) – IgG1b12 could neutralize even when added after the virus to the culture – selection for 400-fold increased affinity did not enhance neutralization by antibody – IgG1b12 was more potent with greater breadth than MAb 2F5 [Kessler II et al.(1997)]
- IgG1b12: Review: MABs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MAbs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs' epitopes [Moore & Trkola(1997)]
- IgG1b12: MAb was slightly more efficient at neutralization than Fab – inhibits viral binding to cells and viral entry – doesn't effect CD4-independent binding to T-cells [Valenzuela et al.(1998)]
- IgG1b12: Viral binding inhibition by IgG1b12 strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- IgG1b12: Major deletions in C1 and C5 and deletions of the V1V2 and V3 loops do not diminish binding [Wyatt et al.(1997)]
- IgG1b12: This is a review that includes a description of IgG1b12, noting approximately equivalent affinities for sgp120 and unprocessed gp160, and somewhat enhanced affinity for the native oligomer on TCLA viruses – primary viruses have reduced affinity, but still in the useful range for neutralization – there can be complete protection in hu-PBL-SCID mice with Ab even when administered several hours after viral challenge – competes with sCD4, but unlike other CD4BS antibodies, it is sensitive to mutations in V2 [Burton & Montefiori(1997)]
- IgG1b12: In this review, the technique and potential application of Fab expression and selection in phage display libraries, and subsequent production of IgG molecules is discussed – b12 is exceptionalty potent at neutralization and can successfully neutralize most B clade primary isolates, and many isolates from other subtypes as well – 3B3 was derived from b12 by selection for higher affinity using the CDR walking strategy – 3B3 has 8-fold enhancement of binding, a linear correlation was found between neutralization and affinity, and 3B3 can neutralize strains b12 cannot [Parren & Burton(1997)]
- IgG1b12: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – IgG1b12 blocks CD4 binding and is the most potent neutralizing Ab – many 15 and 21-mer phage inserts were recognized, but it was not possible to derive a consensus – common features were a W and at least one acidic residue, and one sequence was found multiple times: NWPRWWEEFVDKHSS, and this peptide could compete with gp120 – two short stretches found in the phage peptides might mimic gp120 components of the epitope: positions 382-384, FFY(I), and 423-426 I(FV)I(V)NM [Boots et al.(1997)]
- IgG1b12: Fab b12 is unusual in that it binds to gp140 and monomeric gp120 with similar affinities, and with a higher affinity to the native oligomer – authors propose this antibody may be exceptional because it binds the virus rather than viral debris – IgG1b12 can protect against infection prior to or shortly after challenge of hu-PBL-SCID mice with TCLA strains and primary strains, but the serum concentrations required were higher than for in vitro neutralization [Parren et al.(1997b), Parren et al.(1997a)]

579 cont.

- IgG1b12: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding – IgG1b12 is an unusual CD4BS antibody because it is particularly potent as a neutralizing antibody and it is susceptible to changes in the V1-V2 stem loop structure, and so it may disrupt an interaction between CD4 and conserved amino acids on the V1-V2 stem [Wyatt et al.(1998)]
- IgG1b12: Enhances binding of Hx10 to CD4 positive or negative HeLa cells, inhibits binding to CD4+ T-cell line A3.01 – neutralizes HeLa and A3.01 cell Hx10 infection [Mondor et al.(1998)]
- IgG1b12: IgG1b12, FAb b12 and 3B3 derived from b12 were all included in this study – the rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope – binding affinity of divalent IgG1b12 is 17-fold greater than monovalent Fab b12 [Parren et al.(1998)]
- IgG1b12: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D [Connor et al.(1998)]
- IgG1b12: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MAbs 15e, F91 and IgG1b12 bound better to the deleted protein than to wild type [Binley et al.(1998)]
- IgG1b12: Binds JRSF oligomer with high affinity, as do 205-46-9 and 2G6, but IgG1b12 is neutralizing, the other two are not – conclusions of this paper contrast with [Parren et al.(1998)] – authors propose a model where 205-46-9 and 2G6 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts the neutralizing effect – rank order of CD4BS antibodies oligomer binding is IgG1b12 = 2G6 = 205-46-9 > 205-43-1 = 205-42-15 > 15e = 21h = F91, and the only thing notably distinguishing about neutralizing IgG1b12 is that it depends on residues in V2 [Fouts et al.(1998)]
- IgG1b12: Induces Complement-mediated lysis in MN but not primary isolates – primary isolates are refractive to CML [Takefman et al.(1998)]
- IgG1b12: UK Medical Research Council AIDS reagent: ARP3065
- IgG1b12: NIH AIDS Research and Reference Reagent Program: 2640

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
580	DO8i	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			Fab, human

Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA

References: [Parren et al.(1998)]

NOTES:

- DO8i: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

581	DA48	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			Fab, human

Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA

References: [Parren et al.(1998)]

NOTES:

- DA48: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

582	b3	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			Fab, human

Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA

References: [Parren et al.(1997b), Parren et al.(1998)]

NOTES:

- b3: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- b3: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
583	b11	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			Fab, human

Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA

References: [Parren et al.(1998)]

NOTES:

- b11: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

584	b6	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L		Fab, human

Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA

References: [Parren et al.(1997b), Parren et al.(1998)]

NOTES:

- b6: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- b6: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

585	b13	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			Fab, human

Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA

References: [Parren et al.(1995), Parren et al.(1998)]

NOTES:

- b13: Fab b13 was used as a control in a hu-PBL SCID mouse study – animals were protected from HIV-1 SF2 infection by IgG1b12, somewhat by Fab b12, but not by b13 [Parren et al.(1995), Parren & Burton(1997)]
- b13: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
586	b14	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			Fab, human

Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA

References: [Parren et al.(1998)]

NOTES:

- b14: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

587 F91 gp120(CD4BS dis) gp120(dis) DISCONTINUOUS

Donor: J. Robinson, University of Connecticut, Storrs

References: [Moore & Ho(1993), Moore et al.(1994b), Moore & Sodroski(1996), Fouts et al.(1997), Mondor et al.(1998), Parren et al.(1998), Binley et al.(1998), Fouts et al.(1998)]

NOTES:

- F91: Called F-91 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- F91: Has strong cross-reactivity with gp120 monomers from most subtypes, A-F [Moore et al.(1994b)]
- F91: Unusual pattern of reciprocal enhancement with several anti-V2 and V3 directed MAbs – reciprocal inhibition of other CD4BS MAbs [Moore & Sodroski(1996)]
- F91: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – F91 bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- F91: Weak inhibition of binding of Hx10 to CD4 positive or negative cells, weakly neutralizing [Mondor et al.(1998)]
- F91: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- F91: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MAbs 15e, F91 and IgG1b12 bound better to the deleted protein than to wild type [Binley et al.(1998)]
- F91: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer – conclusions of this paper contrast with [Parren et al.(1998)] [Fouts et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
588	HT6	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L (weak)	HIV-1 infection	human

Donor: Ciba-Geigy AG Basel, Switzerland, and Tanox Biosystems, Houston, Texas

References: [Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997), Fouts et al.(1998)]

NOTES:

- HT6: HT5, HT6, and HT7 are also known as 205-43-1 , 205-42-15, and 205-46-9, respectively [Fouts et al.(1998)]
- HT6: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN [Moore et al.(1995a)]
- HT6: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was not quite as extensively cross-reactive [Moore et al.(1994b)]
- HT6: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts et al.(1997)]
- HT6: HT5 and HT6 bind JRSF oligomer but with low affinity, and are not neutralizing – conclusions of this paper contrast with [Parren et al.(1998)] [Fouts et al.(1998)]

589	HT5	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L (weak)	HIV-1 infection	human

Donor: Ciba-Geigy AG (Basel, Switzerland), and Tanox Biosystems, Houston, Texas

References: [Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997), Fouts et al.(1998)]

NOTES:

- HT5: HT5, HT6, and HT7 are also known as 205-43-1, 205-42-15, and 205-46-9, respectively [Fouts et al.(1998)]
- HT5: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN [Moore et al.(1995a)]
- HT5: 205-46-9 was cross-reactive across clades A-F, 205-43-1 very cross-reactive but not quite as extensive 205-46-9 [Moore et al.(1994b)]
- HT5: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts et al.(1997)]
- HT5: HT5 and HT6 bind JRSF oligomer but with low affinity, and are not neutralizing – conclusions of this paper contrast with [Parren et al.(1998)] [Fouts et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
590	HT7	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L (IIIB)	HIV-1 infection	human

Donor: Ciba-Geigy AG (Basel, Switzerland), and Tanox Biosystems, Houston, Texas
References: [Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997), Fouts et al.(1998)]
NOTES:

- HT7: HT5, HT6, and HT7 are also known as 205-43-1 , 205-42-15, and 205-46-9, respectively [Fouts et al.(1998)]
- HT7: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only neutralizes IIIB well, with sporadic weak neutralization of other isolates [Moore et al.(1995a)]
- HT7: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was cross-reactive, but not quite as extensive [Moore et al.(1994b)]
- HT7: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts et al.(1997)]
- HT7: Binds JRSF oligomer with high affinity, at least as high as IgG1b12, but IgG1b12 is neutralizing, H7 is not – conclusions of this paper contrast with [Parren et al.(1998)] – authors propose a model where H7 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts the neutralizing effect [Fouts et al.(1998)]

591	MAG 55	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994), Moore & Sodroski(1996)]
NOTES:

- MAG 55: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 470 P/L, 475 M/S, 477 D/V – neutralizes MN, IIIB and RF [Kang et al.(1994)]
- MAG 55: Called #55 – binding reciprocally inhibited by other anti-CD4 binding site MAbs, and by some C1-C5 MAbs – binding enhanced by anti-V3 MAb 110.5 and anti-V2 MAbs G3-136 and G3-4 – enhances binding of many anti-V3 and -V2 MAbs. [Moore & Sodroski(1996)]

592	MAG 72	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang or Dr. Hariharam, IDEC Pharmaceuticals Corp, La Jolla, CA
References: [Kang et al.(1994), Ditzel et al.(1997)]
NOTES:

- MAG 72: Also called L72
- MAG 72: Amino acid substitutions that reduce binding 10 fold: 257 T/R or A or G, 262 N/T, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 477 D/V – neutralizes MN, IIIB and RF [Kang et al.(1994)]
- MAG 72: Called L72 – used to bind gp120 to solid phase to select MAbs from a phage selection library [Ditzel et al.(1997)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
593	MAG 86	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994)]
NOTES:
- MAG 86: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 470 P/L, 477 D/V – neutralizes MN, IIIB and RF [Kang et al.(1994)]

594	MAG 96	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994)]
NOTES:
- MAG 96: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R – weak neutralization of IIIB [Kang et al.(1994)]

595	MAG 116	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994)]
NOTES:
- MAG 116: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L – neutralizes MN, IIIB and RF [Kang et al.(1994)]

596	MAG 3B	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994)]
NOTES:
- MAG 3B: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R or A or G, 262 N/T, 368 D/R or T, 370 E/R or Q, 381 E/P, 384 Y/E, 421 K/L, 475 M/S, 477 D/V [Kang et al.(1994)]

597	MAG 12B	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine

Donor: C. Y. Kang, IDEC Inc
References: [Kang et al.(1994)]
NOTES:
- MAG 12B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 477 D/V – weak neutralization of IIIB [Kang et al.(1994)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
598	830D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L		
		References: [Wyatt et al.(1998)]					
		NOTES:					
		• 830D: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)]					
599	MAG 29B	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine
		Donor: C. Y. Kang, IDEC Inc					
		References: [Kang et al.(1994)]					
		NOTES:					
		• MAG 29B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 386 N/Q, 421 K/L – weak neutralization of IIIB [Kang et al.(1994)]					
600	120-1B1	gp120(CD4BS dis)		DISCONTINUOUS	L		human
		Donor: Virus Testing Systems Corp., Houston, TX					
		References: [Watkins et al.(1993)]					
		NOTES:					
		• 120-1B1: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – 120-1B1 was not affected by this mutation [Watkins et al.(1993)]					
601	MAG 6B	gp120(dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine
		Donor: C. Y. Kang, IDEC Inc					
		References: [Kang et al.(1994)]					
		NOTES:					
		• MAG 6B: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R or G or A, 262 N/T, 368 D/R or T, 370 E/R or Q, 381 E/P, 384 Y/E, 421 K/L, 475 M/S, 477 D/V [Kang et al.(1994)]					
602	P43110	gp120(dis)	gp120(dis)	DISCONTINUOUS		unk	
		Donor: Advanced Biosciences (Kensington, MD)					
		References: [di Marzo Veronese et al.(1992), VanCott et al.(1995)]					
		NOTES:					
		• P43110: Does not recognized denatured form of the gp120 protein [VanCott et al.(1995)]					

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
603	17b	gp120(CD4i dis)	gp120(dis)	DISCONTINUOUS	L P (weak)	HIV-1 infection	human

Donor: Advanced Biosciences (Kensington, MD)

References: [Thali et al.(1993), Moore et al.(1993c), Thali et al.(1994), Beretta & Dalgleish(1994), Wyatt et al.(1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Wu et al.(1996), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Weinberg et al.(1997), Ditzel et al.(1997), Cao et al.(1997), Wyatt et al.(1997), Parren et al.(1997b), Kwong et al.(1998), Wyatt et al.(1998), Moore & Binley(1998), Rizzuto et al.(1998), Sullivan et al.(1998), Binley et al.(1998)]

NOTES:

- 17b: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs
- 17b: Epitope is better exposed upon CD4 binding to gp120 – competes with 15e and 21h, anti-CD4 binding site MAbs – 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 433A/L, 438 P/R and 475 M/S confer decreased sensitivity to neutralization [Thali et al.(1993)]
- 17b: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding of 17b [Moore et al.(1993c)]
- 17b: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 21h and 15e) [Thali et al.(1994)]
- 17b: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 17b in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 – similar effect observed for 48d and A32 [Wyatt et al.(1995)]
- 17b: Binds with higher affinity to monomer and oligomer, slow association rate, poor neutralization of lab strain – this is in contrast to 48d, which has very different kinetics [Sattentau & Moore(1995)]
- 17b: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) – anti-V3 MAb 5G11 enhances binding, as do C1-C4 discontinuous epitopes A32 and 2/11c – enhances binding of some anti-V2 MAbs [Moore & Sodroski(1996)]
- 17b: Binding did not result in significant gp120 dissociation from virion, in contrast to 48d, although the the gp41 epitope of MAb 50-69 was exposed [Poignard et al.(1996a)]
- 17b: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of 17b blocks this inhibition [Wu et al.(1996)]
- 17b: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 17b: A low avidity antibody as assessed by urea elution
- 17b: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 17b bound monomer, oligomer, and neutralized JRFL in the presence of sCD4, but if sCD4 was not present, 17b only bound monomer [Fouts et al.(1997)]
- 17b: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 17b has synergistic response in combination with anti-V3 MAb 694/98-D [Li et al.(1997)]
- 17b: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the Can0A V3 peptide, suggesting Can0A V3 is a conformer that mimics the 48d – it does not bind to 17b, distinguishing the epitopes [Weinberg et al.(1997)]

603 cont.

- 17b: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to a CD4BS MAb F105 or sCD4 [Cao et al.(1997)]
- 17b: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – partial reexposure if sCD4 was bound – could not bind to HXBc2 gp120 if the 19 C-term amino acids were deleted in conjunction with amino acids 31-93 in C1, but binding was restored in the presence of sCD4 [Wyatt et al.(1997)]
- 17b: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- 17b: 17b FAb was co-crystallized with a gp120 core and CD4, and it's binding site can be directly visualized – 17b binds to the " bridging sheet" of gp120, an antiparallel β sheet region, contacting residues from the C4 region and the V1/V2 stem – the contact area is small for an Ab-antigen interactive surface, and dominated in the Ab by the heavy chain – the center of the binding region has hydrophobic interactions, and the periphery charge interactions, acidic on 17b and basic on gp120 [Kwong et al.(1998)]
- 17b: Summary of the implications of the crystal structure of a gp120 core bound to CD4 and 17b, combined with what is known about mutations that reduce NAb binding to gp120 – probable mechanism of neutralization is interference with chemokine receptor binding – mutations in 88N, 117K, 121K, 256S, 257T, N262, Δ V3, E370, E381, F 382, R 419, I 420, K 421, Q 422, I 423, W 427, Y 435, P 438, M 475 of HXBc2 (IIIB) reduce binding – the only variable residues in gp120 that contact 17b are 202T and 434M – the contact points for 17b with the crystallized incomplete gp120 are mostly in the heavy chain of the Ab, and there is a gap between 17b's light chain and the partial gp120 which may be occupied by the V3 loop in a complete gp120 molecule – the authors propose that the V2 and V3 loops may mask the CD4i Ab binding site, and that the V2 loop may be repositioned upon CD4 binding [Wyatt et al.(1998)]
- 17b: Moore and Binley provide a commentary on the papers by [Rizzuto et al.(1998)], [Wyatt et al.(1998)] and [Kwong et al.(1998)] – they point out 17b shares binding elements in gp120 with chemokine receptor molecules, and that CD4 needs to bind to gp120 first to make the 17b epitope accessible and it may be stericly blocked in the CD4 bound virus, thus making it a poor NAb for primary isolates [Moore & Binley(1998)]
- 17b: Site directed mutagenesis of a WU2 protein with the V1-V2 loops deleted revealed key residues for 17b-gp120 interaction and interaction of gp120 and CCR5 – mutations in residues that reduced 17b by $\geq$ 70% binding were R/D 419, I/R 420, Q/L 422, Y/S 435, I/S 423, K/D 121 and K/D 421– 17b can neutralize HIV-1 strains that use different chemokine receptors, supporting a common region in gp120 in chemokine-receptor interaction [Rizzuto et al.(1998)]
- 17b: sCD4 induces 17b binding in primary isolates and TCLA strains – amino acids that reduce the efficiency of binding were determined and found also to compromise syncytia formation and viral entry – V1V2 deletion or sCD4 binding can expose the 17b epitope for both HXBc2 and macrophage tropic YU2 – neutralizing potency of 17b is probably weak due to poor exposure of the epitope – 17b epitope exposure upon sCD4 binding can occur over a wide range of temperatures, consistent with the energy of CD4 binding being sufficient to drive the V1/V2 loop into a new conformation [Sullivan et al.(1998)]
- 17b: A panel of MAbs was shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4i MAbs 17b and 48d bound better to the deleted protein than to wild type [Binley et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
604	48d	gp120(CD4i dis)	gp120(dis)	DISCONTINUOUS	L P (weak)	HIV-1 infection	human($IgG_{1\kappa}$)

Donor: J. Robinson, University of Connecticut, Storrs

References: [Thali et al.(1993), Moore & Ho(1993), Moore et al.(1993c), Thali et al.(1994), Moore et al.(1994b), D'Souza et al.(1995), Sattentau(1995), Wyatt et al.(1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Trkola et al.(1996a), Binley et al.(1997), Li et al.(1997), Weinberg et al.(1997), Lee et al.(1997), Ugolini et al.(1997), Wyatt et al.(1997), Parren et al.(1997b), Wyatt et al.(1998), Mondor et al.(1998), Parren et al.(1998), Sullivan et al.(1998), Yang et al.(1998), Binley et al.(1998)]

NOTES:

- 48d: Also called 4.8d and 4.8D
- 48d: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs
- 48d: Epitope is better exposed upon CD4 binding to gp120 – competes with ICR 39.13, 15e and 21h, anti-CD4 binding site MAbs – inhibited by anti-CD4BS MAb ICR 39.13g and linear anti-C4 MAbs G3-42 and G3-508 – 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 421 K/L, 433A/L, 438 P/R and 475 M/S confer decreased sensitivity to neutralization [Thali et al.(1993)]
- 48d: Called 4.8d – Neutralizes IIIB – reactive with SF-2 gp120 – does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- 48d: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding of 17b [Moore et al.(1993c)]
- 48d: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 21h, 15e and 17b) [Thali et al.(1994)]
- 48d: Poor cross-reactivity with gp120 from most clades [Moore et al.(1994b)]
- 48d: Called 4.8D – Found to neutralize MN, but not JRCSF, two B subtype primary isolates, or a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs[D'Souza et al.(1995)]
- 48d: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence of sCD4 involves the V1/V2 loops, with more significant involvement of V2 – similar effect observed for 17b and A32 [Wyatt et al.(1995)]
- 48d: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau et al.(1995)]
- 48d: Binds with similar affinity to monomer and oligomer, moderate association rate, potent neutralization – this is in contrast to 17b, which has very different kinetics [Sattentau & Moore(1995)]
- 48d: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) – anti-C1-C4 discontinuous epitope MAbs A32 and 2/11c enhance binding – reciprocal enhanced binding with some anti-V2 MAbs [Moore & Sodroski(1996)]
- 48d: Binding resulted in gp120 dissociation from virion, mimicking sCD4, and exposure of the gp41 epitope of MAb 50-69, in contrast to CD4BS MAbs [Poignard et al.(1996a)]

604 cont.

- 48d: Neutralizes JR-FL – slightly inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 48d: A low avidity antibody as assessed by urea elution
- 48d: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – all Ab combinations tested showed synergistic neutralization – 48d has synergistic response with MAbs 694/98-D (anti-V3) and F105 [Li et al.(1997)]
- 48d: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the Can0A V3 peptide, suggesting Can0A V3 is a conformer that mimics the 48d, (but not 17b), epitope [Weinberg et al.(1997)]
- 48d: Prefers CD4-gp120 complex to gp120 alone, but does not enhance fusion, in contrast to MAb CG10, in fact it inhibits syncytium formation [Lee et al.(1997)]
- 48d: Viral binding inhibition by 48d was strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 48d: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt et al.(1997)]
- 48d: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- 48d: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization of 48d is interference with chemokine receptor binding – CD4 binding increases exposure of epitope due to V2 loop movement – 88N, 117K, 121K, 256S, 257T, N262, delta V3, E370, E381, F 382, R 419, I 420, K 421, Q 422, I 423, W 427, Y 435, P 438, M 475 mutations in HXBc2 (IIIB) decrease binding [Wyatt et al.(1998)]
- 48d: Inhibits binding of Hx10 to both CD4 positive and CD4 negative HeLa cells [Mondor et al.(1998)]
- 48d: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
- 48d: CD4i MAbs 17b and 48d compete with MAb CG10, and the binding sites may overlap – MAb A32 enhances binding of 17b, 48d and CG10 [Sullivan et al.(1998)]
- 48d: A neutralization assay was developed based on heminested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang et al.(1998)]
- 48d: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4i MAbs 17b and 48d bound better to the deleted protein than to wild type [Binley et al.(1998)]
- 48d: NIH AIDS Research and Reference Reagent Program: 1756

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
605	A32	gp120(CD4i C1-C4 dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(IgG_1)

Donor: J. Robinson, Tulane University, LA

References: [Moore et al.(1994b), Wyatt et al.(1995), Moore & Ho(1995), Moore & Sodroski(1996), Wu et al.(1996), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997), Burton & Montefiori(1997), Wyatt et al.(1997), Boots et al.(1997), Parren et al.(1997b), Sullivan et al.(1998), Binley et al.(1998)]

NOTES:

- A32: Reacted with virtually every gp120 monomer of every clade tested, most conserved gp120 monomer epitope known [Moore et al.(1994b)]
- A32: Epitope is better exposed upon CD4 binding to gp120 – binding of A32 enhances binding of 48d and 17b – studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 [Wyatt et al.(1995)]
- A32: Review: epitope is distinct from CD4BS MAbs, 48d and 17b, and 2G12 [Moore & Ho(1995)]
- A32: Reciprocal inhibition of binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of some anti-V2 and sCD4 inducible MAbs (48d and 17b) – very similar competition pattern to 2/11c, A32 and 211/c are unique among known human and rodent MAbs [Moore & Sodroski(1996)]
- A32: Not neutralizing – binds domains that interact with gp41 – MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 and binding of A32 does not block this inhibition [Wu et al.(1996)]
- A32: Does not neutralize JR-FL, or any strain strongly – partial inhibition of gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- A32: A low avidity antibody as assessed by urea elution
- A32: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – A32 bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- A32: Review [Burton & Montefiori(1997)]
- A32: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt et al.(1997)]
- A32: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)]
- A32: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – A32 has a unique epitope involving mostly C2 but C1 and C4 contribute – six quite variable phage inserts were recognized, with a consensus of LPWYN – a central Trp was the most conserved element, consistent with W427 being an important residue for binding gp120 [Boots et al.(1997)]
- A32: Enhances binding of CD4i MAbs 17b and 48d, and a MAb generated in response to gp120-CD4 complex CG10 [Sullivan et al.(1998)]
- A32: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
606	2/11c	gp120(C1-C4 dis)	gp120(dis)	DISCONTINUOUS	L (weak)	HIV-1 infection	human

Donor: J. Robinson, Tulane University, LA

References: [Moore & Sodroski(1996), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Wyatt et al.(1997), Binley et al.(1998)]

NOTES:

- 2/11c: Also called 211c, 2.11c, 211/c and 2-11c
- 2/11c: Inhibits binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of some anti-V2 and CD4i MAbs (48d and 17b) – similar reactivity pattern to A32, but less cross-reactive and lower affinity – A32 and 211/c are unique among known human and rodent MAbs [Moore & Sodroski(1996)]
- 2/11c: Called 211c – does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)]
- 2/11c: A low avidity antibody as assessed by urea elution
- 2/11c: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – 2/11c bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- 2/11c: Called 2.11c – One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 67 μg/ml [Li et al.(1997)]
- 2/11c: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-74, are deleted [Wyatt et al.(1997)]
- 2/11c: Called 211/c – a panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]

607	N70-2.3a	gp120(272-509 dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(IgG1)

Donor: J. Robinson, Tulane University, LA

References: [Robinson et al.(1990), Takeda et al.(1992)]

NOTES:

- N70-2.3a: Broad reactivity [Robinson et al.(1990)]
- N70-2.3a: Fc receptor mediated enhancement of HIV-1 infection – binds a conformational site in the carboxyl half of gp120, distinct from 1.5e [Takeda et al.(1992)]

608	6E10	gp120 (dis)	gp120(dis)	DISCONTINUOUS	L	rsgp160	

Donor: Phil Berman

References: [Berman et al.(1991)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
609	C31	gp120(unknown) **Donor:** ? **References:** [Boyer et al.(1991)] **NOTES:** • C31: Broadly reactive group specific – high yield cultivation of human MAb [Boyer et al.(1991)]	gp120	?	N	HIV-1 infection	human($IgG_{1\kappa}$)
610	P5-3	gp120(unknown) **Donor:** Evan Hersh and Yoh-Ichi Matsumoto **References:** [Robinson Jr. et al.(1990a), Pincus et al.(1991)] **NOTES:** • P5-3: No enhancing activity for HIV-1 IIIB [Robinson Jr. et al.(1990a)] • P5-3: Poor immunotoxin activity when coupled to RAC – isotype specified as: $IgG_{3\lambda}$ [Pincus et al.(1991)] • P5-3: NIH AIDS Research and Reference Reagent Program: 378	gp120	?		HIV-1 infection	human($IgG_{1\lambda}$)
611	BAT401	gp120(unknown) **Donor:** ? **References:** [Fung et al.(1987)]	gp120	?	L	Intact IIIB	murine(IgG_1)
612	BAT267	gp120(unknown) **Donor:** ? **References:** [Fung et al.(1987)]	gp120	?	L	Inact IIIB	murine(IgG_1)
613	BAT509	gp120(unknown) **Donor:** ? **References:** [Fung et al.(1987)]	gp120	?	L	Inact IIIB	murine(IgG_1)
614	13.10	gp120(unknown) **Donor:** Evan Hersh and Yoh-Ichi Matsumoto **References:** [Lake et al.(1989), Moran et al.(1993), Wisnewski et al.(1996)] **NOTES:** • 13.10: Also called No. 13 • 13.10: First HIV-1 specific human-mouse hybridoma that produces a MAb that binds to gp120 and gp160 [Lake et al.(1989)] • 13.10: Heavy (V_HI) and light (V_λII) chain sequenced – no enhancing or neutralizing activity – called No. 13 [Moran et al.(1993)] • 13.10: 13.10 is V_H1 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)] • 13.10: NIH AIDS Research and Reference Reagent Program: 377	gp120	?	N	HIV-1 infection	human($IgG_{1\lambda}$)

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
615	F285	Env(unknown)	gp120	?		HIV-1 infection	human(IgG_1)

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Wisnewski et al.(1995), Wisnewski et al.(1996)]
NOTES:
- F285: F285 is V_H1 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]

616	HBW4	gp120(unknown IIIB)	gp120	?		HIV-1 infection	human($IgG_{1\lambda}$)

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Moran et al.(1993), Wisnewski et al.(1995), Wisnewski et al.(1996)]
NOTES:
- HBW4: Heavy (V_HII) and light (V_λII) chain sequenced [Moran et al.(1993)]
- HBW4: HBW4 is V_H2 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]

617	multiple Fabs	gp120(unknown)	gp120	?		HIV-1 infection	human

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Burton et al.(1991)]
NOTES:
- A panel of anti-gp120 Fabs was generated by antigen selection from a random combinatorial library prepared from bone marrow from an asymptomatic individual [Burton et al.(1991)]

618	multiple MAbs	gp120(unknown)	gp120	?		gp120 complexed with MAb M77	murine

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Denisova et al.(1996)]
NOTES:
- When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes, as well as an array of MAbs to discontinuous epitope – 10 of 36 MAbs were mapped to linear epitopes and are mentioned elsewhere in this database, the others are: GV5H1, GV4D5, GV4G10, GV1A8, GV10H5, GV8E11, GV2H4, GV6E6, GV1F7, GV1G9, GV4G5, GV6B12, GV1E8, GV2B7, GV1B11, GV6H5, GV6G2, GV6B5, GV1E10, GV5E3, GV5B9, GV5F4, GV6G4, GV1A12, GV5C11, GV6B6, GV3C10 [Denisova et al.(1996)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
619	multiple MAbs	gp120(dis)	gp120	?		gp120	murine

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Denisova et al.(1996)]
NOTES:
- When gp120 was used as an immunogen, in contrast to gp120 bound to an anti-V3 MAb, few MAbs were generated and all bound better to the native than to the denatured protein – MAbs generated were: G1B12, G2F7, G9G8, G12F12, G1B8, G11F11, G9E8, G1B11, G1B6, G6F2, G2E7 [Denisova et al.(1996)]

620	multiple MAbs	gp120(dis)	gp120(dis)	?		gp120-CD4 complex	murine

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Denisova et al.(1996)]
NOTES:
- When gp120-CD4 was used as an immunogen, in contrast to gp120 bound to an anti-V3 MAb, few MAbs were generated and all bound better to the native than to the denatured protein – MAbs generated were: CG43, CG41, CG49, CG53, CG42, CG4, CG46, CG40, CG52, CG51, CG48, CG50, CG125, CG124, CG121 [Denisova et al.(1996)]

621	1025	gp120(dis)	gp120(dis)				

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Berman et al.(1997)]
NOTES:
- 1025: Binds to 1/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)]

622	human sera	gp120	gp120			HIV-1 infection	human(IgG)

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Binley et al.(1997)]
NOTES:
- Retention of anti-Env antibodies and loss of anti-Gag antibodies during progression was studied, and suggested to be the result of the loss of T-cell help and the unique ability of Env to stimulate B cells even in a backdrop of declining CD4 cells, because of the ability of Env to bind to the CD4 molecule [Binley et al.(1997)]

	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
623	polyclonal	gp120	gp120		Y	HIV-1 Pr55gag VLP with anchored gp120 or V3+CD4 linear domains	Macaca mulatta

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Wagner et al.(1998)]
NOTES:

- A VLP is a non-infectious virus-like particle self-assembled from HIV Pr55 gag – macaques were immunized with VLPs bound to either gp120 or V3+CD4 linear domains – gag and env CTL specific CTL were stimulated in each case, and Ab response to gag and gp120 and was elicited, but the gp120 neutralizing response occurred only with whole gp120, not V3+CD4 – despite the CTL and Ab response, immunized macaques were infected by intervenous challenge with SHIV chimeric challenge stock [Wagner et al.(1998)]

624	polyclonal	gp120(IIIB)	gp120			gp120 or gp160 DNA vaccine	murine

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Wagner et al.(1998)]
NOTES:

- DNA vaccinations of BALBc mice with a gp120 or gp160 DNA vaccine elicited a strong T cell proliferative response with Th1-like secretion of γ interferon and IL-2, with little or no IL-4, as well as antigen specific gp120 Abs [Shiver et al.(1997)]

625	polyclonal	gp120	gp120		L	DNA gag/pol, vif, and CMN160 vaccine	murine

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Kim et al.(1997)]
NOTES:

- A gag/pol, vif or CMN160 DNA vaccine, when delivered in conjunction with the plasmid encoding the co-stimulatory molecules B7 and IL-12, gave a dramatic increase in both the cytotoxic and proliferative responses in mice
- The Ab response was detected by ELISA, but the CMN160 DNA vaccinated mice showed a neutralizing Ab response