Table 12: gp160

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
236	M85	gp160(30–51)	gp120(30–51 LAI)	ATEKLWVTVYYGVPVWKEAT-TT	no	451 Env	murine(IgG_1)

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

- M85: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese (1992)]
- M85: C1 domain – mutation 40 Y/D impairs binding – the relative affinity for denatured/native gp120 is < .01, suggesting conformational component [Moore (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 (1997)]

237	7E2/4	gp160(31–50)	gp120(31–50 LAI)	TEKLWVTVYYGVPVWKEATT		Env glycopro	murine(IgG)

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

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

238	M92	gp160(41–50)	gp120(31–50 LAI)	GVPVWKEATT	no	451 Env	rat(IgG_1)

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

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

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
239	4D4#85	gp160(41–50)	gp120( )	GVPVWKEATT		Env	murine(IgG)
	Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA **References:** [Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Wyatt (1997), Binley (1998)] • 4D4#85: C1 domain – the relative affinity, denatured/native gp120 is 0.1 – mutation 45 W/S impairs binding [Moore (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 (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 (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]					
240	M86	gp160(42–61)	gp120( )	VPVWKEATTTLFCASDAKAY	no	451 Env	murine(IgG_1)
	Donor: Fulvia di Marzo Veronese **References:** [di Marzo Veronese (1992), Moore (1994c)] • M86: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese (1992)] • M86: C1 domain – the relative affinity for denatured/native gp120 is 1 [Moore (1994c)]						
241	polyclonal	gp160(51–70)	Env(42–61 LAI)	LFCASDAKAYDTEVHNVWAT	no	Vaccinia expressing Env fused with vaccinia proteins p14 and p39	murine( )
	References: [Collado (2000)] • Vaccinia p14 can elicit NAbs and p39 tends to be immunodominant, so these proteins were fused to regions of HIV-1 Env – reduced glycosylation was noted when p14 or p39 was placed in the N-term region of the fusion protein – chimeric proteins shifted the Env Ab response from V3 to either a C1 or C4 domain, depending on the construct – all chimeric Env proteins: 14kEnv, 39kEnv, and Env39k elicited a strong Ab response to the C1 region of gp120 (LFCASDAKAYDTEVHNVWAT), and Env39k mounted a strong response to the C4 region (KAMYAPPISGQIRCSSNITG) [Collado (2000)]					
242	133/237	gp160(61–70)	gp120( )	YDTEVHNVWA	L	IIIB gp120	murine(IgG_1)
	References: [Niedrig (1992b), Moore (1994c), Moore (1994d)] • 133/237: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig (1992b)] • 133/237: The relative affinity, denatured/native gp120 is 1.4 – mutation of position 69 W/L impairs binding [Moore (1994c)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
243	133/290	gp160(61–70)	gp120( )	YDTEVHNVWA	L	IIIB gp120	murine(IgG_1)

References: [Niedrig (1992b), Thali (1993), Moore (1994c), Moore (1994d), Wyatt (1995), Binley (1997a), Wyatt (1997), Binley (1998)]

- 133/290: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig (1992b)]
- 133/290: The relative affinity for denatured/native gp120 is 2.2 – mutation in position 69 W/L impairs binding [Moore (1994c)]
- 133/290: Used for antigen capture assay, either to bind gp120 to the ELISA plate, or to quantify bound gp120 [Wyatt (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: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt (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 (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

244	133/11	gp160(64–78)	gp120( )	EVHNVWATHACVPTD	L	IIIB gp120	murine(IgG_1)

References: [Niedrig (1992b)]

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

245	D/3G5	gp160(73–82)	gp120( )	ACVPTDPNPQ	no	Baculovirus-expressed rgp120 LAI	murine(IgG_1)

References: [Bristow (1994)]

- D/3G5: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow (1994)]

246	D/6A11	gp160(73–82)	gp120( )	ACVPTDPNPQ	no	Baculovirus-expressed rgp120 LAI	murine( )

References: [Bristow (1994)]

- D/6A11: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow (1994)]

247	D/5E12	gp160(73–92)	gp120( )	ACVPTDPNPQEVVLVNVTEN	no	Baculovirus-expressed rgp120 LAI	murine( )

References: [Bristow (1994)]

- D/5E12: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow (1994)]

248	L5.1	gp160(79–93)	gp120( )	PNPQEVVLVNVTENF		vaccinia gp160	murine(IgG)

References: [Akerblom (1990)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
249	4A7C6	gp160(81–90)	gp120( )	PQEVVLVNVT		Env glycopro	murine(IgG)
	Donor: R. Tedder **References:** [Thiriart (1989), Thali (1993), Moore & Ho(1993), Moore (1994c), Moore (1994d), Moore & Sodroski(1996)] • 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 (1994c)] • 4A7C6: C1 region epitope (88 N/P substitutions abrogates binding), but substitutions 380 G/F and 420 I/R also impaired binding [Moore (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						
250	1D10	gp160(81–100)	gp120( )	PQEVVLVNVTENFDMWKNDM	L	IIIB-rgp120	rat( )
	References: [Dowbenko (1988), Berman (1991), Nakamura (1992), Moore (1994c)] • 1D10: Cross-blocks 5B3 in IIIB-rsgp160 ELISA – type specific in rgp120 ELISA binding [Nakamura (1992)] • 1D10: The relative affinity for denatured/native gp120 is 13 – mutation 88 N/P impairs binding [Moore (1994c)]						
251	B242	gp160(83–92)	gp120( )	EVVLVNVTEN	no	Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	murine(IgG_1)
	References: [Bristow (1994)] • B242: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow (1994)]						
252	133/192	gp160(91–100)	gp120( )	ENFDMWKNDM	L	IIIB gp120	murine(IgG_1)
	Donor: Matthias Niedrig **References:** [Niedrig (1992b), Moore (1993b), Moore (1994c), Moore & Sodroski(1996), Trkola (1996a), Binley (1997a), Binley (1998)] • 133/192: Epitope seems complex, binds multiple peptides – weak neutralization of lab strain [Niedrig (1992b)] • 133/192: The relative affinity for denatured/native gp120 is 1.8 [Moore (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 (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 (1996a)] • 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 (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
253	C6	gp160(91–100)	gp120( )	ENFDMWKNDM		mis-folded LAI rgp160	murine(IgG_1)

References: [Pincus & McClure(1993), Abacioglu (1994), Moore (1994c), Pincus (1996)]

- C6: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu (1994)]
- C6: The relative affinity for denatured/native gp120 is 0.9 [Moore (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 (1996)]
- C6: NIH AIDS Research and Reference Reagent Program: 810

254	B2	gp160(91–100)	gp120( )	ENFDMWKNDM		mis-folded LAI rgp160	murine(IgG_{2b})

References: [Thali (1993), Abacioglu (1994), Moore (1994c), Moore (1994d), Binley (1997a)]

- B2: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu (1994)]
- B2: The relative affinity for denatured/native gp120 is 1.4 [Moore (1994c)]
- B2: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.)

255	B10	gp160(91–100)	gp120( )	ENFDMWKNDM		mis-folded LAI rgp160	murine(IgG_1)

References: [Abacioglu (1994), Moore (1994c)]

- B10: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu (1994)]
- B10: The relative affinity for denatured/native gp120 is 0.4 [Moore (1994c)]
- B10: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.)

256	489.1(961)	gp160(91–100)	gp120( )	ENFDMWKNDM		Env	murine(IgG)

Donor: C. Bruck, SKB, Belgium

References: [Moore (1994c)]

- 489.1(961): The relative affinity for denatured/native gp120 is 1 [Moore (1994c)]
- 489.1(961): NIH AIDS Research and Reference Reagent Program: 961

257	T1.1	gp160(91–100)	gp120( )	ENFDMWKNDM		vaccinia gp160	murine(IgG)

References: [Akerblom (1990), Broliden (1990), Moore (1994c)]

- T1.1: Also reacted in solid phase with gp120(234-248) NGTGPCTNVSTQCT [Akerblom (1990)]
- T1.1: No ADCC activity – reactive peptide: NVTENFNMWKNDMVEQ, IIIB [Broliden (1990)]
- T1.1: C1 region – the relative affinity for denatured/native gp120 is 1 [Moore (1994c)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
258	T7.1	gp160(91–100)	gp120( )	ENFDMWKNDM		Env	murine(IgG)
	References: [Akerblom (1990), Bolmstedt (1990), Moore (1994c), Moore (1994d)] • T7.1: The relative affinity of denatured/native gp120 is 4.0 [Moore (1994c)]						
259	T9	gp160(91–100)	gp120( )	ENFDMWKNDM		Env	murine(IgG)
	Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula **References:** [Akerblom (1990), Bolmstedt (1990), Moore (1994c), Moore (1994d), Binley (1997a)] • T9: The relative affinity of denatured/native gp120 is 7.9 [Moore (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 (1994d)]						
260	5B3	gp160(91–100)	gp120( )	ENFDMWKNDM	no	IIIB-rspg160	murine(IgG)
	References: [Berman (1991), Nakamura (1992), Beretta & Dalgleish(1994), Moore (1994c)] • 5B3: Blocks gp120 -CD4 binding [Berman (1991)] • 5B3: Cross-blocks 1D10 in competitive IIIB-rsgp160 ELISA – no neutralization – blocks IIIB-gp120 sCD4 binding – localized binding to residues 72–106 [Nakamura (1992)] • 5B3: The relative affinity of denatured/native gp120 is 8.3 [Moore (1994c)]						
261	MF49.1	gp160(91–100)	gp120( )	ENFDMWKNDM		Env	murine(IgG)
	References: [Thiriart (1989), Moore (1994c)] • MF49.1: The relative affinity of denatured/native gp120 is 3.8 [Moore (1994c)]						
262	GV4D3	gp160(92–100)	gp120(92–100 IIIB)	NFNMWKNDM		gp120 complexed with MAb M77	murine( )
	References: [Denisova (1996)] • 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 (1996)]						
263	B9	gp160(93–96)	gp120( )	FNMW		mis-folded LAI rgp160	murine(IgG_1)
	References: [Abacioglu (1994)] • B9: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)]						
264	B27	gp160(93–96)	gp120( )	FNMW	no	Baculovirus-expressed mis-folded rgp160 IIIB: NL43, MicroGenSys	murine(IgG_1)
	References: [Abacioglu (1994), Bristow (1994)] • B27: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)] • B27: MAbs generated in the context of a study of the humoral immune response to rgp120 and rgp160 [Bristow (1994)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
265	B35	gp160(93–98)	gp120( )	FNMWKN		mis-folded LAI rgp160	murine(IgG_1)
		References: [Abacioglu (1994)]					
		• B35: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)]					
266	D/4B5	gp160(93–101)	gp120( )	FNMWKNDMV	no	Baculovirus-expressed rgp120 LAI	murine( )
		References: [Bristow (1994)]					
		• D/4B5: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow (1994)]					
267	D/6B2	gp160(93–101)	gp120( )	FNMWKNDMV	no	Baculovirus-expressed rgp120 LAI	murine(IgG_1)
		References: [Bristow (1994)]					
		• D/6B2: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow (1994)]					
268	D/5A11	gp160(93–101)	gp120( )	FNMWKNDMV	no	Baculovirus-expressed rgp120 LAI	murine( )
		References: [Bristow (1994)]					
		• D/5A11: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow (1994)]					
269	B20	gp160(101–110)	gp120( )	VEQMHEDIIS		mis-folded LAI rgp160	murine(IgG_{2a})
		References: [Abacioglu (1994), Moore (1994c)]					
		• B20: C1 region – epitope boundaries mapped by peptide scanning – HEDII core [Abacioglu (1994)]					
		• B20: The relative affinity for denatured/native gp120 is 1 [Moore (1994c)]					
270	B18	gp160(101–110)	gp120( )	VEQMHEDIIS		mis-folded LAI rgp160	murine(IgG_{2a})
		References: [Abacioglu (1994), Moore (1994c)]					
		• B18: C1 region – epitope boundaries mapped by peptide scanning, HEDII core [Abacioglu (1994)]					
		• B18: The relative affinity for denatured/native gp120 is 1 [Moore (1994c)]					

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
271	MF39.1	gp160(101–110)	gp120( )	VEQMHEDIIS		Env	murine(IgG)
	References: [Thiriart (1989), Cook (1994), Moore (1994c)]						
	• 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 (1994)]						
	• MF39.1: The relative affinity of denatured/native gp120 is 30 [Moore (1994c)]						
272	T2.1	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV		Env	murine(IgG)
	Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula						
	References: [Akerblom (1990), Bolmstedt (1990), Moore (1994c), Moore (1994d)]						
	• 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 (1994c)]						
273	6D8	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV		IIIB-rgp120	rat( )
	References: [Dowbenko (1988), Nakamura (1992), Moore (1994c)]						
	• 6D8: Highly cross reactive with multiple stains by rgp120 ELISA [Nakamura (1992)]						
	• 6D8: The relative affinity for denatured/native gp120 is 15 – mutations 113 D/R and 113 D/A impair binding [Moore (1994c)]						
274	M96	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV	no	451 Env	rat(IgG_{2a})
	Donor: Fulvia di Marzo Veronese						
	References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d)]						
	• M96: Immunoblot reactive for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)]						
	• M96: C1 region – the relative affinity for denatured/native gp120 is 6 [Moore (1994c)]						
275	37.1.1(ARP 327)	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV		Env glycopro	murine(IgG)
	Donor: Claudine Bruck						
	References: [Thiriart (1989), Moore & Ho(1993), Moore (1994c)]						
	• 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 (1994c)]						
	• 37.1.1: UK Medical Research Council AIDS reagent: ARP327						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
276	187.2.1	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV		Env glycopro	murine(IgG)

Donor: Claudine Bruck and Clothilde Thiriart

References: [Thiriart (1989), Moore & Ho(1993), Cook (1994), Moore (1994c), Moore (1994d)]

- 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 (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 (1994c)]
- 187.2.1: UK Medical Research Council AIDS reagent: ARP332

277	MF58.1	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV		Env	murine(IgG)

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

278	MF77.1	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV		Env	murine(IgG)

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

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

279	MF119.1	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV		Env	murine(IgG)

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

- 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 (1994c)]

280	MF4.1	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV		Env	murine(IgG)

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

- MF4.1: The relative affinity for denatured/native gp120 is 8 [Moore (1994c)]

281	MF53.1	gp160(101–120)	gp120( )	VEQMHEDIISLWDQSLKPCV		Env	murine(IgG)

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

- MF53.1: The relative affinity for denatured/native gp120 is 10 [Moore (1994c)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
282	11/65	gp160(102–121)	gp120(311–321 HXB10)	EQMHEDIISLWDQSLKPCVK		rgp120 BH10	rat(IgG_{2b})

References: [McKeating (1992a), McKeating (1993b), Peet (1998)]

- 11/65: Binds only soluble gp120, not virion bound – used to quantify gp120 shedding – (numbering is incorrect in original?) [McKeating (1992a)]
- 11/65: Called 11/65a/5h – The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – 11/65 was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 11/65: UK Medical Research Council AIDS reagent: ARP3076

283	W1	gp160(102–121)	gp120( )	EQMHEDIISLWDQSLKPCVK		Env	murine(IgG)

Donor: D. Weiner, U. Penn.

References: [Moore (1994c)]

- W1: The relative affinity for denatured/native gp120 is 6 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore (1994c)]

284	T11	gp160(102–125)	gp120(102–125)	EQMHEDIISLWDQSLKPCVKL-TPL		rec gp140	murine( )

Donor: R. Doms, Univ. of Pennsylvania

References: [Earl (1994), Jagodzinski (1996)]

- 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 (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 (1996)]

285	GV1A8	gp160(105–113)	gp120(105–113 IIIB)	HEDIISLWD		gp120 complexed with MAb M77	murine( )

References: [Denisova (1996)]

- 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 (1996)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
286	135/9	gp160(111–120)	gp120( )	LWDQSLKPCV	L	IIIB gp120	murine(IgG_1)

Donor: Matthias Niedrig

References: [Niedrig (1992b), Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Trkola (1996a), Binley (1997a), Kropelin (1998)]

- 135/9: Defines the epitope as gp120(114-123) MHEDIISLWD (core LWD?) – weak neutralization of lab strain [Niedrig (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 (1994c)]
- 135/9: Substitutions 106 E/A, 113 D/A or R, and 117 K/W impair binding, some substitutions enhance binding [Moore (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 α-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 (1996a)]
- 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 (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]
- 135/9: Noted to bind to C1 peptide HEDIISLWDQSLK – blocks gp120 interaction with CD4+ cells – blocking activity is additive when combined with antibodies which bind in the C4 region of gp120 (F105, 388/389, and b12) [Kropelin (1998)]

287	MF46.1	gp160(111–120)	gp120( )	LWDQSLKPCV		Env	murine(IgG)

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

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

288	C4	gp160(111–120)	gp120( )	LWDQSLKPCV		mis-folded LAI rgp160	murine(IgG_1)

Donor: George Lewis

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

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

289	11	gp160(111–120)	gp120( )	LWDQSLKPCV		Env	murine(IgG)

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

- 11: The relative affinity for denatured/native gp120 is 7.8 – mutation 113 D/R impairs binding [Moore (1994c)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
290	12G10	gp160(111–120)	gp120( )	LWDQSLKPCV		Env	murine(IgG)
		References: [Thiriart (1989), Moore (1994c)]					
		• 12G10: The relative affinity for denatured/native gp120 is 17 – mutation 117 K/W impairs binding [Moore (1994c)]					
291	7C10	gp160(111–120)	gp120( )	LWDQSLKPCV		Env	murine(IgG)
		References: [Thiriart (1989), Moore (1994c)]					
		• 7C10: The relative affinity for denatured/native gp120 is 5.8 – mutation 117 K/W impairs binding [Moore (1994c)]					
292	6D5	gp160(122–141)	gp120( )	LTPLCVSLKCTDLKNDTNTN		Env	murine(IgG)
		Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA					
		References: [Moore (1994c), Moore (1994d)]					
		• 6D5: The relative affinity for denatured/native gp120 is 15 – mutations Delta119-205 and 125 L/G impair binding [Moore (1994c)]					
293	B33	gp160(123–142)	gp120( )	TPLCVSLKCTDLGNATNTNS	no	Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	murine($IgG_{2b}\kappa$)
		Donor: Daniels					
		References: [Abacioglu (1994), Bristow (1994)]					
		• B33: There are two MAbs in the literature named B33. See also gp41, LAI 123–142 [Abacioglu (1994)]					
		• B33: MAbs generated in the context of a study of the humoral immune response to rgp120 and rgp160 [Bristow (1994)]					
		• B33: UK Medical Research Council AIDS reagent: ARP304, gp160/41 binding					
294	polyclonal	gp160(131–151)	Env(131–151)	CTDLKNDTNTNSSSGRMMME-K		HIV-1 infection	human( )
		References: [Carlos (1999)]					
		• Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]					
295	2H1B	gp160(155–161)	gp120( )	RNISFKA	no	Peptide	murine( )
		References: [Matsushita (1995)]					
		• 2H1B: Binds in WB, but binds poorly to Env on the cell surface [Matsushita (1995)]					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
296	697-D	gp160(161–180)	gp120(V2 161–180 IIIB)	ISTSIRGKVQKEYAFFYKLD	P (weak)	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center) or Cellular Products Inc, Buffalo NY
References: [Gorny (1994), Forthal (1995), Moore & Ho(1995), Trkola (1996a), Binley (1997a), Fouts (1997), Parren (1997b), Nyambi (1998), Stamatatos & Cheng-Mayer(1998), Gorny (2000), Hioe (2000), Nyambi (2000)]

- 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 (1994)]
- 697-D: Not neutralizing, no ADCC activity, and no viral enhancing activity [Forthal (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 (1996a)]
- 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 (1997)]
- 697-D: Does not neutralize TCLA strains but neutralizes some primary isolates weakly [Parren (1997b)]
- 697-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – V2 Abs 697-D, 1361, and 1357 tended to bind weakly with a similar pattern of specificity to virions, and bound well to soluble gp120: weak binding to 1/4 B clade viruses (CA5), and weak binding to viruses from subtype A and D [Nyambi (1998)]
- 697-D: Called 697-30D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V2 MAbs G3.4, G3.136, or 687-30D [Stamatatos & Cheng-Mayer(1998)]
- 697-D: Binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V2 MAbs 697-D, 1357 and 1361 favored the monomer by approximately 2-fold[Gorny (2000)]
- 697-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inihibited proliferative responses of gp120 specific T cells – V2 MAb 697-D did not effect proliferation [Hioe (2000)]
- 697-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 anti-V2 MAbs, which showed weak and sporadic binding, with the most frequent binding to C and D clades [Nyambi (2000)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
297	C108G	gp160(162–169)	gp120( )	STSIRGKV	L	IIIB infection	chimpanzee(IgG$_1\kappa$)

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

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

- 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 (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 (1995)]
- C108G: Characterization of MAb variable region [Warrier (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 (1996)]
- C108G: Viral binding inhibition by C108G was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- C108G: Inhibits HX10 binding to both CD4 positive and negative HeLa cells[Mondor (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)]

298	6C4/S	gp160(162–169)	gp120( )	STSIRGKV		BH10 gp120	( )

Donor: S. Ranjbar (NIBSC, UK)

References: [Moore (1993a)]

- 6C4/S: UK Medical Research Council AIDS reagent: ARP3049

299	10/76b	gp160(162–170)	gp120( )	STSIRGKVQ	L (HXB10)	BH10 rgp120	rat(IgG$_{2a}$)

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

- 10/76b: R to L substitution abrogated binding – human sera recognize epitope [McKeating (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 (1995)]
- 10/76b: Included in cross-competition and neutralization studies [Shotton (1995)]
- 10/76b: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (1995)]
- 10/76b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]
- 10/76b: UK Medical Research Council AIDS reagent: ARP3077

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
300	11/4c	gp160(162–170)	gp120(152–181)	STSIRGKVQ	L (HXB2)	BH10 rgp120	rat(IgG_{2a})

References: [McKeating (1993b), Wu (1995), Shotton (1995), Peet (1998)]

- 11/4c: R to L substitution abrogated binding – human sera recognize epitope [McKeating (1993b)]
- 11/4c: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (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 (1995)]
- 11/4c: Called 11/4c/1j/4j – The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – 11/4c was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 11/4c: UK Medical Research Council AIDS reagent: ARP3035

301	11/41e	gp160(162–170)	gp120( )	STSIRGKVQ	L (HXB10)	rgp120 LAI:BH10	rat(IgG_1)

References: [McKeating (1993b), Shotton (1995), Wu (1995)]

- 11/41e: R to L abrogated binding – human sera recognize the epitope [McKeating (1993b)]
- 11/41e: Included in cross-competition and neutralization studies [Shotton (1995)]
- 11/41e: HX10 strain specificity – binds native and deglycosylated gp120 [Wu (1995)]

302	11/4b	gp160(162–170)	gp120( )	STSIRGKVQ	L (HXB10)	rgp120 LAI:BH10	rat(IgG_{2a})

References: [McKeating (1993b), Shotton (1995), Wu (1995), Moore & Sodroski(1996)]

- 11/4b: A change from R to L abrogated binding – human sera recognize epitope [McKeating (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 (1995)]
- 11/4b: HXB10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (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)]

303	RSD-33	gp160(162–170)	gp120( )	STSIRGKVQ		BH10 gp120	( )

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

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
304	12b	gp160(162–181)	gp120( )	STSIRGKVQKEYAFFYKLDI	L (HXB10)	BH10 rgp120	rat(IgG_{2a})

References: [Shotton (1995), McKeating (1996)]

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

305	G3-4	gp160(170–180)	gp120( )	QKEYAFFYKLD	L	IIIB gp120	murine($IgG_{2b}\kappa$)

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

References: [Ho (1991a), Ho (1992), Fung (1992), McKeating (1992a), Moore & Ho(1993), Sullivan (1993), Sattentau (1993), Thali (1993), Moore (1993a), Moore (1994b), Gorny (1994), Thali (1994), Yoshiyama (1994), Wu (1995), Sattentau & Moore(1995), Jagodzinski (1996), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Stamatatos (1997), Ditzel (1997), Wyatt (1997), Parren (1998a), Stamatatos & Cheng-Mayer(1998), Ly & Stamatatos(2000)]

- G3-4: Binding is sensitive to removal of glycans by endo H – 50% neutralization of 4/9 primary isolates – has conformational features [Ho (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 (1992)]
- G3-4: Substitutions in residues 176 to 184 affect MAb recognition – substitutions in V2 can result in gp120-gp41 dissociation [Sullivan (1993)]
- G3-4: Increased binding in the presence of sCD4 [Sattentau (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 (1993a)]
- G3-4: Conformationally sensitive – sporadic cross-reactivity among, and outside, B clade gp120s [Moore (1994b)]
- G3-4: Weakly neutralizing, IC 50 = 53 μg/ml [Gorny (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 (1994)]
- 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 (1994)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
G3-4 cont.						

- 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 (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 (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 (1996a)]
- 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 (1997)]
- G3-4: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (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 (1998a)]
- G3-4: Called G3.4 – Deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V2 MAbs G3.4, G3.136, or 687-30D [Stamatatos & Cheng-Mayer(1998)]
- G3-4: Called G3.4 – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG_1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391-95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
306	G3-136	gp160(170–180)	gp120( )	QKEYAFFYKLD	L	purified IIIB gp120	murine(IgG)

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

References: [Fung (1992), Pirofski (1993), Thali (1993), Moore & Ho(1993), Moore (1993a), Yoshiyama (1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Stamatatos (1997), Ditzel (1997), Wyatt (1997), Parren (1998a), Stamatatos & Cheng-Mayer(1998), Ly & Stamatatos(2000)]

- 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 (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 (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 (1993a)]
- G3-136: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity [Yoshiyama (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 (1996a)]
- 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 (1997)]
- G3-136: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (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 (1998a)]
- G3-136: Called G3.136 – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V2 MAbs G3.4, G3.136, or 687-30D [Stamatatos & Cheng-Mayer(1998)]
- G3-136: Called G3.136 – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG_1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391-95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
307	BAT085	gp160(171–180)	gp120( )	KEYAFFYKLD	L	Inact IIIB	murine(IgG_1)

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

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

- 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 (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 (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 (1993a)]
- BAT085: Multi-lab study for antibody characterization and assay comparison – did not bind MN or SF2 –D'Souza94
- BAT085: Interacts with two overlapping peptides with region of overlap KEYAFFYKLD [Gorny (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 (1994)]
- BAT085: HXB10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (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 (1996a)]
- 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 (1998a)]

308	60b	gp160(172–181)	gp120( )	EYAFFYKLDI	no	BH10 rgp120	rat(IgG_{2b})

References: [Shotton (1995)]

- 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 (1995)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
309	74	gp160(172–181)	gp120( )	EYAFFYKLDI	no	BH10 rgp120	rat(IgG_1)
	References: [Shotton (1995)]						
	• 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 (1995)]						
310	38/12b	gp160(172–191)	gp120( )	EYAFFYKLDIIPIDNDTTSY		BH10 gp120	rat( )
	References: [Wu (1995)]						
	• 38/12b: Broad specificity: HXB2, MN, SF162 – binds native and deglycosylated gp120 [Wu (1995)]						
311	38/60b	gp160(172–191)	gp120( )	EYAFFYKLDIIPIDNDTTSY		BH10 gp120	rat( )
	References: [Wu (1995)]						
	• 38/60b: Strain specificity: HXB2 – binds native and deglycosylated gp120 [Wu (1995)]						
312	polyclonal	gp160(176–196)	Env( )	FYKLDIVPIDNTTTSYRLISC		HIV-1 infection	human( )
	References: [Carlos (1999)]						
	• Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]						
313	3D3.B8	gp160(211–221)	gp120(211–220 LAI)	EPIPIHYCAPA		Env glycopro	murine(IgG)
	References: [Bolmstedt (1990), Moore (1994c)]						
	• 3D3.B8: The relative affinity denatured/native gp120 is greater than 10 [Moore (1994c)]						
314	4C11.D8	gp160(211–221)	gp120(211–220 LAI)	EPIPIHYCAPA		Env glycopro	murine(IgM)
	References: [Bolmstedt (1990), Moore (1994c)]						
	• 4C11.D8: The relative affinity denatured/native gp120 is greater than 10 [Moore (1994c)]						
315	322-151	gp160(211–221)	gp120(201–220 LAI)	EPIPIHYCAPA		Env glycopro	murine(IgG)
	Donor: G. Robey, Abbot Labs						
	References: [Moore (1994c), Moore (1994d)]						
	• 322-151: The relative affinity denatured/native gp120 is 30 [Moore (1994c)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
316	493-156	gp160(211–230)	gp120(211–230 LAI)	EPIPIHYCAPAGFAILKCNN		Env glycopro	murine(IgG)
	Donor: G. Robey, Abbot Labs **References:** [Moore (1994c)] • 493-156: The relative affinity denatured/native gp120 is >10 [Moore (1994c)]						
317	110.1	gp160(212–221)	gp120(200–217)	PIPIHYCAPA	no	Env glycoprotein	human( )
	References: [Pincus & McClure(1993), Pincus (1996), Valenzuela (1998)] • 110.1: There is another antibody with this ID that binds to Env at positions 491–500 in LAI, see [Gosting (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 (1996)]						
318	GV4H3	gp160(219–226)	gp120(219–226 IIIB)	APAGFAIL		gp120 complexed with MAb M77	murine( )
	References: [Denisova (1996)] • GV4H3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes [Denisova (1996)]						
319	J1	gp160(222–231)	gp120(222–231 LAI)	GFAILKCNNK		Peptide	murine(IgG_1)
	Donor: J. Hoxie, U. Penn. **References:** [Moore (1994c), Moore (1994d), Cook (1994)] • J1: The relative affinity denatured/native gp120 is 30 [Moore (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 (1994)]						
320	J3	gp160(222–231)	gp120(222–231 LAI)	GFAILKCNNK		Peptide	murine(IgG_1)
	Donor: J. Hoxie, U. Penn. **References:** [Moore (1994c), Cook (1994)] • J3: The relative affinity denatured/native gp120 is 30 [Moore (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 (1994)]						

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
321	847-D	gp160(236–245)	gp120(241–251)	KGSCKNVSTV			human($IgG_1\lambda$)
	References: [Hioe (2000), Nyambi (2000)]						
	• 847-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inihibited proliferative responses of gp120 specific T cells – C2 MAbs 1006-30-D and 847-D did not effect proliferation [Hioe (2000)]						
	• 847-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including two C2 MAbs – the binding of anti-C2 MABs was weak to isolates from clades B, C, D, E, F, G, and they did not not bind to isolates from subtypes A and H – epitope is suggested to be in a 20 amino acid peptide KGSCKNVSTVQCTHGIRPVV [Nyambi (2000)]						
322	1006-30-D	gp160(236–245)	gp120(241–251)	KGSCKNVSTV			human($IgG_1\lambda$)
	References: [Hioe (2000), Nyambi (2000)]						
	• 1006-30-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inihibited proliferative responses of gp120 specific T cells – C2 MAbs 1006-30-D and 847-D did not effect proliferation [Hioe (2000)]						
	• 847-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including two C2 MAbs – the binding of anti-C2 MABs was weak to isolates from clades B, C, D, E, F, G, and they did not not bind to isolates from subtypes A and H – epitope is suggested to be in a 20 amino acid peptide KGSCKNVSTVQCTHGIRPVV [Nyambi (2000)]						
323	MF87.1	gp160(252–261)	gp120(242–261 LAI)	RPVVSTQLLL		Env	murine(IgG)
	References: [Thiriart (1989), Moore (1994c)]						
	• MF87.1: The relative affinity denatured/native gp120 is 10 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore (1994c)]						
324	MF169.1	gp160(252–261)	gp120(242–261 LAI)	RPVVSTQLLL		Env	murine(IgG)
	References: [Thiriart (1989), Moore (1994c), Moore (1994d)]						
	• MF169.1: The relative affinity denatured/native gp120 is 11 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore (1994c)]						
325	MF170.1	gp160(252–261)	gp120(242–261 LAI)	RPVVSTQLLL		Env	murine(IgG)
	References: [Thiriart (1989), Moore (1994c), Moore (1994d)]						
	• 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 (1994c)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
326	213.1	gp160(252–261)	gp120(242–261 LAI)	RPVVSTQLLL		Env glycopro	murine(IgG_1)

Donor: Claudine Bruck

References: [Thiriart (1989), Moore & Ho(1993), Moore (1994c)]

- 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 (1994c)]
- 213.1: UK Medical Research Council AIDS reagent: ARP334

327	M89	gp160(252–271)	gp120( )	RPVVSTQLLLNGSLAEEEVV	no	451 Env	murine(IgG_1)

Donor: Fulvia di Marzo Veronese

References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d)]

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

328	B12	gp160(252–271)	gp120( )	RPVVSTQLLLNGSLAEEEVV		mis-folded LAI rgp160	murine(IgG)

References: [Moore (1994c)]

- B12: C2 region – the relative affinity for denatured/native gp120 is 27 – mutations 257 T/R and 262 N/T impair binding [Moore (1994c)]

329	B13	gp160(252–271)	gp120( )	RPVVSTQLLLNGSLAEEEVV		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 (1994c), Abacioglu (1994), Moore (1994d), Pincus (1996), Connor (1998)]

- 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 (1994c)]
- B13: C2 region – epitope boundaries mapped by peptide scanning, core epitope: TQLLLN [Abacioglu (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 (1996)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
330	C13	gp160(252–271)	gp120( )	RPVVSTQLLLNGSLAEEEVV		mis-folded LAI rgp160	murine(IgG_1)
		Donor: George Lewis **References:** [Moore & Ho(1993), Moore (1994c), Abacioglu (1994)] • 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 (1994c)] • C13: Epitope boundary extended to RPVVSTQLLLNGSLAEEEVVIR, to take into account the effect of a point mutation [Abacioglu (1994)] • C13: NIH AIDS Research and Reference Reagent Program: 1209					
331	B24	gp160(257–262)	gp120( )	TQLLLN		mis-folded LAI rgp160	murine(IgG_{2a})
		References: [Abacioglu (1994)] • B24: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]					
332	B3	gp160(257–262)	gp120( )	TQLLLN		mis-folded LAI rgp160	murine(IgG_1)
		References: [Abacioglu (1994)] • B3: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]					
333	B21	gp160(257–262)	gp120( )	TQLLLN		mis-folded LAI rgp160	murine(IgG_1)
		References: [Abacioglu (1994)] • B21: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]					
334	B23	gp160(257–262)	gp120( )	TQLLLN		mis-folded LAI rgp160	murine(IgG_{2a})
		References: [Abacioglu (1994)] • B23: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]					
335	B25	gp160(257–262)	gp120( )	TQLLLN		mis-folded LAI rgp160	murine(IgG_1)
		References: [Abacioglu (1994)] • B25: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]					
336	B29	gp160(257–263)	gp120( )	TQLLLNG		mis-folded LAI rgp160	murine(IgG_{2a})
		References: [Abacioglu (1994)] • B29: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
337	B26	gp160(257–263)	gp120( )	TQLLLNG		mis-folded LAI rgp160	murine(IgG_1)
	References: [Abacioglu (1994)]						
	• B26: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]						
338	B36	gp160(257–263)	gp120( )	TQLLLNG		mis-folded LAI rgp160	murine(IgG_1)
	References: [Abacioglu (1994)]						
	• B36: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]						
339	110.E	gp160(262–281)	gp120( )	NGSLAEEEVVIRSVNFTDNA		Env glycopro	murine(IgG)
	Donor: F. Traincard						
	References: [Moore (1994c), Moore (1994d)]						
	• 110.E: The relative affinity for denatured/native gp120 is 7.3 [Moore (1994c)]						
340	110.C	gp160(271–280)	gp120( )	VIRSVNFTDN		Env glycopro	murine(IgG)
	Donor: F. Traincard, Hydridolabs, Institut Pasteur						
	References: [Moore (1994c), Moore (1994d), Valenzuela (1998)]						
	• 110.C: The relative affinity for denatured/native gp120 is 1 [Moore (1994c)]						
	• 110.C: Only slightly reduces LAI viral binding or entry into CEM cells [Valenzuela (1998)]						
341	IIIB-V3-26	gp160(291–307)	gp120( )	SVEINCTRPNNNTRKSI	no	Peptide	murine(IgG_1)
	References: [Laman (1992)]						
	• IIIB-V3-26: Binds to the base of the V3 loop on denatured gp120 [Laman (1992)]						
342	IIIB-V3-21	gp160(294–299)	gp120(299–304 IIIB)	INCTRP	no	Peptide	murine(IgG_1)
	Donor: J. Laman						
	References: [Laman (1992), Laman (1993), Valenzuela (1998)]						
	• IIIB-V3-21: Binds to the base of the V3 loop on denatured gp120 [Laman (1992)]						
	• IIIB-V3-21: Binds to NP40 treated gp120, and epitope is probably obscured by local glycosylation [Laman (1993)]						
	• IIIB-V3-21: Does not block HIV-1 LAI binding or entry into CEM cells [Valenzuela (1998)]						
	• IIIB-V3-21: UK Medical Research Council AIDS reagent: ARP3048						
	• IIIB-V3-21: NIH AIDS Research and Reference Reagent Program: 1725						

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
343	polyclonal	gp160(297–320)	gp120( )	NYNKRKRIHIGPGRAFYTTK	L	V3 peptide vaccine	human( )
	References: [Bartlett (1998)]						
	• V3 peptide vaccine (MN, RF, EV91, and Can0A) with a C4 helper T cell epitope were used to vaccinate HLA-B7 HIV-infected patients – V3 Ab levels and the anti-HIV proliferative response, but no decrease in HIV-1 RNA levels or increase in CD4 levels was observed [Bartlett (1998)]						
344	polyclonal	gp160(297–320)	gp120( )	NYNKRKRIHIGPGRAFYTTK		HIV-1 exposure	human(IgA)
	References: [Kaul (1999)]						
	• HIV-1 Env-specific mucosal IgA found in genital track of 16/21 HIV-1 resistant chronically exposed Kenyan sex workers – 11/21 had detectable Th responses [Kaul (1999)]						
345	MO97/V3	gp160(299–308)	gp120( )	PNNNTRKSIR	no	rpB1 (IIIB Env 286-467)	human(IgM)
	References: [Ohlin (1992)]						
	• MO97: Generated through *in vitro* "immunization" of uninfected-donor lymphocytes [Ohlin (1992)]						
346	8/38c	gp160(300–315)	gp120( )	NNNTRKRIRIQRGPGR	L	rBH10 gp120	rat(IgG_{2a})
	Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK						
	References: [McKeating (1992a), Sattentau & Moore(1995), Jeffs (1996), Parren (1998a), Peet (1998)]						
	• 8/38c: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating (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 (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 (1998a)]						
	• 8/38c: Called 8/38/1c: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 8/38c binding was only diminished by V3 serine substitutions C-term to the tip of the loop – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]						
	• 8/38c: UK Medical Research Council AIDS reagent: ARP3039						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
347	8/64b	gp160(300–315)	gp120( )	NNNTRKRIRIQRGPGR	L	rBH10 gp120	rat(IgM)

References: [McKeating (1992a), Peet (1998)]

- 8/64b: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating (1992a)]
- 8/64b: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 8/64b binding was abrogated by V3 serine substitutions C-term to the tip of the loop – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 8/64b: UK Medical Research Council AIDS reagent: ARP3036

348	55/1l	gp160(300–315)	gp120(300–315)	NNNTRKRIRIQRGPGR?			( )

References: [Peet (1998)]

- 55/1l: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 55/1l binding was abrogated by V3 serine substitutions in the V3 loop – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]

349	polyclonal	gp160(300–322)	gp120( )	CNNTRKSIRIQRGPGRAFVTI-GK	L	?	guinea pig(IgG)

Donor: D. Bolognesi and T. Matthews, Duke University
References: [Allaway (1993)]

- Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway (1993)]

350	polyclonal	gp160(300–328)	Env( )	NNNTRKSIRIGPGRAFYTTGD-IGNIRQ		HIV-1 infection	human( )

References: [Carlos (1999)]

- Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
351	9284	gp160(301–312)	gp120( )	NNTRKSIRIQRG	L	disrupted IIIB virion	murine(IgG_1)

Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware

References: [Skinner (1988b), Skinner (1988a), Sattentau & Moore(1991), Wyatt (1992), McKeating (1992a), Sattentau (1993), Moore (1993b), Trujillo (1993), Thali (1993), VanCott (1994), Thali (1994), Cook (1994), Okada (1994), Sorensen (1994), Sattentau & Moore(1995), VanCott (1995), Fontenot (1995), Moore & Sodroski(1996), Poignard (1996a), Cao (1997), Binley (1997a), Parren (1998a), Schonning (1998)]

- 9284: IIIB type-specific binding and neutralization [Skinner (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 (1992)]
- 9284: Increased binding in the presence of sCD4 [Sattentau (1993)]
- 9284: Inhibits C4 region antibodies (G3-299, G3-519) which have conformational requirements [Moore (1993b)]
- 9284: Peptide RIQRGPGRAFVTIGKIGNMRQA – Reacts with three human brain proteins of 35, 55, 110 kd – called NEA-9284 [Trujillo (1993)]
- 9284: Does not bind MN gp120, just IIIB [VanCott (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 (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 (1994)]
- 9284: Binding domain aa 301-310: TRKSIRIQRG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: R306T, R309T and R313G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5β – called NEA9284 [Okada (1994)]
- 9284: Did not neutralize infection of HIV/HTLV-I pseudotype [Sorensen (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 (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 (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 (1997)]
- 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 (1998a)]
- 9284: In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, 9284 was found to have an inaccessible epitope on the oligomeric form of Env and anti-V3 MAbs were found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan more efficiently than HIV-BRU [Schonning (1998)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
352	polyclonal	gp160(301–325)	gp120( )	NNTRKSIRIQRGPGRAFVTIG-KIGN	L	oral immunization – peptide plus cholera toxin adjuvant	murine(IgA)
	References: [Bukawa (1995)] • 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 (1995)]						
353	polyclonal	gp160(301–325)	gp120( )	NNTRKSIRIQRGPGRAFVTIG-KIGN	L	DNA vaccine IIIB env + rev	murine(IgA22a)
	References: [Sasaki (1998)] • An anti- env response was sought, and co-expression of Rev was required – intramuscular versus nasal vaccination with DNA vaccine with a QS-21 adjuvant was studied – QS-21 enhanced the IgG_{2a} response mediated via Th1 cytokines IFNγ and IL-2 [Sasaki (1998)]						
354	MAG 49	gp160(302–321)	gp120( )	NTRKSIRIQRGPGRAFVTIG	L	sCD4-(rHXB2 gp120)-complex	murine( )
	References: [Kang (1994), Moore & Sodroski(1996)] • 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 (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)]						
355	MAG 53	gp160(302–321)	gp120( )	NTRKSIRIQRGPGRAFVTIG	L	sCD4-(rHXB2 gp120)-complex	murine( )
	References: [Kang (1994)] • 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 (1994)]						
356	MAG 56	gp160(302–321)	gp120( )	NTRKSIRIQRGPGRAFVTIG	L	sCD4-(rHXB2 gp120)-complex	murine( )
	References: [Kang (1994)] • 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 (1994)]						
357	MAG 109	gp160(302–321)	gp120( )	NTRKSIRIQRGPGRAFVTIG	L	sCD4-(rHXB2 gp120)-complex	murine( )
	References: [Kang (1994)] • 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 (1994)]						

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
358	1324-E	gp160(303–308)	Env( )	TRTSVR	L	HIV-1 E clade infection	human($IgG_1\kappa$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 1324-E: A human MAb was derived from an HIV-1 E clade infection from a US service man who had served in Thailand, selected with the consensus V3 peptide from clade E – cross-reactive with V3 peptides, and 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, 1324E was comparable to 447-52D [Gorny (1998)]
- 1324-E: E clade stimulated MAb did not cross-react with B clade peptides nor did B clade derived peptides with an E clade V3 loop, but both E and B clade stimulated Abs can cross-react with some peptides from other clades – this Ab showed strong binding to several E, A and F peptides, one C peptide, and no reactivity with B peptides and most D peptides [Zolla-Pazner (1999a)]
- 1324-E: MAb reacted with peptides from E clade, while B clade derived MAbs could not [Zolla-Pazner (1999b)]
- 1324-E: Called 1324E – A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 1324E showed poor cross-reactivity, and was the only MAb tested that was derived from a non-B clade infected patient, an E clade infection was the source of 1324E [Nyambi (2000)]

359	polyclonal	gp160(303–319)	gp120( )	CKRKIHIGPGQAFYT		Peptide-ISCOM	murine(IgG_{2a},IgG_{2b})

References: [Ahluwalia (1997)]

- 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 IgG_{2a}/IgG_{2b} antibody response was enhanced by the presentation in the ISCOM suggestive of a Th1 response, Ahluwalia97

360	MO99/V3	gp160(304–308)	gp120( )	RKSIR	no	rpB1 (IIIB Env 286-467)	human(IgM)

References: [Ohlin (1992)]

- MO99: Generated through *in vitro* "immunization" of uninfected-donor lymphocytes [Ohlin (1992)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
361	C311E	gp160(304–313)	gp120( )	RKRIHIGP	L	IIIB infection	chimpanzee(IgG_1)

References: [Warrier (1996), Alsmadi & Tilley(1998)]

- C311E: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier (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)]

362	924	gp160(304–314)	gp120( )	RKSIRIQRGPG		vaccinia-gp160 IIIB	murine($IgG_1\kappa$)

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

- 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 (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 (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 (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 (1996)]

363	907	gp160(304–314)	gp120( )	RKSIRIQRGPG	L	vaccinia-gp160 IIIB	murine($IgG_1\kappa$)

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

- 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 (1989)]
- 907: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIB strain-specific [Pincus (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 (1996)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
364	polyclonal	gp160(304–318)	gp120( )	RKSIRIQRGPGRAFV		?	human(IgG,IgM)
	References: [Chin (1995)]						
	• Mimicking the humoral immune response *in vitro* supports isotype switching – human IgG MAbs were generated from naive donors [Chin (1995)]						
365	polyclonal	gp160(304–318)	gp120( )	RKSIRIQRGPGRAFV		Peptide	human(IgG,IgM)
	References: [Andersson (1997)]						
	• 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 (1997)]						
366	polyclonal	gp160(304–320)	gp120( )	RKRIHIGPGRAFYTT	L (MN ALA-1)	HIV-1 infection	human( )
	References: [Spear (1994)]						
	• 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 (1994)]						
367	10F10	gp160(304–320)	gp120( )	RKRIHIGPGRAFYTT	L	Peptide	murine(IgG_1)
	References: [Duarte (1994)]						
	• 2C4: Putative epitope lies within IHIGPGRAFYT – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAFYTT) peptides, lower affinity for SF2 [Duarte (1994)]						
368	2C4	gp160(304–320)	gp120( )	RKRIHIGPGRAFYTT	L (MN)	Peptide	murine(IgG_{2a})
	References: [Duarte (1994)]						
	• 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 (1994)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
369	412-D	gp160(304–320)	gp120( )	RKRIHIGPGRAFYTT	L	HIV-1 infection	human(IgG$_1\kappa$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1993), Spear (1993), VanCott (1994), Fontenot (1995), Gorny (1998), Nyambi (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 412-D: Neutralizes MN, does not bind SF2 or HXB2 – not reactive with hexa or heptapeptides by Pepscan [Gorny (1993)]
- 412-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear (1993)]
- 412-D: Called 412-10D – relatively rapid dissociation and weak homologous neutralization [VanCott (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 (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 (1998)]
- 412-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 412-D was bound only to B clade virions and to D clade MAL [Nyambi (1998)]
- 412-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 412-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 391.5, 412 and 418, all selected with MN V3 peptide – the core amino acids HIGPGR tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 412-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 412-D showed limited reactivity [Nyambi (2000)]

370	CGP 47 439	gp160(304–322)	gp120( )		L	IIIB gp120	human(Ig)

References: [Liou (1989), Safrit (1993), Gunthard (1994), Gauduin (1998), Jacobson(1998)]

- CGP 47 439: passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus – CGP 47 439 is a BAT123-human Ig chimera [Safrit (1993)]
- CGP 47 439: Phase I/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 (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 (1998)]
- CGP 47 439: Review of passive immunotherapy, summarizing [Gunthard (1994)] in relation to other studies [Jacobson(1998)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
371	178.1	gp160(305–309)	gp120( )	KSiRI	L	yeast rgp160 IIIB	murine(IgG_{2a})

Donor: C. Thiriart, Smith Kline and MRC AIDS reagent project

References: [Thiriart (1989), Back (1993), Moore & Ho(1993), Cook (1994)]

- 178.1: Reacts to gp120 and gp160 in RIPA EIA and immunoblot [Thiriart (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 (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 (1994)]
- 178.1: UK Medical Research Council AIDS reagent: ARP331

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
372	257-D	gp160(305–309)	gp120( )	KRIHI	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1991), D'Souza (1991), Karwowska (1992b), Gorny (1993), Cavacini (1993a), Spear (1993), D'Souza (1994), VanCott (1994), D'Souza (1995), Zolla-Pazner (1995), Schutten (1995a), Schutten (1995b), Fontenot (1995), Wisnewski (1996), Schutten (1996), Schutten (1997), Stamatatos (1997), Hill (1997), LaCasse (1998), Yang (1998), Gorny (1998), Stamatatos & Cheng-Mayer(1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Beddows (1999), Oggioni (1999), Nyambi (2000), Park (2000)]

- 257-D: Called 257-2-D-IV – potent neutralizing MAb –D'Souza91
- 257-D: Reacts with MN, NY5, CDC4 and SF2, does not cross-react with RF, WM52, or HXB2 [Karwowska (1992b)]
- 257-D: Neutralizes MN – binds SF2: KSIYI – specificity: MN, SF2, NY5, RF. [Gorny (1993)]
- 257-D: Additive MN or SF2 neutralization when combined with CD4 binding site MAb F105 – does not neutralize RF [Cavacini (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 (1993)]
- 257-D: Included a multi-lab study for antibody characterization and assay comparison – best NAb against MN, but not IIIB –D'Souza94
- 257-D: Potent MN neutralization, slow dissociation constant [VanCott (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'Souza95
- 257-D: In serotyping study using flow-cytometry, bound only to virus with KRIHI [Zolla-Pazner (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 (1995a)]
- 257-D: Comparable affinity for SI and NSI viruses, in contrast to MAb MN215 [Schutten (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 (1996)]
- 257-D: IIIB neutralizing MAbs *in vitro* fail to neutralize in a mouse model it *in vivo* [Schutten (1996)]
- 257-D: Neutralized (>90%) an SI-env chimeric virus and enhanced (>200%) an NSI-env chimeric virus [Schutten (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 (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 (1997)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
257-D cont.						

- 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 (1998)]
- 257-D: A neutralization assay was developed based on hemi-nested 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 (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 (1998)]
- 257-D: Called 257D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V3 MAbs 391-95D or 257D [Stamatatos & Cheng-Mayer(1998)]
- 257-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 257-D: MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 257-D: rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – 257-D bound rgp120 W61D but could only neutralize the W61D isolate following T-cell line adaptation [Beddows (1999)]
- 257-D: Study of a live-vector mucosal vaccine that expresses HIV-1 V3 domains using the bacterium Streptococcus gordonii which can express heterologous Ag and can colonize the oral cavity and vagina of mice – 268-D and 257-D recognized S. gordonii expressing the V3 domain of MN – the vaccine stimulated V3-specific IgG_{2a} in mice [Oggioni (1999)]
- 257-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 257-D showed intermediate reactivity [Nyambi (2000)]
- 257-D: Called 257D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100-fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- 257-D: UK Medical Research Council AIDS reagent: ARP3023
- 257-D: NIH AIDS Research and Reference Reagent Program: 1510

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
373	41148D	gp160(305–313)	gp120( )	KRIHIGP	L	HIV-1 infection	human(IgG_1)

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

- 41148D: Neutralizes less potently than 4117C, reacts with MN, IIIB, SF2 [Pinter (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 $10 times$ less efficient at neutralization, showing ADCC and neutralization don't always correlate [Alsmadi & Tilley(1998)]

374	311-11-D	gp160(305–313)	gp120( )	KRIHIGP	L	HIV-1 infection	human($IgG_1 \lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1991), Gorny (1993), Spear (1993), Gorny (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 311-11-D: Neutralizes MN – binds SF2: KSIYIGP [Gorny (1993)]
- 311-11-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear (1993)]
- 311-11-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 311-11-D: MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 311-11-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 311-11D showed weak reactivity [Nyambi (2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
375	391/95-D	gp160(305–318)	gp120( )	KRIHIGPGRAFY	L	HIV-1 infection	human($IgG_1\kappa$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1991), Gorny (1993), Fontenot (1995), Seligman (1996), Stamatatos (1997), Stamatatos & Cheng-Mayer(1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Ly & Stamatatos(2000), Park (2000)]

- 391/95-D: Neutralizes MN – binds to SF2, not IIIB [Gorny (1993)]
- 391/95-D: Competition ELISAs with serial deletions estimated the epitope to be KRIHIGPGRAFY – unconstrained peptide had higher affinity than cyclic [Seligman (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 (1997)]
- 391/95-D: Called 391-95D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V3 MAbs 391-95D or 257D [Stamatatos & Cheng-Mayer(1998)]
- 391/95-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 391/95-D: Called 391.5 – MAb peptide-reactivity pattern clustered with immunological related MAbs: 391.5, 412 and 418, all selected with MN V3 peptide – the core amino acids HIGPGR tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 391/95-D: Called 391-95D – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG_1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391-95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]
- 391/95-D: Called 391/95D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100-fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]

376	Aw	gp160(305–320)	gp120( )	KSITIGPGRAFHAI	L	V3 peptide	rat( )

References: [McKnight (1995)]

- 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 wildtype and v strains [McKnight (1995)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
377	Bw	gp160(305–320)	gp120( )	KSITIGPGRAFHAI	L	V3 peptide	rat( )
	References: [McKnight (1995)]						
	• 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 wildtype strain, does not bind to variant [McKnight (1995)]						
378	Dv	gp160(305–320)	gp120( )	KSITIGSGRAFHAI	L	V3 peptide	rat( )
	References: [McKnight (1995)]						
	• 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 (1995)]						
379	Fv	gp160(305–320)	gp120( )	KSITIGSGRAFHAI	L	V3 peptide	rat( )
	References: [McKnight (1995)]						
	• 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 (1995)]						
380	Gv	gp160(305–320)	gp120( )	KSITIGSGRAFHAI	L	V3 peptide	rat( )
	References: [McKnight (1995)]						
	• 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 (1995)]						
381	Hv	gp160(305–320)	gp120( )	KSITIGSGRAFHAI	L	V3 peptide	rat( )
	References: [McKnight (1995)]						
	• 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 (1995)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
382	DO142-10	gp160(305–320)	gp120( )	KRIHIGPGRAFYTT	L	HIV-1 infection	human Fab(IgG_1)

References: [Seligman (1996), Ditzel (1997), Parren (1997b), Parren & Burton(1997), Parren (1998a), Sullivan (1998a)]

- DO142-10: Fab fragment – competition ELISAs with serial deletions defined the epitope KRIHIGPGRAFYTT [Seligman (1996)]
- 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 (1997)]
- DO142-10: Neutralizes TCLA strains but not primary isolates [Parren (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 (1998a)]
- DO124-10: The HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – Fab Ab fragment DO124-10 also enhances YU2 entry, ruling out Fc interactions or Env cross-linking as a mechanism – while DO124-10 enhances YU2 entry 6-fold, it neutralizes HXBc2 under identical conditions [Sullivan (1998a)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
383	50.1	gp160(306–310)	gp120( )	RIHIG	L	V3 MN peptide	murine($IgG_1\kappa$)

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

References: [D'Souza (1991), White-Scharf (1993), Potts (1993), Ghiara (1993), Rini (1993), Bou-Habib (1994), VanCott (1994), Robert-Guroff (1994), Moore (1994b), VanCott (1995), Fontenot (1995), Seligman (1996), Berman (1997), LaCasse (1998), Stanfield (1999), Park (2000)]

- 50.1: Called R/V3-50.1 – potent neutralizing of lab strains–D'Souza91
- 50.1: Epitope defined by peptide reactivity and changes affinity with amino acid substitutions – epitope RIHIGP [White-Scharf (1993)]
- 50.1: No synergistic neutralization of MN when combined with CD4BS MAb F105 – isotype stated to be IgG_{2a} [Potts (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 (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 (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 (1994)]
- 50.1: Potent MN neutralization, slow dissociation rate [VanCott (1994)]
- 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FACS signal, Ab affinity, and viral neutralization [Robert-Guroff (1994)]
- 50.1: Shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore (1994b)]
- 50.1: Used to monitor HIV-1 Env expression in infected H9 cells [VanCott (1995)]
- 50.1: Competition ELISAs with serial deletions produced comparable estimate of epitope length to crystal structure and alanine substitution – KRIHIGP [Seligman (1996)]
- 50.1: Binds to 6/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (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 (1998)]
- 50.1: The crystal structure of V3 loop peptides bound to Fabs was obtained – conformational changes in the tip of the V3 loop (GPGR) were observed when different Fabs were bound [Stanfield (1999)]
- 50.1: Called R/V3-50.1 – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100-fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes – 50.1 could only neutralize the sensitive form [Park (2000)]
- 50.1: NIH AIDS Research and Reference Reagent Program: 1289

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
384	polyclonal	gp160(306–318)	gp120( )	KKGIAIGPGRTLY			(IgM)
	References: [Metlas (1999a), Metlas (1999b)]						
	• Auto-Abs that react with the V3 loop of NY5 are present in the sera of HIV- individuals, and are predominantly IgM [Metlas (1999a)]						
385	BAT123	gp160(306–322)	gp120( )	RIRIQRGPGRAFVTIGK	L	Inact IIIB	murine($IgG_1\kappa$)
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY						
	References: [Fung (1987), Liou (1989), Fung (1990), Moore & Ho(1993), Safrit (1993), Thali (1993), Pirofski (1993), Gauduin (1995), Sattentau & Moore(1995), Poignard (1996a), Andrus (1998), Parren (1998a), Gauduin (1998)]						
	• BAT123: 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 (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 (1993)]						
	• BAT123: Variable region sequenced – heavy chain: V 3660-SB32, D unknown, J H3 – light chain: Vκ21, Jκ2 [Pirofski (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 (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 (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 (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 (1998a)]						
	• 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 IgG2 MAb might perform better [Gauduin (1998)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
386	838-D	gp160(307–311)	Env( )	KSITK	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1997), Nyambi (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Gorny (2000), Nyambi (2000)]

- 838-D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 838-D was cross-reactive with V3 peptides from clade A and C, and could bind to 5/8 B clade V3 peptides – 50% neutralization of RF was obtained [Gorny (1997)]
- 838-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 838-D bound B clade virions but had limited cross-reactivity with other clades, with low levels of binding to A and D virions [Nyambi (1998)]
- 838-D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to many A, B, C and F peptides, poor binding to D and E [Zolla-Pazner (1999a)]
- 838-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 838-D: Binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V3 MAbs 447-52D, 838-D, and 1334 bound with a 7–10-fold preference for the oligomer [Gorny (2000)]
- 838-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 838-D showed intermediate reactivity [Nyambi (2000)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
387	782-D	gp160(307–312)	Env( )	KSITKG	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 782-D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 782-D was not cross-reactive with V3 peptides from clade A or E, but could bind to 3/8 B clade V3 peptides, and 1/2 C clade V3 peptides – 50% neutralization of RF was obtained [Gorny (1997)]
- 782-D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to several B and F peptides, one C peptide, and some reactivity with A and D peptides [Zolla-Pazner (1999a)]
- 782-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 782-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 782-D showed intermediate reactivity [Nyambi (2000)]

388	1006-15D	gp160(307–312)	gp120( )	KSITKG	no	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 1006-15D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – was somewhat cross-reactive with V3 peptides from clade A, C and other B clade V3 peptides, but not E clade [Gorny (1997)]
- 1006-15D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to several B and F peptides, one C peptide, and some reactivity with A peptides – no binding was observed with D and E peptides [Zolla-Pazner (1999a)]
- 1006-15D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 1006-15D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 1006-15D showed strong cross-reactivity [Nyambi (2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
389	908-D	gp160(307–312)	gp120( )	KSITKG	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 908-D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 908-D was not cross-reactive with V3 peptides from clade E, but could bind to 6/8 B clade V3 peptides, 2/4 A clade, and 1/2 C clade – 50% neutralization of RF was obtained [Gorny (1997)]
- 908-D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to several A, B, C and F peptides, and poor binding to E and D peptides [Zolla-Pazner (1999a)]
- 908-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 908-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross -reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 908-D showed strong cross-reactivity, but achieved only 50% neutralization on 2/5 isolates tested [Nyambi (2000)]

390	1027-15D	gp160(307–313)	Env( )	KSITKGP	no	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 1027-15D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 1027-15D was not cross-reactive with V3 peptides from clade A or E, but could bind to 3/8 B clade V3 peptides, and 1/2 C clade V3 peptides [Gorny (1997)]
- 1027-15D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed moderate binding to several B and F peptides, one C peptide, and was not reactivity with A, D and E peptides [Zolla-Pazner (1999a)]
- 1027-15D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 1027-15D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 1027-15D showed strong cross-reactivity [Nyambi (2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
391	F19.48-3	gp160(307–319)	gp120( )	IRIQRGPGRAFVT	L	IIIB rgp120 294-474	murine($IgG_{2a}\kappa$)
	References: [Boudet (1994)]						
	• F19.48-3: Strain specific – used to raise anti-idiotype antibodies [Boudet (1994)]						
392	F19.26-4	gp160(307–319)	gp120( )	IRIQRGPGRAFVT	L	IIIB rgp120 294-474	murine($IgG_{2a}\kappa$)
	References: [Boudet (1994)]						
	• F19.26-4: Strain specific – used to raise anti-idiotype antibodies [Boudet (1994)]						
393	F19.57-11	gp160(307–319)	gp120( )	IRIQRGPGRAFVT	L (LAI)	IIIB rgp120 294-474	murine($IgG_1\kappa$)
	References: [Boudet (1991), Boudet (1994), Boudet (1995)]						
	• F19.57-11: MAb F19.57-11 is strain specific for LAI – used to raise anti-idiotype rabbit antibodies (called 57-B Ab2) [Boudet (1994)]						
	• F19.57-11: Anti-anti-idiotypic antibodies (Ab3) were raised in BALBc 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 (1995)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
394	M77	gp160(307–320)	gp120( )	IRIQRGPGRAFVTI	L	HIV-1 infection	human(IgG)

Donor: Advanced BioScience Laboratories, Rockville, MD, commercial

References: [Pal (1992), di Marzo Veronese (1992), di Marzo Veronese (1993), Watkins (1993), Cook (1994), Devico (1995), Denisova (1995), Denisova (1995), Watkins (1996), Denisova (2000)]

- M77: IIIB-specific MAb, immunoprecipitates deglycosylated form [di Marzo Veronese (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 (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 (1994)]
- M77: Reacted with both reduced and non-reduced covalently cross-linked gp120-CD4 complex [Devico (1995)]
- M77: Conformational rearrangements upon binding of M77 to gp120 generates novel epitopes called metatopes [Denisova (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 (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 (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 (1996)]
- M77: M77 is highly strain specific for IIIB, but anti-idiotypic Abs directed against M77 can in turn elicit an Ab response with expanded HIV cross-reactivity – this mechanism may serve to prolong the primary response and to counter-balance viral immune evasion by mutation [Denisova (2000)]

395	SP.BAL114	gp160(308–317)	gp120( )	SIHIGPGRAF	L	?	murine?($IgG_{2a}\kappa$)

References: [Arendrup (1995)]

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

396	SP.SF2:104	gp160(308–317)	gp120( )	SIYIGPGRAF	L	HIV-1 infection	($IgG_{2a}\kappa$)

References: [Arendrup (1993), Arendrup (1995)]

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

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
397	polyclonal	gp160(308–319)	gp120( )	RIHIGPGRAFYT		HIV-1 infection	human(IgG,IgM)

References: [Langedijk (1995)]

- 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 (1995)]

398	19b	gp160(308–320)	gp120( )	-I—-G–FY-T	L	HIV-1 infection	human(IgG_1)

Donor: James Robinson, University of Connecticut, Storrs

References: [Scott Jr (1990), Moore (1994b), Moore (1994a), Sattentau(1995), Moore (1995b), Moore (1995a), Moore & Ho(1995), Gauduin (1996), Wu (1996), Trkola (1996a), D'Souza (1997), Binley (1997a), Fouts (1997), Ugolini (1997), Boots (1997), Parren (1997b), Mondor (1998), Parren (1998a), Trkola (1998), Binley (1999), Park (2000)]

- 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 (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 (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 (1995)]
- 19b: Binds to some gp120s from clades A,B,C,E, and F – weakly neutralized some B and one C clade virus [Moore (1995b)]
- 19b: Despite broad gp120 binding reactivity, not broadly neutralizing [Moore (1995a)]
- 19b: Review: more broadly cross-reactive than anti-V3 tip MAb 447-D [Moore & Ho(1995)]
- 19b: Not as effective as IgG_1b12 at neutralization *ex vivo* of virus direct from plasma of HIV-1 infected individuals [Gauduin (1996)]
- 19b: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of 19b blocks this inhibition [Wu (1996)]
- 19b: Inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)]
- 19b: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – there were four sequences with variations in the defined epitope among the 9 isolates tested –D'Souza97
- 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 (1997)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
19b cont.						

- 19b: Viral binding inhibition by 19b was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (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 (1997)]
- 19b: Neutralizes TCLA strains but not primary isolates [Parren (1997b)]
- 19b: Used as a control in this Hx10 binding and neutralizing MAb study because 19b does not bind to Hx10 [Mondor (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 (1998a)]
- 19b: No detectable neutralizing activity among primary isolates with different co-receptor usage – some neutralization of TCLA strains [Trkola (1998)]
- 19b: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG_1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519 – nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes [Binley (1999)]
- 19b: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100-fold more efficient at neutralizing the sensitive form but 19b was an exception and required around 950 ng/ml to neutralize either form [Park (2000)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
399	G3-523	gp160(308–322)	gp120( )	RIQRGPGRAFVTIGK		?	murine( )
	References: [Matsushita (1988), Jagodzinski (1996)] • 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 (1996)]						
400	4G10	gp160(308–322)	gp120( )	RIQRGPGRAFVTGK		V3-loop HBcAg hybrid	murine( )
	Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany **References:** [von Brunn (1993)] • 4G10: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn (1993)] • 4G10: NIH AIDS Research and Reference Reagent Program: 2534						
401	5F7	gp160(308–322)	gp120( )	RIQRGPGRAFVTGK		V3-loop HBcAg hybrid	murine( )
	Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany **References:** [von Brunn (1993)] • 5F7: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn (1993)] • 5F7: NIH AIDS Research and Reference Reagent Program: 2533						
402	MN215	gp160(308–322)	gp120( )	RIHIGPGRAFYTTKN	L	HIV-1 infection	human(IgG_1)
	References: [Schutten (1995b)] • 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 (1995b)]						
403	Nea 9301	gp160(308–323)	gp120( )	RIQRGPGRAFVTIGKI			murine( )
	Donor: Dupont, commercial **References:** [Wagner (1996)]						
404	4117C	gp160(309–315)	gp120( )	IXIGPGR	L	HIV-1 infection	human($IgG_1\lambda$)
	References: [Tilley (1991b), Tilley (1992), di Marzo Veronese (1993), Pinter (1993a), Pinter (1993b), Alsmadi & Tilley(1998)] • 4117C: Potent neutralizing activity against MN, SF-2, and NY-5 – synergy with CD4BS MAb 1125H [Tilley (1991b)] • 4117C: Neutralizes SF2 and MN synergistically combined with anti-CD4 binding site discontinuous MAb [Pinter (1993a), Tilley (1992)] • 4117C: Binds V3 loop – does not immunoprecipitate soluble gp120, does react with gp120 on intact virions [Pinter (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)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
405	453-D	gp160(309–315)	gp120( )	IHIGPGR	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1991), Gorny (1993), VanCott (1994), Fontenot (1995), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 453-D: Neutralizes MN – binds SF2: IYIGPGR – specificity: MN, SF2, NY5, RF [Gorny (1993)]
- 453-D: Moderate homologous neutralization, moderately slow dissociation rate [VanCott (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 (1995)]
- 453-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 453-D : MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group – MAb 268, with a previously defined core epitope identical to 453 (HIGPGR), was not part of this reactivity group, illustrating that context can be critical [Zolla-Pazner (1999b)]
- 453-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 453-D showed intermediate reactivity [Nyambi (2000)]

406	504-D	gp160(309–315)	gp120( )	IHIGPGR	L	HIV-1 infection	human($IgG_1\kappa$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1993), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 504-D – Neutralizes MN – binds SF2: IYIGPGR [Gorny (1993)]
- 504-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 504-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 504-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 504-D showed weak reactivity [Nyambi (2000)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
407	419-D	gp160(309–315)	gp120( )	IHIGPGR	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Karwowska (1992b), Gorny (1993), Spear (1993), Fontenot (1995), Nyambi (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 419-D: MN, NY5 and SF2 strain specific, does not cross-react with RF, CDC4, WM52 or HXB2 [Karwowska (1992b)]
- 419-D: Neutralizes MN – binds SF2: IYIGPGR [Gorny (1993)]
- 419-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear (1993)]
- 419-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 419-D bound to 3/4 B clade virions, and to D clade MAL [Nyambi (1998)]
- 419-D: Review of clade specificity and anti-V3 HIV-1-Abs – epitope is described as KRIHIGP [Zolla-Pazner (1999a)]
- 419-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 419-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 419-D showed intermediate reactivity, and no neutralization when tested against five strains – discrepancy between the epitope as described in earlier papers and as described here, KRIHIGP [Nyambi (2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
408	83.1	gp160(309–315)	gp120( )	IYIGPGR	L	cyclic V3 MN peptide	murine(IgG_1)

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

References: [White-Scharf (1993), Potts (1993), Jelonek (1999), Keller & Arora(1999), Binley (1999)]

- 83.1: Neutralizes SF2 [White-Scharf (1993)]
- 83.1: 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 (1993)]
- 83.1: Maternally transferred anti-V3 loop MAb selectively inhibits the anti-V3 loop Ab component of the IgG response to rgp120 SF2 in 21 day old BALBc mice [Jelonek (1999)]
- 83.1: 19 day old mice injected with 83.1 have a shift in IgG_1 response away from the V3 loop upon vaccination, without decreasing the total IgG anti-gp120 response, suggesting that prior treatment with a MAb can mask immunogenic sites and shift the immune response to vaccination [Keller & Arora(1999)]
- 83.1: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG_1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519 – nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes [Binley (1999)]

409	5023B	gp160(309–316)	gp120( )	IQRGPGra	no	15 mer synthetic BH10 V3 peptide	murine(IgG)

References: [Langedijk (1991)]

- 5023B: Generation and fine mapping of murine MAbs [Langedijk (1991)]

410	F58/D1	gp160(309–316)	gp120( )	IxxGPGRA	L	virus derived gp120	murine(IgG_1)

References: [Akerblom (1990), Broliden (1991), Moore (1993b), Millar (1998), Jackson (1999)]

- 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 (1993b)]
- F58/D1: The interaction of a 17-amino-acid neutralizing microantibody (MicroAB) based on F58 and HIV-1 env was studied by electrospray ionization mass spectrometry [Millar (1998)]
- F58/D1: A 17 amino acid MicroAB was made from the third complementarity-determining region of the heavy chain of MAb – F58 neutralized 5x's more efficiently in terms of mass than the original MAb, 32-fold less on a molar basis – neutralization does not involve initial attachment, but fusion and events in early infection [Jackson (1999)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
411	P1/D12	gp160(309–316)	gp120( )	IxxGPGRA	L	virus derived IIIB gp120	murine(IgG)

References: [Akerblom (1990), Moore (1993b)]

- 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 (1993b)]

412	P4/D10	gp160(309–316)	gp120( )	IxxGPGRA	L	virus derived IIIB gp120	murine($IgG_1\kappa$)

References: [Akerblom (1990), Broliden (1990), Broliden (1991), Marks (1992), Moore (1993b), Arendrup (1993), Hinkula (1994), Jacobson(1998), Schonning (1998), Schonning (1999)]

- P4/D10: Neutralizing and ADCC activity [Broliden (1990)]
- P4/D10: Variable domain sequenced and is identical to F58/H3 [Marks (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 (1993b)]
- P4/D10: Primary isolates from different time points from one individual were not susceptible to neutralization by P4/D10 [Arendrup (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 (1994)]
- P4/D10: Review of passive immunotherapy, summarizing [Hinkula (1994)] in relation to other studies [Jacobson(1998)]
- P4/D10: Called P4D10 – In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, anti-V3 MAbs were found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan more efficiently than HIV-BRU – Ab binding site was suggested to be 314–323 of BRU [Schonning (1998)]
- P4/D10: Called P4D10 – the stoichiometry of MAb neutralization was tested and the data indicated that binding for neutralization was was incremental not all or none, i.e., each envelope oligomer binds a single MAb and each Env oligomer bound reduces the chances of infection – MAb BC1071 was used for virion quantitation – P4D10 binds only to Env with a glycosylation site mutation at the base of the V3 loop A308T [Schonning (1999)]

413	IIIB-34 V3	gp160(309–317)	gp120( )	IQRGPGRAF	L	Peptide	murine(IgG_1)

References: [Laman (1992), Laman (1993)]

- IIIB-34 V3: Neutralizes IIIB but not MN – QXGPG are critical amino acids for binding by Pepscan analysis [Laman (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 (1993)]
- IIIB-34 V3: UK Medical Research Council AIDS reagent: ARP3047

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
414	IIIB-13 V3	gp160(309–317)	gp120( )	IQRGPGRAF	L	Peptide	murine(IgG_1)

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

- 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 (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'Souza94
- 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 (1993)]
- IIIB-13 V3: UK Medical Research Council AIDS reagent: ARP3046
- IIIB-13 V3: NIH AIDS Research and Reference Reagent Program: 1727

415	A47/B1	gp160(309–318)	gp120( )	IQRGPGRAFV	L	IIIB gp120	murine(IgG)

References: [Akerblom (1990)]

416	G44/H7	gp160(309–318)	gp120( )	IQRGPGRAFV	L	IIIB gp120	murine(IgG)

References: [Akerblom (1990)]

417	D59/A2	gp160(309–318)	gp120( )	IQRGPGRAFV	L	IIIB gp120	murine(IgG)

References: [Akerblom (1990)]

418	mu5.5	gp160(309–319)	gp120( )	IHIGPGRAFYT	P L		murine($IgG_1\kappa$)

References: [Maeda (1992), Okamoto (1998)]

- mu5.5: sCD4 causes loss of IIIB type-specificity for MAb 0.5β, allowing binding and neutralization of MN, in contrast to MAb μ5.5 [Maeda (1992)]
- mu5.5: Rmu5.5 is a humanized antibody of mouse MAb m5.5 – neutralized primary isolates with similar V3 loops – passive transfer of MAb to SCID-hu or hu-PBL-SCID mice conferred protection [Okamoto (1998)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
419	loop 2	gp160(309–320)	gp120( )	SISGPGRAFYTG	L	HIV-1 infection	human Fab( )

Donor: D. Burton, Scripps Research Institute, La Jolla, CA

References: [Barbas III (1993), Moore (1994b), Wu (1996), Ditzel (1997), Ugolini (1997), Parren (1997b), Parren & Burton(1997), Mondor (1998), Parren (1998a), Sullivan (1998a)]

- loop2: Also known as Loop 2, IgG_1 Loop 2 was a obtained by engineering Fab loop2 into an IgG_1 molecule
- loop 2: Sequences of the heavy and light chain Fab variable regions were generated [Barbas III (1993)]
- loop 2: Called Loop 2 – shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore (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 (1996)]
- loop 2: Binds to gp120 from MN and SF2 but not LAI [Ditzel (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 (1997)]
- loop 2: Epitope is suggested to be GPGRAF – binds to 10/17 US clade B monomeric gp120s – IgG_1 form can neutralize MN and 2 primary isolates tested [Parren & Burton(1997)]
- loop 2: Neutralizes TCLA strains but not primary isolates [Parren (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 IgG_1 loop 2 is only 2-fold greater than monovalent Fab loop 2, suggesting the IgG_1 form may bind with only one arm [Parren (1998a)]
- loop 2: The HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – loop 2 enhances YU2 at concentrations up to 20 ug/ml [Sullivan (1998a)]

420	5042A	gp160(310–315)	gp120( )	QrGPGR	L	15 mer synthetic BH10 V3 peptide	murine(IgG)

References: [Langedijk (1991), Gorny (1991)]

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

421	5042B	gp160(310–315)	gp120( )	QRGPGr	no	15 mer synthetic BH10 V3 peptide	murine(IgG)

References: [Langedijk (1991)]

- 5042B: Generation and fine mapping of murine MAbs [Langedijk (1991)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
422	268-D	gp160(310–315)	gp120( )	HIGPGR	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1991), D'Souza (1991), Karwowska (1992b), Gorny (1993), Spear (1993), VanCott (1994), Zolla-Pazner (1995), Fontenot (1995), McKeating (1996), Wisnewski (1996), Stamatatos (1997), LaCasse (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Beddows (1999), Oggioni (1999), Laisney & Strosberg(1999), Hioe (2000), Nyambi (2000), Park (2000)]

- 268-D: Called 268-11-D-IV – strain specific weakly neutralizing –D'Souza91
- 268-D: Reacts with MN, NY5, CDC4, RF and SF2, does not cross-react with WM52 or HXB2 [Karwowska (1992b)]
- 268-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny (1993)]
- 268-D: Mediated deposition of complement component C3 on HIV infected cells, but not in the presence of sCD4 [Spear (1993)]
- 268-D: Moderate dissociation rate and homologous neutralization titer [VanCott (1994)]
- 268-D: Serotyping study using flow-cytometry, if H of HIGPGR was substituted in virus, 268-D did not bind [Zolla-Pazner (1995)]
- 268-D: Failed to neutralize HXB2 and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (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 (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 (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 (1998)]
- 268-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 268-D: Peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group – MAb 453, with an identical core epitope to 268 based on prior experiments (HIGPGR), was not part of this reactivity group, illustrating that context can be critical [Zolla-Pazner (1999b)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
268-D cont.			• 268-D: rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – 268-D bound rgp120 W61D but could only neutralize the W61D isolate following T-cell line adaptation [Beddows (1999)] • 268-D: Called 268-11D – Study of a live-vector mucosal vaccine that expresses HIV-1 V3 domains using the bacterium Streptococcus gordonii which can express heterologous Ag and can colonize the oral cavity and vagina of mice – 268-D and 257-D recognized S. gordonii expressing the V3 domain of MN – the vaccine stimulated V3-specific IgG_{2a} in mice [Oggioni (1999)] • 268-D: Called MAb 268 – To identify potential mimotopes of V3, a hexapeptide phage library was screened with MAb 268 – two hexamers were identified, HLGPGR or KAIHRI that bind to 268 with the same binding site as the V3 loop and inhibit 268 MN gp120 – KLH conjugated hexamer KAIHRI stimulates Abs in rabbits that cross-react with ML gp120 [Laisney & Strosberg(1999)] • 268-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inihibited proliferative responses of gp120 specific T cells – V3 MAbs 447-52-D and 268-10-D did not effect proliferation [Hioe (2000)] • 268-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 268-D showed weak reactivity [Nyambi (2000)] • 268-D: Called 268D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100-fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)] • 268-D: UK Medical Research Council AIDS reagent: ARP3024 • 268-D: NIH AIDS Research and Reference Reagent Program: 1511			

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
423	386-D	gp160(310–315)	gp120( )	HIGPGR	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Karwowska (1992b), Gorny (1993), VanCott (1994), Fontenot (1995), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 386-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny (1993)]
- 386-D: Slow dissociation rate, potent homologous neutralization [VanCott (1994)]
- 386-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 386-D: Peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 386-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 386-D showed intermediate reactivity [Nyambi (2000)]

424	5025B	gp160(310–316)	gp120( )	QRGPGra	no	15 mer synthetic BH10 V3 peptide	murine(IgG)

References: [Langedijk (1991)]

- 5025B: Generation and fine mapping of murine MAbs [Langedijk (1991)]

425	418-D	gp160(310–316)	gp120( )	HIGPGRA	L	HIV-1 infection	human($IgG_1\kappa$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Karwowska (1992b), Gorny (1993), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 418-D: MN strain specific, does not cross-react with SF2, NY5, RF, CDC4 WM52 or HXB2 [Karwowska (1992b)]
- 418-D: Neutralizes MN, does not bind to SF2 or HXB2 [Gorny (1993)]
- 418-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 418-D: Called 418 – MAb peptide-reactivity pattern clustered with immunological related MAbs: 391.5, 412 and 418, all selected with MN V3 peptide – the core amino acids HIGPGR tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 418-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 418-D showed intermediate reactivity [Nyambi (2000)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
426	5021	gp160(310–316)	gp120( )	QrGPGRa	L	15 mer BH10 V3 peptide	murine(IgG)
		References: [Durda (1988), Durda (1990), Langedijk (1991), Moore (1993b)] • 5021: Generation and fine mapping of murine MAbs [Langedijk (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 (1993b)]					
427	5042	gp160(310–316)	gp120( )	QRGPGRA	L	Peptide	murine( )
		References: [Durda (1988), Durda (1990), Moore (1993b)] • 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 (1993b)]					
428	110.3	gp160(310–317)	gp120( )	QRGPGRAF	L	BRU infected cell lysates	murine($IgG_1\kappa$)
		References: [Kinney Thomas (1988), Evans (1989), Langedijk (1992), Pirofski (1993), Connelly (1994)] • 110.3: Included as a control [Evans (1989)] • 110.3: MAb variable region sequenced – heavy chain: V 7138(40), D deletion, J H4 – light chain: Vκ21(47), Jκ2 [Pirofski (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 (1994)]					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
429	110.4	gp160(310–317)	gp120( )	QRGPGRAF	L	BRU infected cell lysates	murine(IgG$_1\kappa$)

Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas
References: [Kinney Thomas (1988), Thali (1992b), Langedijk (1992), Thali (1993), Pirofski (1993), Arendrup (1993), Thali (1994), Boudet (1994), Connelly (1994), McDougal (1996), Valenzuela (1998), Cao (1997), Guillerm (1998)]

- 110.4: 313 P/S substitution in the V3 region disrupts binding [Thali (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: Vκ21, Jκ2 [Pirofski (1993)]
- 110.4: Primary isolates from different time points from one individual were not susceptible to neutralization by 110.4 [Arendrup (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 (1994)]
- 110.4: An anti-idiotypic MAb generated against 110.3 also blocks binding of 110.4 [Connelly (1994)]
- 110.4: Neutralizes HIV-1 LAI [McDougal (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 (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 (1997)]
- 110.4: Used for flow cytometry in a study of the anti-CD4, CDR3 loop MAb called 13B8.2, in a study of HIV-1 induced programmed cell death [Guillerm (1998)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
430	110.5	gp160(310–317)	gp120( )	QRGPGRAF	L	BRU infected cell lysates	murine(IgG$_1\kappa$)

Donor: E. Kinney-Thomas or Genetic Systems, Seattle WA
References: [Kinney Thomas (1988), Moore (1990), Cordell (1991), Sattentau & Moore(1991), Langedijk (1992), McKeating (1992a), Pirofski (1993), Moore (1993b), Thali (1993), Klasse (1993a), Sattentau (1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), McDougal (1996), Jeffs (1996), Binley (1997a), Ugolini (1997), Parren (1998a)]

- 110.5: Did not induce dissociation of gp120, as sCD4 did – discrepancy with [Poignard (1996a)], that was suggested to be due to MAb interference with detection, as the gp120-MAb complex was denatured in the Poignard study [Moore (1990)]
- 110.5: Binding insensitive to gp120 reduction [Cordell (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: Vκ21, Jκ2 [Pirofski (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 (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 (1988), Klasse (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 (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 (1996a)]
- 110.5: Neutralizes HIV-1 LAI [McDougal (1996)]
- 110.5: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs (1996)]
- 110.5: Viral binding inhibition by 110.5 was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (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 (1998a)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
431	58.2	gp160(310–317)	gp120( )	HIGPGRAF	L	MN V3 peptide	murine($IgG_1\kappa$)

References: [White-Scharf (1993), Potts (1993), Moore (1994b), Seligman (1996), Stanfield (1999)]

- 58.2: Epitope defined by peptide reactivity and changes in affinity with amino acid substitutions – 4/7 primarily isolates were neutralized [White-Scharf (1993)]
- 58.2: Did not synergistically neutralize MN in combination with MAb F105 – there was synergistic neutralization when combined with sCD4 [Potts (1993)]
- 58.2: Modest cross-reactivity among B clade gp120s, little outside B clade – core epitope as I-IHIG [Moore (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 (1996)]
- 58.2: The crystal structure of Fab 58.2 bound to V3 loop peptides was obtained – conformational changes in the tip of the V3 loop (GPGR) were observed when different MAbs were bound – 58.2's epitope was defined as KRKRIHIGPGRAFY [Stanfield (1999)]

432	537-D	gp160(311–315)	gp120( )	IGPGR	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Karwowska (1992b), Gorny (1992), Gorny (1993), VanCott (1994), Fontenot (1995), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 537-D: Reacts with MN, NY5, CDC4, RF, WM52 and SF2, but does not cross-react with HXB2 [Karwowska (1992b)]
- 537-D: MN type specific neutralization observed – binds SF2, also IGPGR [Gorny (1992), Gorny (1993)]
- 537-D: Moderate homologous neutralization, relatively rapid dissociation constant [VanCott (1994)]
- 537-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 537-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 537-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 537-D showed weak reactivity [Nyambi (2000)]

433	5020	gp160(311–316)	gp120( )	RGPGRA	no	15 mer synthetic BH10 V3 peptide	murine(IgG)

References: [Langedijk (1991)]

- 5020: Generation and fine mapping of murine MAbs [Langedijk (1991)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
434	5023A	gp160(311–317)	gp120( )	RgPGRAF	L	15 mer synthetic BH10 V3 peptide	murine(IgG)

Donor: Paul Durda, Du Pont de Nemours and Co

References: [Langedijk (1991), D'Souza (1991), Back (1993), Rovinski (1995), Schonning (1998)]

- 5023A: Generation and Fine mapping of murine MAbs [Langedijk (1991)]
- 5023A: Called 5023 – Langedijk also has an MAb called 5023B – strong cross-reactive neutralizing MAb –D'Souza91
- 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 (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 (1995)]
- 5023A: Called NEA-9205 – The N306 glycan of the V3 loop makes the tip of the V3 loop inaccessible to this MAb in oligomeric Env, loss of this glycan enhances neutralization sensitivity [Schonning (1998)]

435	110.6	gp160(311–318)	gp120( )	RGPGRAFV	L (weak)	BRU infected cell lysates	murine($IgG_1\lambda$)

References: [Kinney Thomas (1988), Pirofski (1993), Langedijk (1992)]

- 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 (1993)]

436	polyclonal	gp160(311–318)	gp120( )	IGPGRAFY	L	gp120-B. abortus com-plex (SF2 or MN)	murine(IgG_{2a})

References: [Golding (1995)]

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

437	10/54	gp160(311–321)	gp120( )	RGPGRAFVTIG	L (HXB10)	rgp120 BH10	rat(IgG_1)

References: [McKeating (1992a), McKeating (1993a), McKeating (1993b), Peet (1998)]

- 10/54: Binding to virion gp120 enhanced by sCD4 [McKeating (1992a)]
- 10/54: Studied in the context of a neutralization escape mutant [McKeating (1993a)]
- 10/54: Called 10/54ow/6i/6i: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, but anti-V3 MAb 10/54 binding was dramatically diminished by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
438	10/36e	gp160(311–321)	gp120( )	RGPGRAFVTIG	L (HXB10)	rgp120 BH10	rat(IgG_{2a})
	References: [McKeating (1992a), McKeating (1993b), Peet (1998)] • 10/36e: Binding to virion gp120 enhanced by sCD4 [McKeating (1992a)] • 10/36e: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, but anti-V3 MAb 10/36e binding was dramatically diminished by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]					
439	11/85b	gp160(311–321)	gp120( )	RGPGRAFVTIG	L (HXB2)	rgp120 BH10	rat(IgG_{2b})
	References: [McKeating (1992a), McKeating (1993b)] • 11/85b: Binding to virion gp120 enhanced by sCD4 [McKeating (1992a)]						
440	polyclonal	gp160(311–322)	gp120( )	IGPGRAFYTTKN	L (MN ALA-1)	IGPGRAFYTTKN HRV14:HIV-1 chimera	guinea pig( )
	References: [Smith (1998)] • 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 (1998)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
441	0.5β	gp160(311–324)	gp120( )	RGPGRAFVTIGKIG	L (IIIB)	IIIB Env	murine(IgG$_1\kappa$)

Donor: Shuzo Matsushita or Toshio Hattori of Kumamoto University

References: [Matsushita (1988), Skinner (1988b), Skinner (1988a), Reitz (1988), Nara (1990), D'Souza (1991), Matsushita (1992), Emini (1992), Maeda (1992), McKeating (1992a), Sperlagh (1993), di Marzo Veronese (1993), Moore (1993b), Klasse (1993a), Watkins (1993), Cook (1994), Thali (1994), Okada (1994), Boudet (1994), Broder (1994), Zvi (1995b), Zvi (1995a), Jagodzinski (1996), Warrier (1996), McDougal (1996), Jeffs (1996), Huang (1997), Zvi (1997), Wyatt (1997), Faiman & Horovitz(1997), Fortin (2000), Jagodzinski & Trzeciak(2000), Tugarinov (2000), Zvi (2000)]

- 0.5β: Type-specific neutralization of IIIB – does not neutralize MN or RF [Matsushita (1988), Skinner (1988b)]
- 0.5β: Emergence of virus resistant to MAb 0.5β and autologous sera neutralization in IIIB infected chimps [Nara (1990)]
- 0.5β: Potent neutralizing activity –D'Souza91
- 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 (1992)]
- 0.5β: sCD4 causes loss of IIIB type-specificity, allowing binding and neutralization of MN, in contrast to MAb mu5.5 [Maeda (1992)]
- 0.5β: Monoclonal anti-idiotype antibodies that mimic the 0.5β epitope were generated [Sperlagh (1993)]
- 0.5β: Neutralization of virus carrying an A to T substitution (contrast with MAb M77) [di Marzo Veronese (1993)]
- 0.5β: Binding to native gp120 100–300-fold greater than to denatured [Moore (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 (1988), Klasse (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 (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 (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 (1994)]
- 0.5β: Binding domain aa 310-319: RGPGRAFVTIGKIG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: R306T, R309T and R313G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5β [Okada (1994)]
- 0.5β: Type-specific neutralization of IIIB – does not neutralize SF2 [Broder (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 (1995b)]
- 0.5β: NMR of 0.5β bound NNTRKSIRIQRGPGRAFVTIGKIG suggests that the bound amino acids are in the region SIRIQRGP-GRAFVT [Zvi (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 (1996)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)

0.5β cont.

- 0.5β: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier (1996)]
- 0.5β: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs (1996)]
- 0.5β: Relative to the native peptide, an O-linked α-galactosamine modified V3 peptide enhanced binding to 0.5β, while an N-linked β-glucosamine modified peptide showed reduced binding [Huang (1997)]
- 0.5β: The structure of a 17 amino acid V3 peptide bound to the Fab was studied using NMR [Zvi (1997)]
- 0.5β: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- 0.5β: The Fv fragment was purified and the temperature dependence and effect of mutations was studied [Faiman & Horovitz(1997)]
- 0.5β: Host encoded intercellular adhesion molecule (ICAM-1) is incorporated by the HIV-1 virion and enhances viral infectivity – ICAM-1 does not modify virus sensitivity to antibodies 0.5β or 4.8D or sCD4, but neutralizing ability of F105 was diminished in ICAM bearing virions in the presence of lymphocyte function-association antigen-1 (LFA-1) Ab [Fortin (2000)]
- 0.5β: MAbs 0.5β and G3-42 were used to study synthesis of oligomeric and monomeric forms of Env – inhibition of glycosylation by tunicamycin results in reduction of oligomeric gp120 at the cell surface and of monomer in the cytoplasm – neither MAb recognized non-glycosylated Env precursor [Jagodzinski & Trzeciak(2000)]
- 0.5β: 14/18 residues of peptide P1053, RKSIRIQRGPGRAFVTIG, were shown to be involved in the Ab recognition site using NMR – QRGPGR forms a β-hairpin turn at the center of the binding pocket [Tugarinov (2000)]
- 0.5β: NMR and mutation cycles were employed to generate a model of the peptide-antibody complex, showing aa residues that interact or do not contribute to the binding of MAb 0.5β Fv with the peptide – F96(L) of 0.5β binds to Pro13, H52(H) interacts with Ile7, Ile9, Gln10, and D56(H) interacts with Arg11 of the V3 loop peptide – RGPG retains hairpin conformation binds in the center of a groove [Zvi (2000)]
- 0.5β: UK Medical Research Council AIDS reagent: ARP3025
- 0.5β: NIH AIDS Research and Reference Reagent Program: 1591

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
442	Cβ1	gp160(311–324)	gp120( )	RGPGRAFVTIGKIG	L	IIIB Env	human(IgG_1)
	References: [Emini (1992)]						
	• Cβ1: passive transfer to chimpanzees confers protection against challenge with homologous cell-free virus – mouse 0.5β human IgG_1 chimera [Emini (1992)]						
443	NM-01	gp160(312–315)	gp120( )	GPGR	L	IIIB MN	murine(IgG)
	Donor: M. Terada						
	References: [Ohno (1991), Yoshida (1997), Smith (1998)]						
	• NM-01: Resistance mutation selected by propagation of molecular cloned isolate in the presence of NM-01 [Yoshida (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 (1998)]						
444	1026	gp160(312–317)	gp120( )	GPGRAF	L	rgp120 MN	murine(IgG)
	References: [Nakamura (1993), Bou-Habib (1994)]						
	• 1026: Bound diverse strains, neutralizing activity against MN, close to GPGRAF [Nakamura (1993)]						
	• 1026: Greater affinity for T cell-tropic strain T-CSF, derived from JR-CSF, than to the primary isolate JR-CSF [Bou-Habib (1994)]						
445	1034	gp160(312–317)	gp120( )	GPGRAF	L	rgp120 MN	murine(IgG)
	References: [Bou-Habib (1994), Berman (1997)]						
	• 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 (1994)]						
	• 1034: Binds to 5/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
446	59.1	gp160(312–317)	gp120(308–313 MN)	GPGRAF	L	cyclic V3 MN peptide	murine(IgG_1)

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

References: [D'Souza (1991), White-Scharf (1993), Potts (1993), Ghiara (1993), Bou-Habib (1994), D'Souza (1994), Seligman (1996), Ghiara (1997), Smith (1998), Stanfield (1999)]

- 59.1: Called R/V3-59.1 – potent neutralizing MAb –D'Souza91
- 59.1: Epitope defined by peptide reactivity and binding affinity with amino acid substitutions – GPGRAF [White-Scharf (1993)]
- 59.1: Synergistic neutralization of MN when combined with sCD4 or the CD4BS MAb F105 [Potts (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 (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 (1994)]
- 59.1: Multi-lab study for antibody characterization and assay comparison – neutralizes MN and IIIB –D'Souza94
- 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 (1996)]
- 59.1: A conformationally restricted analog of the tip of the V3 loop was constructed and bound with Fab 59.1 – crystal structure 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 (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 (1998)]
- 59.1: The crystal structure of V3 loop peptides bound to Fabs was obtained – conformational changes in the tip of the V3 loop (GPGR) were observed when different MAbs were bound [Stanfield (1999)]

447	polyclonal	gp160(312–317)	gp120(316–321)	GPGRAF		multiple-epitope vaccine CG-GPGRAFY-G-ELDKWA-G-RILAVERYLKD conjugated to BSA	rabbit and murine(Ig)

References: [Lu (2000b), Lu (2000a)]

- High titer response to ELDKWA and RILAVERYLKD was observed, weak response to GPGRAFY – immunization with CG-$(ELDKWA\text{-}GPGRAFY)_2$-K was also tried, yielding a strong Ab response to ELDKWA, weak to GPGRAF – gp160 vaccination yielded strong Ab response but not to any of the peptides studied here [Lu (2000b), Lu (2000a)]

448	polyclonal	gp160(312–318)	gp120(317–323)	GPGRAFY		peptide coupled to BSA, C-$(GPGRAF)_4$-BSA or C-(TRPNNNTRKSIRIQRGPGRAFYTIGKI)-BSA	murine and rabbit( )

References: [Yu (2000)]

- High levels of epitope-specific Abs were induced by the peptide-BSA conjugates but not by rgp160 vaccine [Yu (2000)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
449	N11-20	gp160(312–320)	gp120( )	GPGRAFVTI	L (LAI)	unk	murine($IgG_1\kappa$)
	Donor: J. C. Mazie, Hybridolab, Institut Pasteur **References:** [Valenzuela (1998)] • 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 (1998)]						
450	5025A	gp160(313–317)	gp120( )	pgRAF	L	15 mer synthetic BH10 V3 peptide	murine(IgG)
	Donor: Paul Durda, Du Pont de Nemours and Co **References:** [Langedijk (1991), D'Souza (1991)] • 5025A: Generation and fine mapping of murine MAbs [Langedijk (1991)] • 5025: Called 5025 – strain specific weakly neutralizing –D'Souza91						
451	N70-1.9b	gp160(313–318)	gp120( )	PGRAFY	L	HIV-1 infection	human(IgG_1)
	References: [Robinson (1990a), Scott Jr (1990)] • N70-1.9b: Type specificity [Robinson (1990a)] • N70-1.9b: Type specific neutralization, ADCC directed against MN infected cells [Scott Jr (1990)]						
452	902	gp160(313–324)	gp120( )	PGRAFVTIGKIG	L	vaccinia-gp160 IIIB	murine($IgG_1\kappa$)
	Donor: Bruce Chesebro, Rocky Mountain National Laboratory, Montana **References:** [Chesebro & Wehrly(1988), Laman (1993), Broder (1994), Earl (1994)] • 902: Strain specific neutralization of HIV [Chesebro & Wehrly(1988)] • 902: Epitope may be partially masked or altered in the oligomeric molecule [Broder (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 (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 (1997)] • 902: NIH AIDS Research and Reference Reagent Program: 522						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
453	694/98-D	gp160(314–317)	gp120(V3 IIIB)	GRAF	L	HIV-1 infection	human($IgG_1\lambda$)

Donor: Drs. S. Zolla-Pazner and M. Gorny, NYU Med Center NY, NY

References: [Gorny (1991), Gorny (1992), Gorny (1993), Cavacini (1993a), Spear (1993), Gorny (1994), Laal (1994), VanCott (1994), Cook (1994), VanCott (1995), Zolla-Pazner (1995), Forthal (1995), Li (1997), Zolla-Pazner (1997), Smith (1998), Li (1998), Andrus (1998), Nyambi (1998), Schonning (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Altmeyer (1999), Nyambi (2000), Park (2000)]

- 694/98-D: MAb first described [Skinner (1988b)]
- 694/98-D: Type-specific lab isolate neutralization was observed – binds with 1–3-fold greater affinity to gp120 than to peptides [Gorny (1992)]
- 694/98-D: Neutralizes MN and IIIB (GRAF) – binds SF2 (GRAF) – binding reactivity: MN, IIIB, SF2, NY5, RF, CDC4, WM52 [Gorny (1993)]
- 694/98-D: Called 694-D – complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear (1993)]
- 694/98-D: 50% neutralization of HIV-IIIB at a concentration of 0.15μg/ml [Gorny (1994)]
- 694/98-D: Potent neutralization of IIIB – no neutralization synergy in combination with CD4 binding domain MAbs [Laal (1994)]
- 694/98-D: GRVY did not alter peptide binding – GRVI and GQAW enhanced dissociation – GQVF and GQAL did not bind [VanCott (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 (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 (1995)]
- 694/98-D: Serotyping study using flow-cytometry – bound GRAX bearing virus in 10/11 cases – somewhat conformation dependent [Zolla-Pazner (1995)]
- 694/98-D: ADCC activity, and no viral enhancing activity [Forthal (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 (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 – 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 (1998)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
694/98-D cont.						

- 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 (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 (1998)]
- 694/98-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 694/98-D bound only to B and D clade virions and had limited cross reactivity [Nyambi (1998)]
- 694/98-D: In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, anti-V3 MAbs were found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan more efficiently than HIV-BRU [Schonning (1998)]
- 694/98-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 694/98-D: MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 694/98-D: A Semliki Forest virus (SFV) expression system carrying BX08 env was used to study the conformation of gp120 – intracytoplasmic gp120 was recognized by the anti-V3 MAbs K24 and F5.5, while gp120 at the plasma membrane was detected only by conformation dependent MAbs 2G12, 670-D and 694/98D and not linear V3 MAbs – expression in rat brain also showed that surface-expressed Env was recognized only by the conformation-dependent antibodies and not by anti-V3 antibodies [Altmeyer (1999)]
- 694/98-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 694/98-D showed intermediate reactivity [Nyambi (2000)]
- 694/98-D: Called 694/98D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100-fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
454	9205	gp160(315–317)	gp120( )	RAF (core reactivity)	L	IIIB V3 Peptide	murine(IgG_1)

Donor: NEN, Boston MA, commercial

References: [Durda (1990), Trujillo (1993), Allaway (1993), VanCott (1994), Fontenot (1995), Schonning (1999)]

- 9205: Called NEA-9205, epitope RIQRGPGRAFVTIGK – reacts with three human brain proteins of 35, 55, 110 kd molecular weight – similar to 9284 –RAF is the core reactivity [Trujillo (1993)]
- 9205: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway (1993)]
- 9205: Neutralizes IIIB but not MN – significantly slower dissociation constant for IIIB than MN [VanCott (1994)]
- 9205: Called NEA-9205 – the stoichiometry of MAb neutralization was tested and the data indicated that binding for neutralization was was incremental not all or none, i.e., each envelope oligomer binds a single MAb and each Env oligomer bound reduces the chances of infection – 9205 binds only to Env with a glycosylation site mutation in the V3 loop, A308T [Schonning (1999)]

455	110.I	gp160(316–322)	gp120( )	AFVTIGK	L	recombinant gp120	murine( )

Donor: F. Traincard, Pasteur Institute, France

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

- 110.I: Binds to carboxy-terminal side of the V3 loop – inhibits binding of C4 region MAb G3-299 [Moore (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 (1996a)]
- 110.I: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (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 (1998a)]

456	anti-HIV-2 polyclonal	gp160(317–320)	gp120(315–318 and 329–331 SBL6669,HIV-2)	FHSQ...WCR		HIV-2 V3 region peptides	guinea pig(IgG)

References: [Morner (1999)]

- Neutralizing Abs against HIV-2 V3 are produced when peptides spanning two non-contiguous parts of the V3 loop are used for vaccination including amino acids 315–318 near the tip (FHSQ) and 329–331 (WCR) at the C-term Cys [Morner (1999)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
457	IIIB-V3-01	gp160(320–328)	gp120( )	IGKIGNMRQ	no	IIIB carboxy-terminus V3-loop peptide	murine(IgG_1)
		Donor: Jon Laman **References:** [Laman (1993)] • IIIB-V3-01: Specific for carboxy-terminal flank of the IIIB V3 loop – epitope is hidden native gp120, exposed on denaturation [Laman (1993)] • IIIB-V3-01: UK Medical Research Council AIDS reagent: ARP3046 • IIIB-V3-01: NIH AIDS Research and Reference Reagent Program: 1726					
458	4D7/4	gp160(360–380)	gp120( )	IFKQSSGGDPEIVTHSFNCGG		Env glycopro	murine(IgG)
		Donor: S. Ranjbar, NIBSC, UK **References:** [Moore (1994c)] • 4D7/4: C3 region – the relative affinity for denatured/native gp120 is >10 [Moore (1994c)] • 4D7/4: UK Medical Research Council AIDS reagent: ARP3051					
459	36.1(ARP 329)	gp160(361–381)	gp120( )	FKQSSGGDPEIVTHSFNCGGE		Env glycopro	murine(IgG)
		References: [Thiriart (1989), Moore (1994c)] • 36.1: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P impair binding [Moore (1994c)] • 36.1: UK Medical Research Council AIDS reagent: ARP329					
460	C12	gp160(361–381)	gp120( )	FKQSSGGDPEIVTHSFNCGGE		mis-folded LAI rgp160	murine(IgG_1)
		Donor: George Lewis **References:** [Moore & Ho(1993), Moore (1994c), Abacioglu (1994), Moore (1994d)] • 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 (1994c)] • C12: C3 region – epitope boundaries mapped by peptide scanning, core FNCGG [Abacioglu (1994)]					
461	110.D	gp160(380–393)	gp120( )	GEFFYCNSTQLFNS	no	Env glycopro	murine(IgG)
		Donor: F. Traincard, Pasteur Institute, France **References:** [Moore (1994c), Valenzuela (1998)] • 110.D: The relative affinity for denatured/native gp120 is >50 [Moore (1994c)]					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
462	B32	gp160(380–393)	gp120(380–393 LAI)	GEFFYCNSTQLFNS		mis-folded LAI rgp160	murine(IgG_1)
	References: [Moore (1994c), Abacioglu (1994)]						
	• 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 (1994c)]						
	• B32: C3 region – epitope boundaries mapped by peptide scanning – FFY(core) [Abacioglu (1994)]						
463	B15	gp160(395–400)	gp120( )	WFNSTW		mis-folded LAI rgp160	murine(IgG_{2b})
	Donor: George Lewis						
	References: [Moore & Ho(1993), Moore (1993b), Abacioglu (1994)]						
	• 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 (1993b)]						
	• B15: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)]						
464	B34	gp160(395–400)	gp120( )	WFNSTW		mis-folded LAI rgp160	murine(IgG_{2b})
	References: [Abacioglu (1994)]						
	• B34: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)]						
465	polyclonal	gp160(396–418)	Env( )	FNSTWFNSTWSTEGSNNTEGS-DT		HIV-1 infection	human( )
	References: [Carlos (1999)]						
	• Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]						
466	5C2E5	gp160(422–431)	gp120( )	QFINMWQEVK		purified gp120	murine( )
	Donor: T. Gregory and R. Ward, Genentech, San Francisco						
	References: [Lasky (1987), Cordell (1991)]						
	• 5C2E5: Blocks the gp120-CD4 interaction [Lasky (1987)]						
	• 5C2E5: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell (1991)]						

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
467	G3-211	gp160(423–437)	gp120( )	IINMWQKVGKAMYAP	L	virus derived IIIB gp120	murine(IgG_1)
	References: [Sun (1989)] • 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 (1989)]						
468	G3-537	gp160(423–437)	gp120( )	IINMWQKVGKAMYAP	L	virus derived IIIB gp120	murine(IgG_1)
	References: [Sun (1989), Ho (1991b), McKeating (1992b)] • 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 (1989)] • G3-537: Weakly neutralizing – binds to a linear binding domain of gp120, NMWQEVGKAMYAPPISG [McKeating (1992b)]						
469	polyclonal	gp160(425–436)	gp120( )	NMWQEVGKAMYA	L	oral immunization – peptide plus cholera toxin adjuvant	murine(IgA)
	References: [Bukawa (1995)] • 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 (1995)]						
470	1795	gp160(425–441)	gp120(425–441 IIIB)	NMWQEVGKAMYAPPISG	L	poliovirus env chimera	( )
	References: [McKeating (1992b)] • 1795: CD4 binding site – weakly neutralizing – binding inhibited by WQEVGKAMYA, GKAM may be involved [McKeating (1992b)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
471	G3-42	gp160(429–438)	gp120( )	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

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

References: [Sun (1989), Moore (1993b), Thali (1993), Sattentau & Moore(1995), Jagodzinski (1996), Moore & Sodroski(1996), Poignard (1996a), Trkola (1996a), Binley (1997a), Binley (1999), Jagodzinski & Trzeciak(2000)]

- G3-42: Neutralization of IIIB but not RF [Sun (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 (1993b)]
- G3-42: Inhibits binding of CD4 inducible MAb 48d [Thali (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 (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 (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 (1996a)]
- G3-42: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG_1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519 – nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes [Binley (1999)]
- G3-42: MAbs 0.5β and G3-42 were used to study synthesis of oligomeric and monomeric forms of Env – inhibition of glycosylation by tunicamycin results in reduction of oligomeric gp120 at the cell surface and of monomer in the cytoplasm – neither MAb recognized non-glycosylated Env precursor [Jagodzinski & Trzeciak(2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
472	G3-299	gp160(429–438)	gp120( )	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

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

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

- G3-299: Best neutralization of IIIB in panel of 7 MAbs that bind overlapping epitope [Sun (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 (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 (1996a)]
- G3-299: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (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 (1998a)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
473	G3-508	gp160(429–438)	gp120( )	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

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

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

- G3-508: Neutralization of IIIB and RF [Sun (1989)]
- G3-508: Inhibits binding of CD4 inducible MAb 48d [Thali (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 (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 (1996a)]
- G3-508: Called G3 508 – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)]
- 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 (1998a)]
- 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 (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
474	G3-519	gp160(429–438)	gp120( )	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Sun (1989), Moore & Ho(1993), Moore (1993b), D'Souza (1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Wyatt (1997), Parren (1998a), Binley (1999)]

- G3-519: Best neutralization of RF in panel of 7 MAbs that bind overlapping epitope [Sun (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 (1993b)]
- G3-519: Included in a multi-lab study for antibody characterization, and binding and neutralization assay comparison, also binds IIIB: IINMWQKVGKAMYAPP –D'Souza94
- 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 (1996a)]
- G3-519: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (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 (1998a)]
- G3-519: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG_1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519 – nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes [Binley (1999)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
475	G3-536	gp160(429–438)	gp120( )	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG_1)

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

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

- G3-536: Weak neutralization of IIIB and RF – cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – epitope:IINMWQKVGKAMYAP [Sun (1989)]
- G3-536: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell (1991)]
- G3-536: Weakly neutralizing – binds to a linear determinant in the CD4 binding domain of gp120 [McKeating (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 (1993b)]
- G3-536: Enhances binding of anti-V2 MAb 697-D [Gorny (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 (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 (1998a)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
476	ICR38.1a	gp160(429–438)	gp120(427–436 BRU)	EVGKAMYAPP	L	rBH10 gp120	rat(IgG_{2b})

References: [Cordell (1991), McKeating (1992b), McKeating (1992a), McKeating (1992c), McKeating (1993b), McKeating (1993a), Moore (1993b), Jeffs (1996), Peet (1998), Kropelin (1998)]

- ICR38.1a: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with MAbs G3-536, 5C2E5, and ICR38.8f [McKeating (1992b), Cordell (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 (1992a)]
- ICR38.1a: Studied in the context of a neutralization escape mutant [McKeating (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 (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 (1996)]
- ICR38.1a: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – ICR38.1a was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- ICR38.1a: Called 388/389 – anti-C1 region MAb 87-135/9 blocks gp120 interaction with CD4+ cells – blocking activity is additive when combined with antibodies which bind in the C4 region of gp120 (F105, 388/389, and b12) [Kropelin (1998)]
- ICR38.1a: UK Medical Research Council AIDS reagent: ARP388/ARP389

477	ICR38.8f	gp160(429–438)	gp120( )	EVGKAMYAPP	L	rBH10 gp120	rat(IgG_{2b})

References: [Cordell (1991)]

- ICR38.8f: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with ICR38.1a, 5C2E5, and G3-536 [Cordell (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 (1993b)]

478	MO86/C3	gp160(429–443)	gp120( )	EVGKAMYAPPISGQI		rIIIB Env 286-467	human(IgM)

References: [Ohlin (1992)]

- MO86: Generated through *in vitro* "immunization'' of uninfected-donor lymphocytes [Ohlin (1992)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
479	G45-60	gp160(431–440)	gp120( )	GKAMYAPPIS	L	virus derived IIIB gp120	murine(IgG_1)

References: [Sun (1989), Moore (1993b), Gorny (1994), Moore & Sodroski(1996), Jagodzinski (1996)]

- 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 (1993b)]
- G45-60: Enhances binding of anti-V2 MAb 697-D [Gorny (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 (1996)]

480	13H8	gp160(431–440)	gp120( )	GKAMYAPPIS	L	rgp120 MN	murine(IgG)

References: [Nakamura (1992), Nakamura (1993), Jeffs (1996)]

- 13H8: Cross blocks 5C2 in IIIB-rsgp160 ELISA – reactive with diverse strains in rgp120 ELISA [Nakamura (1992)]
- 13H8: Bound diverse strains, neutralizing activity against MN [Nakamura (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 (1996)]

481	polyclonal	gp160(432–451)	gp120( )	KAMYAPPISGQIRCSSNITG	no	Vaccinia expressing Env fused with vaccinia proteins p14 and p39	murine( )

References: [Collado (2000)]

- Vaccinia p14 can elicit NAbs and p39 tends to be immunodominant, so these proteins were fused to regions of HIV-1 Env – reduced glycosylation was noted when p14 or p39 was placed in the N-term region of the fusion protein – chimeric proteins shifted the Env Ab response from V3 to either a C1 or C4 domain, depending on the construct – all chimeric Env proteins: 14kEnv, 39kEnv, and Env39k elicited a strong Ab response to the C1 region of gp120 (LFCAS-DAKAYDTEVHNVWAT), and Env39k mounted a strong response to the C4 region (KAMYAPPISGQIRCSSNITG) [Collado (2000)]

482	1662	gp160(433–439)	gp120( )	AMYAPPI	no	poliovirus-antigen chimera	( )

References: [McKeating (1992b)]

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

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
483	1663	gp160(433–439)	gp120( )	AMYAPPI	no	poliovirus-antigen chimera	( )
		References: [McKeating (1992b)]					
		• 1663: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]					
484	1664	gp160(433–439)	gp120( )	AMYAPPI	no	poliovirus-antigen chimera	( )
		References: [McKeating (1992b)]					
		• 1664: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]					
485	1697	gp160(433–439)	gp120( )	AMYAPPI	no	poliovirus-antigen chimera	( )
		References: [McKeating (1992b)]					
		• 1697: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]					
486	1794	gp160(433–442)	gp120( )	AMYAPPISGQ	no	poliovirus env chimera	( )
		References: [McKeating (1992b)]					
		• 1794: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]					
487	1804	gp160(433–442)	gp120( )	AMYAPPISGQ	no	poliovirus env chimera	( )
		References: [McKeating (1992b)]					
		• 1804: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]					
488	1807	gp160(433–442)	gp120( )	AMYAPPISGQ	no	poliovirus env chimera	( )
		References: [McKeating (1992b)]					
		• 1807: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]					
489	1808	gp160(433–442)	gp120( )	AMYAPPISGQ	no	poliovirus env chimera	( )
		References: [McKeating (1992b)]					
		• 1808: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]					
490	polyclonal	gp160(454–474)	Env( )	LTRDGGNNNNESEIFRPGGGD		HIV-1 infection	human( )
		References: [Carlos (1999)]					
		• Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
491	polyclonal	gp160(460–467)	gp120( )	NNNNGSEI		HIV-1 infection augmented by gp160 vaccine	human( )

References: [Loomis-Price (1997)]

- 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 (1997)]

492 M91 — gp160(461–470) — gp120( ) — SNNESEIFRL — no — 451 Env — rat(IgG_{2a})

Donor: Fulvia di Marzo Veronese

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

- M91: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)]
- M91: The relative affinity for denatured/native gp120 is 24 – mutation in position 470 P/L impairs binding [Moore (1994c)]
- M91: 470 P/L impairs binding, but not 475 D/V, in contrast to CRA1 – some C2 mutations can enhance binding [Moore (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 (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

493 CRA1(ARP 323) — gp160(461–470) — gp120( ) — SNNESEIFRL — no — Env glycopro — murine(IgG)

Donor: M. Page, NIBSC, UK

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

- 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 (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 (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 (1996a)]
- CRA1: UK Medical Research Council AIDS reagent: ARP323

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
494	9201	gp160(471–482)	gp120( )	GGGDMRDNWRSE	no		murine( )
	Donor: Du Pont **References:** [McDougal (1996)] • 9201: Does not neutralize LAI [McDougal (1996)]						
495	9301	gp160(471–490)	gp120( )	GGGDMRDNWRSELYKYKVVK		Env glycopro	murine(IgG)
	Donor: Dupont, commercial **References:** [Skinner (1988b), Moore & Ho(1993), Moore (1994c), Moore (1994d), Wagner (1996)] • 9301: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • 9301: The relative affinity for denatured/native gp120 is 19 [Moore (1994d)] • 9301: Wagner *et al.* claim that Nea 9301 is anti-V3 – might they have meant MAb 9305? [Wagner (1996)]						
496	1C1	gp160(471–490)	gp120( )	GGGDMRDNWRSELYKYKVVK		Env glycopro	murine(IgG)
	Donor: Repligen Inc, Cambridge, MA, commercial **References:** [Moore (1994c), Moore (1994d), VanCott (1995), Moore & Sodroski(1996)] • 1C1: The relative affinity for denatured/native gp120 is 15 [Moore (1994c)] • 1C1: C2 and V3 regions substitutions can influence binding [Moore (1994d)] • 1C1: Linear epitope not exposed on conformationally intact gp120 [VanCott (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)]						
497	B221	gp160(471–490)	gp120( )	GGGDMRDNWRSELYKYKVVK		Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	murine($IgG_1\kappa$)
	Donor: Rod Daniels **References:** [Moore & Ho(1993), Bristow (1994), Moore (1994c)] • 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 (1994)] • B221: The relative affinity for denatured/native gp120 is 12 – mutation 477 D/V impairs binding [Moore (1994c)] • B221: Called 221 – C2 and V3 substitutions influence binding [Moore (1994d)] • B221: UK Medical Research Council AIDS reagent: ARP301						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
498	660-178	gp160(471–490)	gp120( )	GGGDMRDNWRSELYKYKVVK		Env glycopro	murine(IgG)
	Donor: G. Robey, Abbott Labs **References:** [Moore (1994c), Moore (1994d)] • 660-178: The relative affinity for denatured/native gp120 is >100 [Moore (1994c)] • 660-178: DeltaV1/V2 and DeltaV1/V2/V3 reduce binding – C2 and C5 mutations enhance binding [Moore (1994d)]						
499	8C6/1	gp160(471–490)	gp120( )	GGGDMRDNWRSELYKYKVVK		Env glycopro	murine(IgG)
	Donor: S. Ranjbar, NIBSC, UK **References:** [Moore (1994c)] • 8C6/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>30-fold) – mutation 485 K/V impairs binding [Moore (1994c)] • 8C6/1: UK Medical Research Council AIDS reagent: ARP3052						
500	5F4/1	gp160(471–490)	gp120( )	GGGDMRDNWRSELYKYKVVK		Peptide	murine( )
	Donor: S. Ranjbar, NIBSC, UK **References:** [Moore (1994c)] • 5F4/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>10-fold) – mutation 485 K/V impairs binding [Moore (1994c)]						
501	3F5	gp160(471–490)	gp120( )	GGGDMRDNWRSELYKYKVVK		Env	murine(IgG)
	Donor: S. Nigida, NCI, USA **References:** [Moore (1994c)] • 3F5: The relative affinity for denatured/native gp120 is 100 [Moore (1994c)]						
502	H11	gp160(472–477)	gp120( )	GGDMRD			murine( )
	References: [Pincus & McClure(1993), Pincus (1996)] • 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 (1996)]						
503	W2	gp160(472–491)	gp120( )	GGDMRDNWRSELYKYKVVKI		Env	murine(IgG)
	Donor: D. Weiner, U. Penn., USA **References:** [Moore (1994c)] • W2: The relative affinity for denatured/native gp120 is 30 – mutation 485 K/V impairs binding [Moore (1994c)]						

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
504	1331A	gp160(483–508)	gp120(510–516)	dwVVQREKR		HIV-1 infection	human($IgG_3\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Nyambi (1998), Gorny (2000), Hochleitner (2000b), Nyambi (2000)]

- 1331A: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-C5 Abs 670-D and 1331A bound to 3/4 B clade viruses (they don't bind to IIIB), and to subtype D MAL [Nyambi (1998)]
- 1331A: Core epitope dwVVQREKR maps to gp120(510-516) – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – C5 MAbs 858-D, 989-D and 1331A bound with a 5–10-fold preference for the monomer[Gorny (2000)]
- 1331A: The Ab binding site was studied with epitope excision (protein is bound in native conformation to immobilized MAb, then digested with proteolytic enzymes) and extraction (protein is digested then allowed to react with Ab), followed by mass spectroscopy – two non-contiguous aa in C5 were protected, E-507 and I-487, which are thought to be located on opposite sides of hydrophobic pocket involved in gp120/gp41 interaction [Hochleitner (2000b)]
- 1331A: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 858-D bound only 4/26, the worst of all anti-C5 MAbs tested, while MAb 1331A, which shares the same core epitope (positions 495-516), bound to 18/26 [Nyambi (2000)]

505	M38	gp160(485–504)	gp120( )	KYKVVKEIPLGVAPTKAKRR	no	IIIB immunization	murine( )

References: [Beretta (1987), Grassi (1991), Lopalco (1993), DeSantis (1994), Beretta & Dalgleish(1994)]

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

506	polyclonal	gp160(489–511)	gp120(495–516)	KIEPLGVAPTKAKRRVVQREKR	no	HIV-1 infection	human( )

References: [Hernandez (2000)]

- Chimeric peptide combining two peptides gp160(495-516 and 584-612) served as a specific and broadly reactive antigen for diagnostic detection of HIV-1 [Hernandez (2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
507	42F	gp160(491–500)	gp120( )	IEPLGVAPTK	no	HIV-1 infection	human($IgG_1\lambda$)

References: [Alsmadi (1997), Alsmadi & Tilley(1998)]

- 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 (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)]

508	43F	gp160(491–500)	gp120( )	IEPLGVAPTK	no	HIV-1 infection	human($IgG_1\lambda$)

References: [Alsmadi (1997)]

- 43F: 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 (1997)]

509	RV110026	gp160(491–500)	gp120( )	IEPLGVAPTK		Peptide	human( )

Donor: Commercial, Olympus Inc

References: [Moore (1994c), Moore (1994d)]

- RV110026: Preferentially binds SDS-DTT denatured gp120 (15-fold using R1/87 as capture reagent) [Moore (1994c)]

510	110.1	gp160(491–500)	gp120( )	IEPLGVAPTK	no	BRU infected cell lysates	murine($IgG_1\kappa$)

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

References: [Gosting (1987), Linsley (1988), Kinney Thomas (1988), Pincus (1991), Moore (1994c), Cook (1994), McDougal (1996), Binley (1997a), Valenzuela (1998)]

- 110.1: There is another antibody with this ID that binds to gp120, but at aa 200–217 [Pincus (1996)]
- 110.1: Referred to as 110–1 – does not inhibit CD4-gp120 binding or neutralize HIV-1 strains [Linsley (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 (1991)]
- 110.1: The relative affinity for denatured/native gp120 is 0.7 [Moore (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 (1994)]
- 110.1: Does not neutralize HIV-1 LAI [McDougal (1996)]
- 110.1: Does effect LAI viral binding or entry into CEM cells [Valenzuela (1998)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
511	105-306	gp160(492–500)	gp120(498-505 HAM112)	KPFSVAPTP		group O rec Env pGO-8PL	murine(IgG$_1\kappa$)

References: [Scheffel (1999)]

- 105-306: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity – 105–306 bound to two overlapping peptides [Scheffel (1999)]

512	GV1G2	gp160(494–499)	gp120( )	LGVAPT		gp120 complexed with MAb M77	murine( )

References: [Denisova (1996)]

- 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 (1996)]

513	670-D	gp160(498–504)	gp120(503–509)	PTKAKRR	no	HIV-1 infection	human(IgG$_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY

References: [Zolla-Pazner (1995), Forthal (1995), Hill (1997), Gorny (1997), Gorny (1998), Nyambi (1998), Altmeyer (1999), Gorny & Zolla-Pazner(2000), Nyambi (2000)]

- 670-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner (1995)]
- 670-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity, numbering provided suggests epitope is RRVVQRE [Forthal (1995)]
- 670-D: 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 (1997)]
- 670-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-C5 Abs 670-D and 1331A bound to 3/4 B clade viruses (they didn't bind to IIIB), and to subtype D MAL – 670-D also reacted with subtype A[Nyambi (1998)]
- 670-D: A Semliki Forest virus (SFV) expression system carrying BX08 env was used to study the conformation of gp120 – intracytoplasmic gp120 was recognized by the anti-V3 MAbs K24 and F5.5, while gp120 at the plasma membrane was detected only by conformation dependent MAbs 2G12, 670-D and 694/98D and not V3 MAbs – expression in rat brain also showed that surface-expressed Env was recognized only by the conformation-dependent antibodies and not by anti-V3 antibodies [Altmeyer (1999)]
- 670-D: A gp120 C5 MAb used as a negative control in a study of anti-gp41 MAbs [Gorny & Zolla-Pazner(2000)]
- 670-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 670-D bound 21/26, and was the most cross-reactive C5 MAb [Nyambi (2000)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
514	450-D	gp160(498–504)	gp120( )	PTKAKRR (or RRVVQRE, or MRDNWRSELYKY depending on reference)	no	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU Med Center, NY, NY
References: [Durda (1988), Karwowska (1992a), Karwowska (1992b), Spear (1993), Laal (1994), Gorny (1994), Cook (1994), Forthal (1995), Manca (1995), Li (1997), Hioe (2000)]

- 450-D: Bound to MN, SF-2 and IIIB, but was not neutralizing [Karwowska (1992a)]
- 450-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear (1993)]
- 450-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal (1994)]
- 450-D: Epitope is defined as PTKAKRR [Gorny (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 (1994)]
- 450-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 450-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (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 (1997)]
- 450-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inihibited proliferative responses of gp120 specific T cells – C5 MAbs 450-D and 750-D did not effect proliferation [Hioe (2000)]

515	750-D	gp160(498–504)	gp120( )	PTKAKRR	no	HIV-1 infection	human($IgG_3\lambda$)

References: [Forthal (1995), Hioe (2000)]

- 750-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 750-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inihibited proliferative responses of gp120 specific T cells – C5 MAbs 450-D and 750-D did not effect proliferation [Hioe (2000)]

516	polyclonal	gp160(503–509)	gp120(471–477)	RRVVQRE		peptide APTKAKRRVVQR-EKR	murine(IgG)

References: [Jeyarajah (1998)]

- Epitope excision and extraction combined with mass spectrometry was used to map the fine structure of epitopes recognized by polyclonal Ab to HIV-1 Env – a major epitope was identified between positions 472 and 478 [Jeyarajah (1998)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
517	722-D	gp160(503–509)	gp120( )	RRVVQRE	no	HIV-1 infection	human(IgG$_1\kappa$)
	References: [Laal (1994), Forthal (1995)]						
	• 722-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal (1994)]						
	• 722-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]						
518	polyclonal	gp160(503–511)	gp120( )	RRVVQREKR		HIV-1 infection	human( )
	References: [Palker (1987), Loomis-Price (1997)]						
	• 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 (1997)]						
519	858-D	gp160(505–511)	gp120(495–516 LAI)	VVQREKR	no	HIV-1 infection	human(IgG)
	Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)						
	References: [Zolla-Pazner (1995), Forthal (1995), Gorny (2000), Nyambi (2000)]						
	• 858-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner (1995)]						
	• 858-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]						
	• 858-D: Binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – C5 MAbs 858-D, 989-D and 1331A bound with a 5–10-fold preference for the monomer[Gorny (2000)]						
	• 858-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 858-D bound only 4/26, the worst of all anti-C5 MAbs tested, while MAb 1331A, which shares the same core epitope (positions 495-516), bound to 18/26 [Nyambi (2000)]						
520	989-D	gp160(505–511)	gp120( )	VVQREKR		HIV-1 infection	human(IgG)
	Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)						
	References: [Zolla-Pazner (1995), Gorny (2000), Nyambi (2000)]						
	• 989-D: In serotyping study using flow-cytometry, showed B clade specificity, but only reacted with 7/11 B clade virus [Zolla-Pazner (1995)]						
	• 989-D: Binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – C5 MAbs 858-D, 989-D and 1331A bound with a 5–10-fold preference for the monomer[Gorny (2000)]						
	• 989-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 989-D bound to 6/26 [Nyambi (2000)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
521	1131-A	gp160(505–511)	gp120( )	VVQREKR	no	HIV-1 infection	human($IgG_3\lambda$)
	References: [Bandres (1998)]						
	• 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 (1998)]						
522	5F3	gp160(525–543)	gp41(526–543 BH10)	AAGSTMGAASMTLTVQARQ	no	HIV-1 infection	human($IgG_1\kappa$)
	Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria						
	References: [Buchacher (1994)]						
	• 5F3: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells [Buchacher (1994)]						
523	25C2	gp160(525–543)	gp41(526–543 BH10)	AAGSTMGAASMTLTVQARQ	no	HIV-1 infection	human($IgG_1\kappa$)
	Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX						
	References: [Buchacher (1992), Buchacher (1994), Sattentau (1995)]						
	• 25C2: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells – binds oligomeric and monomeric gp41, and gp160 [Buchacher (1994)]						
	• 25C2: Called IAM 41-25C2 – Binding domain overlaps sites that are critical for gp120-gp41 association – binding is enhanced by sCD4 – binding region defined as: gp41(21-38 BH10) [Sattentau (1995)]						
524	24G3	gp160(525–543)	gp41(526–543 BH10)	AAGSTMGAASMTLTVQARQ	no	HIV-1 infection	human($IgG_1\kappa$)
	Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria						
	References: [Buchacher (1992), Buchacher (1994)]						
	• 24G3: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells [Buchacher (1994)]						
525	1A1	gp160(525–543)	gp41(526–543 BH10)	AAGSTMGAASMTLTVQARQ	no	HIV-1 infection	human($IgG_1\kappa$)
	Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria						
	References: [Buchacher (1994)]						
	• 1A1: Human MAb generated using EBV transformation of PBL from HIV-1+ volunteers [Buchacher (1994)]						
526	α(566-586)	gp160(561–581)	gp41(566–586 BRU)	AQQHLLQLTVWGIKQLQARIL		HIV-1 infection	human( )
	References: [Poumbourios (1992)]						

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
527	PC5009	gp160(572–591)	gp41(577–596 BRU)	GIKQLQARILAVERYLKDQQ		rgp160	murine( )
		References: [Poumbourios (1992)]					
		• PC5009: Recognized only monomeric gp41 [Poumbourios (1992)]					
528	polyclonal α(577-596)	gp160(572–591)	gp41(577–596 BRU)	GIKQLQARILAVERYLKDQQ		HIV-1 infection	human plasma( )
		References: [Poumbourios (1992)]					
		• α(577-596): Affinity purified from HIV-1+ plasma – preferentially bind oligomer [Poumbourios (1992)]					
529	polyclonal	gp160(576–592)	gp41(583–599)	LQARILAVERYLKDQQL		HIV-1 infection	human sera( )
		References: [Klasse (1993b)]					
		• 42 HIV-1 positive human sera were tested against wildtype peptide, and peptide with substitution 589 A to T: 11/42 reacted strongly with wildtype, weakly with A589T – 31 reacted weakly with parental, even more weakly with substituted [Klasse (1993b)]					
530	polyclonal	gp160(579–589)	gp41(586–596 IIIB)	RILAVERYLKD		peptide vaccine C(RILAVERYLKD)$_2$-BSA	mice and rabbits( )
		References: [Xiao (2000a)]					
		• Strong epitope-specific neutralizing antibody responses were induced using the peptide, but not full gp160 [Xiao (2000a)]					
531	polyclonal	gp160(579–589)	gp41(586–596)	RILAVERYLKD		multiple-epitope vaccine CG-GPGRAFY-G-ELDKWA-G-RILAVERYLKD conjugated to BSA	rabbit(Ig)
		References: [Lu (2000b), Lu (2000a)]					
		• High titer response to ELDKWA and RILAVERYLKD was observed, weak response to GPGRAFY – immunization with CG-(ELDKWA-GPGRAFY)$_2$-K was also tried, yielding a strong Ab response to both ELDKWA and GPGRAFY – gp160 vaccination yielded strong Ab response but not to any of the peptides studied here [Lu (2000b), Lu (2000a)]					
532	polyclonal	gp160(579–599)	gp41(583–604)	RILAVERYLKDQQLLGIWGCS	no	desialylated HIV-1 gp160	rabbit sera( )
		References: [Benjouad (1993)]					
		• MAbs raised against desialylated HIV-1 gp160 cross-react with HIV-2 gp140 due to immunodominant conserved epitope in gp41 [Benjouad (1993)]					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
533	2A2/26	gp160(579–601)	gp41(584–606 BRU)	RILAVERYLKDQQLLGIWGCS-GK		viral gp41( )	murine(IgG)

References: [Poumbourios (1992), Poumbourios (1995)]

- 2A2/26: Immunodominant region, binds both oligomer and monomer [Poumbourios (1992)]
- 2A2/26: Delta 550–561 (Delta LLRAIEAQQHLL), a region important for oligomer formation diminishes binding, Delta (550-561 +571-581) abrogates binding [Poumbourios (1995)]

534	50-69	gp160(579–603)	gp41(579–603 BH10)	RILAVERYLKDQQLLGIWGCS-GKLI	no	HIV-1 infection	human($IgG_{2\kappa}$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY

References: [Till (1989), Pinter (1989), Gorny (1989), Xu (1991), Robinson (1991), Sattentau & Moore(1991), Eddleston (1993), Spear (1993), Laal (1994), Chen (1995), Sattentau (1995), Manca (1995), McDougal (1996), Poignard (1996a), Binley (1996), Klasse & Sattentau(1996), Stamatatos (1997), Boots (1997), Mitchell (1998), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000)]

- 50-69: Combined with deglycosylated A chain of ricin is toxic to lines of HIV-infected T cells (H9) and monocytes (U937) [Till (1989)]
- 50-69: Reacts preferentially with gp160 oligomer, compared to gp41 monomer [Pinter (1989)]
- 50-69: Kills HIV-infected cells when coupled to deglycosylated ricin A chain [Gorny (1989)]
- 50-69: The epitope is affected by the conformation conferred by the two cysteines at amino acids 598 and 604 [Xu (1991)]
- 50-69: Enhances HIV-1 infection *in vitro* – synergizes with huMAb 120–16 *in vitro* to enhance HIV-1 infection to level approaching that found in polyclonal anti-HIV serum [Robinson (1991)]
- 50-69: Two-fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 50-69: Called SZ-50.69 – binds to an epitope within aa 579–613 [Eddleston (1993)]
- 50-69: Did not mediate deposition of complement component C3 on HIV infected cells unless cells were pre-incubated with sCD4 – complement mediated virolysis of MN and IIIB in the presence of sCD4 [Spear (1993)]
- 50-69: Epitope described as Cluster I, 601-604, conformational – does not neutralize IIIB or synergize neutralization by anti-V3 MAb 447-52D or by CD4 BS MAbs [Laal (1994)]
- 50-69: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen (1995)]
- 50-69: Preferentially binds oligomer – binding increased after pretreatment of infected cells with sCD4 – binding domain overlaps site that is critical for gp120-gp41 association [Sattentau (1995)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)

50-69 cont.

- 50-69: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 50-69: Does not neutralize HIV-1 LAI [McDougal (1996)]
- 50-69: Prebinding of anti-V3, and CD4i MAbs 48d and 17b, but not anti-V2 neutralizing MAbs, expose the 50–69 epitope [Poignard (1996a)]
- 50-69: Binds to a linear epitope located in the Cluster I region – binding of 50–69 and 240-D inhibited by Fabs A1, A4, M8B, M26B, M12B and T2 [Binley (1996)]
- 50-69: Used to test exposure of gp41 upon sCD4 binding [Klasse & Sattentau(1996)]
- 50-69: Binding of anti-gp120 MAbs IgG_1b12 or 654-30D does not mediate significant exposure of the gp41 epitopes for MAbs 2F5 and 50–69 [Stamatatos (1997)]
- 50-69: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – 50–69 maps to an immunodominant domain in gp41 – three groups of peptides were selected, one which seems most closely related to gp41 sequence peptide consensus is WGCxx(RK)(x n)LxC – the analogous gp41 sequence WGCSGKLIC is present in most M group clades, except D with a common L to H substitution [Boots (1997)]
- 50-69: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50-69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 – identifies non-contiguous W596-G597-C598 and C604-T605 as minimal epitope [Mitchell (1998)]
- 50-69: A cluster I epitope that binds to rgp41 567-647, recognizes a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41 – this MAb doesn't react with either of the peptides N51 or C43 individually – MAbs 50–69 and 1367 had similar properties – MAb 50–69 bound the fusogenic form of the protein in liquid phase [Gorny & Zolla-Pazner(2000)]
- 50-69: Binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5-fold preference for the oligomer, while other gp41 MAbs (1367, 98-6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)]
- 50-69: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 50–69 bound the majority of isolates although binding was moderate to weak – specifies discontinuous binding site range as aa 579–613 [Nyambi (2000)]
- 50-69: NIH AIDS Research and Reference Reagent Program: 531

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
535	98-43	gp160(579–604)	gp41(579–604 HXB2)	RILAVERYLKDQQLLGIWGCS-GKLIC	no	HIV-1 infection	human($IgG_2\kappa$)

References: [Pinter (1989), Gorny (1989), Tyler (1990), Xu (1991)]

- 98-43: Reacts equally well with oligomer and monomer [Pinter (1989)]
- 98-43: Poor ADCC (in contrast to MAb 120-16, gp41(644-663)) [Tyler (1990)]
- 98-43: 579–604 binds in the immunodominant region [Xu (1991)]
- 98-43: NIH AIDS Research and Reference Reagent Program: 1241

536	9-11	gp160(579–604)	gp41(584–609)	RILAVERYLKDQQLLGIWGCS-GKLIC		gp160	murine(IgG_1)

References: [Mani (1994)]

- 9-11: required the C-C disulfide bridge and loop formation, can bind simultaneously with 41–1 [Mani (1994)]

537	Fab A1	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab A1: Binds to Cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

538	Fab A4	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab A4: Binds to Cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

539	Fab M8B	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab M8B: Binds to Cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

540	Fab M26B	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab M26B: Binds to Cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
541	Fab T2	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab T2: Binds to Cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

542	Fab M12B	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab M12B: Binds to Cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

543	41.4	gp160(579–608)	gp41(584–609)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV			( )

Donor: Jan McClure, Bristol-Myers Squibb Pharmaceutical Res Inst, Seattle, WA
References: [Pincus & McClure(1993)]

- 41.4: Binds to peptide weakly, but to gp160 with higher affinity than 41.1, and cross-competes with 41.1 – probably conformational – MAb was coupled to ricin A chain (RAC) – sCD4 enhances the efficacy of immunotoxins *in vitro* 30-fold [Pincus & McClure(1993)]

544	41-1	gp160(579–608)	gp41(584–609)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV		gp160	murine($IgG_1\kappa$)

References: [Gosting (1987), Dalgleish (1988), Pincus (1991), Pincus & McClure(1993), Mani (1994), Pincus (1996), Pincus (1998)]

- 41-1: This antibody to gp41(584-609) [Mani (1994)] seems to have been named the same as a different MAb to gp41(735-752 IIIB) [Dalgleish (1988)]
- 41-1: Also called 41.1, although possibly not, the literature is confusing because two gp41 MAbs that bind to this region with similar names (dash versus period) are listed as murine and human
- 41-1: Broadly reactive [Gosting (1987)]
- 41-1: This antibody seems to have been named the same as a different MAb to gp41(735-752) [Dalgleish (1988)]
- 41-1: Efficacious as an immunotoxin when coupled to RAC – gave linear epitope as gp160 579–603 [Pincus (1991)]
- 41-1: Called 41.1, and described as a human MAb – cross-competes with 41.4 – sCD4 enhances the efficacy of immunotoxins *in vitro* 30-fold – MAb was coupled to ricin A chain (RAC) – [Pincus & McClure(1993)]
- 41-1: Did not require the C-C disulfide bridge and loop formation, can bind simultaneously with 9–11 [Mani (1994)]
- 41-1: Called 41.1, and described as a human MAb, binding 579–604 – a panel of immunotoxins was generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
545	polyclonal	gp160(580–597)	gp41(584–602)	ILAVERYLKDQQLLGIWG	no	HIV-1 infection	human sera( )
	References: [Petrov (1990)]						
	• Immunodominant and broadly reactive peptide [Petrov (1990)]						
546	polyclonal	gp160(582–589)	gp41(589–596)	AVERYLKD		HIV-1 infection	human sera( )
	References: [Klasse (1991)]						
	• Substitutions and deletions in peptide 583–599 were systematically studied – alterations in AVERYLKD abrogated the antigenicity of peptides with most of 14 human sera [Klasse (1991)]						
547	polyclonal	gp160(584–604)	gp41( )	ERYLKDQLLGIWGCSGKLIC		HIV-1 infection	human( )
	References: [Shafferman (1989)]						
	• Immunogenic domain useful for diagnostics [Shafferman (1989)]						
548	polyclonal	gp160(584–612)	gp41(587–617)	ERYLKDQQLLGIWGCSGKLIC-TTAVPWNA	no	HIV-1 infection	human( )
	References: [Hernandez (2000)]						
	• Chimeric peptide combining two peptides gp160(495-516 and 584-612) served as a specific and broadly reactive antigen for diagnostic detection of HIV-1 [Hernandez (2000)]						
549	2F11	gp160(589–600)	gp41( )	DQQLLGIWGCSG	no	HIV-1 infection	human(IgG_1)
	References: [Eaton (1994)]						
	• 2F11: Enhances infectivity even in the absence of complement – does not mediate ADCC or neutralize virus [Eaton (1994)]						

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
550	246-D	gp160(590–597)	gp41(579–604 HXB2)	qqLLGIWg	no	HIV-1 infection	human(IgG$_1\kappa$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU Med Center, NY, NY

References: [Xu (1991), Robinson (1991), Spear (1993), Eddleston (1993), Forthal (1995), Manca (1995), Saarloos (1995), Earl (1997), Gorny & Zolla-Pazner(2000), Nyambi (2000)]

- 246-D: Fine mapping indicates core is LLGI [Xu (1991)]
- 246-D: Did not mediate deposition of complement component C3 on HIV infected cells unless cells were pre-incubated with sCD4 [Spear (1993)]
- 246-D: No neutralizing activity, some enhancing activity [Robinson (1991)]
- 246-D: Called SZ-246.D [Eddleston (1993)]
- 246-D: No neutralizing activity, both ADCC and viral enhancing activity [Forthal (1995)]
- 246-D: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 246-D: 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 (1995)]
- 246-D: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50-69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 [Mitchell (1998)]
- 246-D: This antibody, along with murine MAb D61, can be blocked by any of a group of 8 conformational MAbs (M10, D41, D54, T4, T6, T9, T10 and T35) [Earl (1997)]
- 246-D: Core epitope aa 591 to 597, a cluster I epitope that does not bind to either a peptide complex that approximates the core of the fusogenic form of gp41 or the individual peptides N51 and C43 that form this structure – MAbs 181-D and 246-D had similar properties [Gorny & Zolla-Pazner(2000)]
- 246-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 246-D bound strongly or moderately to all 26 HIV-1 group M clades viruses tested and showed the strongest binding of all anti-Env MAbs tested, including the V3 and C5 region MAbs – notes core epitope as LLGI – no neutralizing activity was observed when 246-D was tested with five isolates [Nyambi (2000)]
- 246-D: NIH AIDS Research and Reference Reagent Program: 1245

551	9G5A	gp160(591–594)	gp41(596–599 IIIB)	QLLG		Anti-idiotype against M38	murine(IgM)

References: [Lopalco (1993), Beretta & Dalgleish(1994)]

- 9G5A: Anti-idiotype to gp120 C terminus (C5 region) MAb M38 [Lopalco (1993)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
552	181-D	gp160(591–597)	gp41(591–597 HXB2)	qLLGIWg	no	HIV-1 infection	human($IgG_{2\kappa}$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY
References: [Xu (1991), Robinson (1991), Eddleston (1993), Forthal (1995), Fontenot (1995), Gorny & Zolla-Pazner(2000), Nyambi (2000)]

- 181-D: Fine mapping indicates core is LLGIW [Xu (1991)]
- 181-D: No enhancing or neutralization activity [Robinson (1991)]
- 181-D: Called SZ-181.D [Eddleston (1993)]
- 181-D: No neutralizing, no ADCC, and no viral enhancing activity [Forthal (1995)]
- 181-D: Core epitope aa 591 to 597, a cluster I epitope that does not bind to either a peptide complex that approximates the core of the fusogenic form of gp41 or the individual peptides N51 and C43 that form this structure – MAbs 181-D and 246-D had similar properties [Gorny & Zolla-Pazner(2000)]
- 181-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 181-D bound the majority of isolates although binding was moderate to weak [Nyambi (2000)]

553	240-D	gp160(592–600)	gp41(592–600 HXB2)	LLGIWGCSG	no	HIV-1 infection	human( )

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY
References: [Xu (1991), Robinson (1991), Spear (1993), Binley (1996), Wisnewski (1995), Wisnewski (1996), Mitchell (1998), Nyambi (2000)]

- 240-D: Fine mapping indicates core is IWG [Xu (1991)]
- 240-D: No neutralizing activity, some enhancing activity [Robinson (1991)]
- 240-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear (1993)]
- 240-D: Binds to a linear epitope located in the Cluster I region – binding of 50–69 and 240-D inhibited by Fabs A1, A4, M8B, M26B, M12B and T2 [Binley (1996)]
- 240-D: Called F240: F240 in 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 (1996)]
- 240-D: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50-69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 [Mitchell (1998)]
- 240-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 246-D bound strongly or moderately to 24/26 HIV-1 group M clades viruses tested [Nyambi (2000)]
- 240-D: NIH AIDS Research and Reference Reagent Program: 1242

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
554	F240	gp160(592–606)	gp41(592–606 BH10)	LLGIWGCSGKLICTT	no	HIV-1 infection	human(IgG$_1\kappa$)

Donor: L. Cavacina or M. Posner, Dept. of Med. Harvard Med. School, Boston MA, USA
References: [Cavacini (1998a)]

- F240: Seems to be distinct from MAb 240-D, an antibody with a similar epitope in the immunodominant region of gp41 – dose-dependent reactivity with HIV isolates RF, SF2, IIIB, and MN was observed – F240 had no neutralizing activity and enhances infection in the presence of complement – reactivity of F240 is enhanced by preincubation of cells with sCD4 or anti-CD4BS MAb F105 – heavy and light chain variable domains were sequenced, and a strong homology to hu MAb 3D6 was observed, as 3D6 binds to the same epitope, these MAbs may define a human Ab clonotype [Cavacini (1998a)]

555	D61	gp160(592–608)	gp41(592–608 HXB2)	LLGIWGCSGKLICTTAV		dimeric Env	murine( )

References: [Earl (1994), Richardson Jr (1996), Weissenhorn (1996), Earl (1997)]

- D61: Linear gp41 epitope in the cluster I region – human sera blocked binding in oligomeric ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 [Richardson Jr (1996)]
- D61: Does not precipitate gp41(21-166), but due to a structural difference in the disulfide bonding region near the two cysteines – the authors propose that this region may change conformation during the activation of the membrane fusion state of the HIV-1 glycoprotein [Weissenhorn (1996)]
- D61: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues – this antibody, along with human MAb 246-D, can be blocked by any of a group of 8 conformational MAbs (M10, D41, D54, T4, T6, T9, T10 and T35) – members of this competition group are blocked by sera from HIV-1+ individuals [Earl (1997)]

556	D49	gp160(592–608)	gp41(597–613)	LLGIWGCSGKLICTTAV		dimeric Env	murine( )

References: [Earl (1994), Earl (1997)]

- D49: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues [Earl (1997)]

557	T32	gp160(592–608)	gp41(597–613)	LLGIWGCSGKLICTTAV		tetrameric Env	murine( )

References: [Earl (1994), Earl (1997)]

- T32: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues [Earl (1997)]

558	T34	gp160(592–608)	gp41(597–613)	LLGIWGCSGKLICTTAV		tetrameric Env	murine( )

References: [Earl (1994), Earl (1997)]

- T34: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues [Earl (1997)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
559	115.8	gp160(593–604)	gp41(598–609)	LGLIWGCSGKLIC		peptide LGLIWGCS-GKLIC (aa 598-609)	murine(IgM)
		References: [Oldstone (1991)] • 115.8: Poor reactivity with CSGKLIC – reacts well with longer HIV-2 peptide NSWGCAFRQVC as well as CAFRQVC – disulfide bond between cysteines required [Oldstone (1991)]					
560	M-22	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_{2b})
		References: [Yamada (1991)] • M-22: Strongest reaction of 12 anti-HIV-1 gp41 MAbs to a cellular 43-kd protein found in rat and human astrocytes [Yamada (1991)]					
561	M-24	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_1)
		References: [Yamada (1991)] • M-24: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]					
562	M-28	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_1)
		References: [Yamada (1991)] • M-28: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]					
563	M-2	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_{2b})
		References: [Yamada (1991)] • M-2: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]					
564	M-11	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_1)
		References: [Yamada (1991)] • M-11: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]					
565	M-13	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_{2b})
		References: [Yamada (1991)] • M-13: Reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]					

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
566	M-25	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_1)
		References: [Yamada (1991)]					
		• M-25: Reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]					
567	M-1	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_1,IgG_{2b})
		References: [Yamada (1991)]					
		• M-1: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]					
568	M-4	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_{2b})
		References: [Yamada (1991)]					
		• M-4: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]					
569	M-6	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_{2b})
		References: [Yamada (1991)]					
		• M-6: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]					
570	M-29	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_1)
		References: [Yamada (1991)]					
		• M-29: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]					
571	M-36	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	murine(IgG_1)
		References: [Yamada (1991)]					
		• M-36: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]					
572	polyclonal α(598-609)	gp160(594–601)	gp41(598–609)	GIWGCSGK		HIV-1 infection	human( )
		References: [Poumbourios (1992)]					
		• α(598-609): Affinity purified from HIV-1+ plasma – immunodominant region, binds oligomer and monomer [Poumbourios (1992)]					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
573	1B8.env	gp160(594–604)	gp41(594–605 HXB2)	GIWGCSGKLIC	no	HIV-1 infection	human($IgG_2\lambda$)
	References: [Banapour (1987)] • 1B8.env: Highly conserved epitope recognized by the majority of HIV-1 infected people [Banapour (1987)]						
574	polyclonal	gp160(594–609)	gp41(601–616)	GIWGCSGKLICTTAVP	no	HIV-1 infection	human sera( )
	References: [Petrov (1990)] • Immunodominant and broadly reactive peptide [Petrov (1990)]						
575	clone 3	gp160(597–606)	gp41( )	GCSGKLICTT	L	HIV-1 infection	human(IgG_1)
	References: [Cotropia (1992), Cotropia (1996)] • clone 3: Core binding domain gcsgkLIC – lack of serological activity to this region correlates with rapid progression in infants ([Broliden (1989)]) [Cotropia (1992)] • clone 3: Inhibits replication of three diverse HIV-1 laboratory strains, as well as an AZT-resistant isolate [Cotropia (1996)]						
576	41-6	gp160(598–604)	gp41(598–609)	CSGKLIC		peptide LGLIWGCS-GKLIC (aa 598-609)	murine(IgG_{2b})
	References: [Oldstone (1991)] • 41-6: Poor cross-reactivity with HIV-2 peptide CAFRQVC – slightly more reactive with LGLIWGCSGKLIC and HIV-2 form NSWGCAFRQVC – disulfide bond between cysteines required [Oldstone (1991)]						
577	4	gp160(598–604)	gp41(598–609)	CSGKLIC		peptide LGLIWGCS-GKLIC (aa 598-609)	murine(IgG_{2b})
	References: [Oldstone (1991)] • 4: There is another MAb with this ID that reacts with integrase [Oldstone (1991), Bizub-Bender (1994)] • 4: Poor cross-reactivity with HIV-2 peptide CAFRQVC – slightly more reactive with longer HIV-2 peptide NSWG-CAFRQVC [Oldstone (1991)]						
578	75	gp160(598–604)	gp41(598–609)	CSGKLIC		peptide LGLIWGCS-GKLIC (aa 598-609)	rat(IgG)
	References: [Oldstone (1991)] • 75: Poor cross-reactivity with HIV-2 peptide CAFRQVC – more reactive with longer HIV-2 peptide NSWG-CAFRQVC [Oldstone (1991)]						

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
579	68.1	gp160(598–604)	gp41(598–609)	CSGKLIC		peptide LGLIWGCS-GKLIC (aa 598-609)	murine(IgM)
	References: [Oldstone (1991)] • 68.1: Cross-reactive with HIV-2 peptide CAFRQVC – more reactive with longer HIV-1 peptide LGLIWGCSGKLIC and HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)]						
580	68.11	gp160(598–604)	gp41(598–609)	CSGKLIC		peptide LGLIWGCS-GKLIC (aa 598-609)	murine(IgM)
	References: [Oldstone (1991)] • 68.11: Cross-reactive with HIV-2 peptide CAFRQVC – more reactive with longer HIV-1 peptide LGLIWGCSGKLIC and HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)]						
581	41-7	gp160(598–604)	gp41(605–611)	CSGKLIC	no	HIV-1 infection	human($IgG_1\kappa$)
	References: [Bugge (1990)] • 41-7: Sera from 6/6 HIV-1 positive, but no HIV-2 positive individuals, interfered with 41–7 binding [Bugge (1990)]						
582	105-732	gp160(599–606)	gp41(601-608 HAM112)	KGRLICYT		group O rec Env pGO-8PL	murine($IgG_{2b}\kappa$)
	References: [Scheffel (1999)] • 105-732: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity – 105–732 bound to two overlapping peptides [Scheffel (1999)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
583	3D6	gp160(599–613)	gp41(604–617 BH10)	SGKLICTTAVPWNAS	no	HIV-1 infection	human($IgG_1\kappa$)

Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX
References: [Felgenhauer (1990), He (1992), Chen (1994b), Sattentau (1995), Stigler (1995), Wisnewski (1996), Kunert (1998), Cavacini (1998b), Cavacini (1998a), Cavacini (1999)]

- 3D6: Sequence of cDNA encoding V- regions [Felgenhauer (1990)]
- 3D6: Fab fragment crystal structure [He (1992)]
- 3D6: This MAb binds to HIV gp41, and to a 43 kd protein found in human T, B and monocyte cell lines, proposed molecular mimicry [Chen (1994b)]
- 3D6: Called IAM 41-3D6: binding increased after pretreatment of infected cells with sCD4 – binding domain overlaps site that is critical for gp120-gp41 association [Sattentau (1995)]
- 3D6: Optimum peptide for binding 3D6 Fab was CSGKLICTTAVPW [Stigler (1995)]
- 3D6: 3D6 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 (1996)]
- 3D6: The complete V, J and D(H) domain was sequenced – in contrast the sequences of five neutralizing MAbs, 3D6 had very little somatic mutation, with homologies of 97-98% relative to germline genes [Kunert (1998)]
- 3D6: Binds to the immunodominant region of gp41 – a strong homology between heavy variable domains of hu MAb 3D6 and MAb F20 was observed, these MAbs may define a human Ab clonotype [Cavacini (1998a)]
- 3D6: Cavacini *et al*. note that both MAbs F223 and 3D6 are anti-HIV-1 Env MAbs that have an autoimmune response and that both use uses VH3 germline genes [Cavacini (1999)]

584	F172-D8	gp160(604–615)	gp41(609–620)	CTTAVPWNASWS?			human( )

References: [Legastelois & Desgranges(2000)]

- F172-D8: As an approach to intercellular immunization using a single-chain variable fragment, scFvD8 was constructed based on the MAb F172-D8, directed at a loop in gp41 between the two heptad repeat regions – intracellular scFvD8 expression decreased gp160 expression and a scFvD8 transfected cell line did not support infection by HIV-1 Ba-L or primary isolates [Legastelois & Desgranges(2000)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
585	D50	gp160(632–655)	gp41(642–665)			dimeric Env	murine( )

References: [Earl (1994), Binley (1996), Richardson Jr (1996), Earl (1997)]

- D50: Thought to be a discontinuous epitope recognizing residues between 649–668 – designated cluster II – Fabs D5, D11, G1, T3, M12, M15, S6, S8, S9, S10 block binding [Binley (1996)]
- D50: Richardson suggests this is a linear gp41 epitope [Richardson Jr (1996)]
- D50: Found to bind to a linear peptide, between Env amino acids 642–655 – can be blocked by the conformation dependent MAbs D16, D17, D31, D36, D37, D40, D44, D55, D59, T37, and T45 – the region is in the immunogenic cluster two region – reactive with 9/10 HIV-1 strains tested, all except HIV-1 ADA, in which the change E659D and E662A may result in the loss of binding (ELLE to DLLA) [Earl (1997)]

586	5-21-3	gp160(642–665)	gp41(642–665 HXB2)	IHSLIEESQNQQEKNEQELLE-LDK		rec soluble gp41	murine( )

References: [Hunt (1990), Scheffel (1999)]

- 5-21-3: Recognizes a contiguous, conformation-dependent epitope in a hydrophilic region [Hunt (1990)]
- 5-21-3: Binds group M gp41, used as a control in a study of group O MAbs [Scheffel (1999)]

587	120-16	gp160(644–663)	gp41(644–663 HXB2)	SLIEESQNQQEKNEQELLEL	no	HIV-1 infection	human(IgG$_{2\kappa}$)

References: [Andris (1992), Robinson (1990b), Tyler (1990), Xu (1991), Robinson (1991), Eddleston (1993), Forthal (1995), Wisnewski (1996)]

- 120-16: Antibody dependent enhancement (ADE) of HIV-1 IIIB infectivity, synergistically enhanced by MAb V10-9 [Robinson (1990b)]
- 120-16: Potent ADCC (in contrast to MAb 98-43, gp41(579-604)) [Tyler (1990)]
- 120-16: Less reactive region than AVERY region – most Abs involving this region bound conformational epitopes, this was the only linear one [Xu (1991)]
- 120-16: Synergizes with huMAb 50–69 *in vitro* to enhance HIV-1 infection [Robinson (1991)]
- 120-16: Called SZ-120.16 [Eddleston (1993)]
- 120-16: No neutralizing activity, both ADCC and viral enhancing activity [Forthal (1995)]
- 120-16: 120–16 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 (1996)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
588	98-6	gp160(644–663)	gp41(644–663 HXB2)	SLIEESQNQQEKNEQELLEL	no	HIV-1 infection	human(IgG$_{2\kappa}$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY

References: [Pinter (1989), Gorny (1989), Till (1989), Robinson (1990b), Tyler (1990), Andris (1992), Sattentau & Moore(1991), Robinson (1991), Xu (1991), Eddleston (1993), Spear (1993), Tani (1994), Laal (1994), Chen (1995), Forthal (1995), Manca (1995), Sattentau (1995), Wisnewski (1996), Nyambi (1998), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000)]

- 98-6: Reacts preferentially with gp160 oligomer, compared to gp41 monomer [Pinter (1989)]
- 98-6: Kills HIV-infected cells when coupled to deglycosylated ricin A chain [Gorny (1989)]
- 98-6: Toxic to HIV-infected T cells (H9) and monocytes (U937) when coupled to deglycosylated A chain of ricin [Till (1989)]
- 98-6: No neutralizing or enhancing activity for HIV-1 IIIB [Robinson (1990b)]
- 98-6: Serves as target for antibody-dependent cellular cytotoxicity, ADCC [Tyler (1990)]
- 98-6: Two-fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 98-6: No neutralizing or enhancing activity [Robinson (1991)]
- 98-6: Appeared to be specific for a conformational or discontinuous epitope [Xu (1991)]
- 98-6: Called SZ-98.6 – binds to a conformational domain within aa 644–663 of gp41, and reacts with astrocytes, as do 167–7 and ND-15G1 [Eddleston (1993)]
- 98-6: Did not mediate deposition of complement component C3 on HIV infected cells, binding enhanced by sCD4 [Spear (1993)]
- 98-6: This MAb was expressed as a surface anti-gp41 monoclonal antibody receptor for gp41 on a CD4-negative B-cell line. Transfected cells could bind HIV Envelope, but could not be infected by HIV-1. When CD4 delivered by retroviral constructs was expressed on these cells, they acquired the ability to replicate HIV-1, and sIg/gp41 specifically enhanced viral replication [Tani (1994)]
- 98-6: Epitope described as Cluster II, 644-663, conformational – does not neutralize IIIB or synergize neutralization by anti-V3 MAb 447-52D or by CD4 BS MAbs [Laal (1994)]

Table of HIV MAbs

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
98-6 cont.						

- 98-6: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen (1995)]
- 98-6: No neutralizing activity, positive ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 98-6: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 98-6: Preferentially recognizes oligomeric form of gp41 – enhanced binding to HIV-1 infected cells at 37 degrees relative to 4 degrees – addition of sCD4 enhances binding [Sattentau (1995)]
- 98-6: 98–6 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 (1996)]
- 98-6: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-gp41 Abs 98-6, 1367 and 1342 were not able to bind detectably with any of the viruses from any clade [Nyambi (1998)]
- 98-6: 98–6 and 2F5 both bind to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, and to C43 alone but not to N51 alone – 98–6 and 2F5 have comparable affinities for C43, but 98–6 has a higher affinity for the complex and the binding of 98–6 is not inhibited by N51 [Gorny & Zolla-Pazner(2000)]
- 98-6: Binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5-fold preference for the oligomer, while other gp41 MAbs (1367, 98-6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)]
- 98-6: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistently to cluster II MAbs – no neutralizing activity was observed when tested against 5 isolates, but 98–6 did not bind to these isolates [Nyambi (2000)]
- 98-6: NIH AIDS Research and Reference Reagent Program: 1240

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
589	167-7	gp160(644–663)	gp41(644–663)	SLIEESQNQQEKNEQELLEL		HIV-1 infection	human($IgG_2\lambda$)

References: [Xu (1991), Eddleston (1993)]

- 167-7: Specific for a conformational epitope [Xu (1991)]
- 167-7: Called SZ-167.7 – binds to a conformational domain within aa 644–663 of gp41, and reacts with astrocytes, as do 98–6 and ND-15G1 [Eddleston (1993)]

590	ND-15G1	gp160(644–663)	gp41(644–663 HXB2)	SLIEESQNQQEKNEQELLEL		HIV-1 infection	human($IgG_1\kappa$)

References: [Eddleston (1993)]

- ND-15G1: Mapped to the conformational epitope within aa 644-663, and reacts with astrocytes, as do 98–6 and 167–7 [Eddleston (1993)]

591	167-D	gp160(644–663)	gp41(644–663 HXB2)	SLIEESQNQQEKNEQELLEL	no	HIV-1 infection	human($IgG_1\lambda$)

Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY

References: [Spear (1993), Forthal (1995), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000)]

- 167-D: Did not mediate deposition of complement component C3 on HIV infected cells – complement mediated virolysis of MN and IIIB in the presence of sCD4 [Spear (1993)]
- 167-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 167-D: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 167-D: This cluster II MAb binds to a conformational epitope in the region 644–663 – like most cluster II MAbs (126-6, 167-D, 1281, 1342, and 1379 all reacted similarly) it binds to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, but not to C43 nor to N51 alone [Gorny & Zolla-Pazner(2000)]
- 167-D: Binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5-fold preference for the oligomer, while other gp41 MAbs (1367, 98-6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)]
- 167-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistenently to cluster II MAbs [Nyambi (2000)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
592	polyclonal	gp160(662–667)	gp41( )	ELDKWA	L P	C-domain peptide linked to carrier	rabbit( )
		References: [Liao (2000)] • 2F5: Low levels of anti-ELDKWA antibodies are observed in HIV-1+ individuals, so a C-domain P2 peptide linked to a carrier was used to immunize mice and rabbits, and stimulated a high-level anti-ELDKWA response in mice and rabbits – vaccine was C-TSLIHSLIEESQNQQEKNEQELLELDKWA linked to carrier peptide K/G [(KGGG)_7-K] [Liao (2000)]					
593	polyclonal	gp160(662–667)	gp41(669–674)			peptide vaccine $C(ELDKWAG)_4$-BSA	mice and rabbits( )
		References: [Xiao (2000a)] • Strong epitope-specific neutralizing antibody responses were induced using the peptide, but not full gp160 [Xiao (2000a)]					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
594	2F5	gp160(662–667)	gp41(662–667 BH10)	ELDKWA	L P	HIV-1 infection	human(IgG$_3\kappa$)

Donor: Hermann Katinger, U. of Bodenkultur, or Polymun Scientific Inc., Vienna, Austria, or Viral Testing Systems Corp., Houson TX

References: [Buchacher (1992), Muster (1993), Allaway (1993), Klasse (1993a), Purtscher (1994), Laal (1994), Buchacher (1994), D'Souza (1994), Conley (1994b), Thali (1994), Chen (1994b), Muster (1994), Beretta & Dalgleish(1994), D'Souza (1995), Trkola (1995), Sattentau (1995), Moore & Ho(1995), Neurath (1995), Kessler (1995), Calarota (1996), McKeating(1996), Poignard (1996b), Sattentau(1996), Conley (1996), Pincus (1996), McKeating (1996), Stoiber (1996), Purtscher (1996), Schutten (1997), D'Souza (1997), Mo (1997), Li (1997), Kessler II (1997), Moore & Trkola(1997), Mascola (1997), Stamatatos (1997), Turbica (1997), Ugolini (1997), Burton & Montefiori(1997), Earl (1997), Gorny (1997), Andrus (1998), Mondor (1998), Connor (1998), Parren (1998a), Yang (1998), Trkola (1998), Fouts (1998), Ernst (1998), Takefman (1998), Li (1998), Jiang (1998), Parren (1998b), Geffin (1998), Kunert (1998), Frankel (1998), Montefiori & Evans(1999), Poignard (1999), Beddows (1999), Muhlbacher (1999), Parren (1999), Baba (2000), Gorny & Zolla-Pazner(2000), Kunert (2000), Liao (2000), Lu (2000b), Lu (2000a), Nyambi (2000), Park (2000), Xiao (2000b)]

- 2F5: DKWA defined as the core sequence – highly conserved epitope neutralizing MAb [Buchacher (1992), Muster (1993)]
- 2F5: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway (1993)]
- 2F5: Called IAM-41-2F5 – reports MAb to be IgG$_1$ – 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 2F5 is not affected [Klasse (1993a)]
- 2F5: Broadly reactive neutralizing activity, ELDKWA is relatively conserved – neutralized 2 primary isolates [Purtscher (1994)]
- 2F5: Failed to show synergy with anti-CD4 binding site IIIB neutralizing antibodies [Laal (1994)]
- 2F5: MAb generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]
- 2F5: Included in a multi-lab study for antibody characterization binding and neutralization assay comparison – D'Souza94
- 2F5: Called IAM-41-2F5 – neutralized lab and primary isolates – $t_{1/2}$ dissociation 122 min for the peptide, and 156 min for gp41 – core D(K/R)W – Ab resistant isolate had the sequence KLDNWA [Conley (1994b)]
- 2F5: gp41 mutation (582 A/T) that reduces neutralization of anti-CD4 binding site MAbs does not alter 2F5′s ability to neutralize [Thali (1994)]
- 2F5: 2F5 epitope ELDKWA inserted into an immunogenic loop in influenza virus hemagglutinin can elicit IIIB, MN and RF neutralizing sera in immunized mice [Muster (1994)]
- 2F5: Found to neutralize MN, JRCSF, and two B subtype primary isolates, but not a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs –D'Souza95

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)

2F5 cont.

- 2F5: Cross-clade primary virus neutralizing activity – LDKW defined as the core epitope [Trkola (1995)]
- 2F5: Called IAM 41-2F5 – exposed in the presence of gp120 on the cell surface, while most of gp41 is masked – binds proximal to transmembrane region [Sattentau (1995)]
- 2F5: Review: binds to the only generally accepted strong neutralizing epitope outside of gp120, one of only 3 MAbs with strong broad activity against primary viruses, the others are 2G12 and IgG_1b12 – unique member of epitope cluster [Moore & Ho(1995)] and John Moore, per comm 1996
- 2F5: MAb binding decreases the accessibility or alters the conformation of the gp41 fusion domain and of gp120 domains, including the binding site for the CD4 cell receptor [Neurath (1995)]
- 2F5: Broad cross-clade neutralization of primary isolates – additive neutralization in combination with anti-CD4BS MAb IgG_1b12 (Called BM12) [Kessler (1995)]
- 2F5: Only 4/20 Argentinian and 3/43 Swedish HIV+ sera reacted with LLELDKWASL – sera reacting with peptides that contained ELDKWA tended to have high neutralization titers – the region carboxyl terminal to EDLKWA was found to be more important for polyclonal sera AB binding, 670–675 WNWFDI – 2F5 bound most strongly to the peptide QELLELDKWA [Calarota (1996)]
- 2F5: ELDKWAS is in a gp41 binding region for the negative regulator of complement factor H (CFH) – Abs to HIV generally do not cause efficient complement-mediated lysis, but binding of 2F5 can interfere with CHF binding, facilitating HIV destruction by complement [Stoiber (1996)]
- 2F5: Primary isolates from clade A, B, and E are neutralized by 2F5 – neutralization requires the LDKW motif – neutralization resistant isolates or 2F5 selected variants all had substitutions in the D or K [Purtscher (1996)]
- 2F5: Neutralizes HXB2, primary isolates, and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]
- 2F5: Review: one of three MAbs (IgG_1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard (1996b)]
- 2F5: 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)]
- 2F5: 2F5 was infused into two chimpanzees which were then given an intravenous challenge with a primary HIV-1 isolate – both became infected, but with delayed detection and prolonged decrease in viral load relative to controls, indicating that preexisting, neutralizing antibodies (passively administered or actively elicited) affect the course of acute-phase virus replication and can be influential after the Ab can no longer be detected in the peripheral circulation [Conley (1996)]
- 2F5: 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 (1996)]
- 2F5: Called IAM 2F5 – antibody mediated enhancement or inhibition seemed to be determined by isolate rather than antibody specificity – in this study, only 2F5 inhibited the entry of all the viruses studied, irrespective of their phenotype, and directly proportional to its affinity to monomeric HIV-1 gp160 [Schutten (1997)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)

2F5 cont.

- 2F5: Of three neutralizing MAbs (257-D, IgG_1b12, and 2F5), 2F5 was the only one to inhibit the entry of all viruses studied, both SI and NSI, with a potency proportional to its affinity for monomeric gp126 [Schutten (1997)]
- 2F5: In a multilab evaluation of monoclonal antibodies, only IgG_1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 mug per ml for 90% viral inhibition – the isolates with no 2F5 neutralizing susceptibility had the sequences ALGQWA or ELDTWA instead of EDLKWA – 7/9 primary isolates were neutralized, and ALDKWQ and ALDKWA were susceptible to neutralization –D'Souza97
- 2F5: A JRCSF variant that was selected for IgG_1b12 resistance remained sensitive to MAbs 2G12 and 2F5, for combination therapy [Mo (1997)]
- 2F5: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB Env – strong neutralizer of SHIV-vpu+ – all Ab combinations tested showed synergistic neutralization – 2F5 has synergistic response with MAbs 694/98-D (anti-V3), 2G12, b12, and F105 [Li (1997)]
- 2F5: IgG_1b12 was more potent with greater breadth than MAb 2F5 in an infection reduction assay including 35 primary isolates [Kessler II (1997)]
- 2F5: Review: MABs 2F5, 2G12 and IgG_1b12 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)]
- 2F5: Binding of anti-gp120 MAbs IgG_1b12 or 654-30D does not mediate significant exposure of the gp41 epitopes for MAbs 2F5 and 50–69 [Stamatatos (1997)]
- 2F5: 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 (1997)]
- 2F5: Used to standardize polyclonal response to CD4 BS [Turbica (1997)]
- 2F5: The only MAb out of a large panel to show no correlation between Viral binding inhibition and neutralization [Ugolini (1997)]
- 2F5: This review summarizes results about 2F5: it binds extracellularly, near the transmembrane domain, it is the only gp41 MAb that is neutralizing, it reacts with many non-B clade viruses and has a paradoxically weak binding to virus, given the neutralizing titers [Burton & Montefiori(1997)]
- 2F5: 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 (1998)]
- 2F5: This MAb and the results of [Ugolini (1997)] are discussed – the authors propose that an Ab bound to gp41 would typically project less from the surface of the virion and so be unable to interfere with attachment [Parren (1998a)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)

2F5 cont.

- 2F5: 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, IgG_1b12, 2F5 and 447-52D [Connor (1998)]
- 2F5: A neutralization assay was developed based on hemi-nested 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 (1998)]
- 2F5: A wide range of neutralizing titers was observed that was independent of co-receptor usage – 2F5 was the most potent of the MAbs tested [Trkola (1998)]
- 2F5: Points out that 2G12 and 2F5, potent neutralizing antibodies, were identified by screening for cell surface (oligomeric Envelope) reactivity [Fouts (1998)]
- 2F5: The ELDKWA epitope was inserted into the antigenic site B of influenza hemagglutinin and expressed on baculovirus infected insect cells, flanked by 3 additional random amino acids, xELDKWAxx – FACS was used to isolate the clone that displayed the epitope with the most markedly increased binding capacity for 2F5, to identify particularly specific immunogenic constructs – PELDKWAPP was a high affinity form selected by FACS [Ernst (1998)]
- 2F5: Induces complement-mediated lysis in MN but not primary isolates – primary isolates are refractive to CML [Takefman (1998)]
- 2F5: 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 (1998)]
- 2F5: Used as a control in the study of anti-gp41 MAb NC-1 – 2F5 does not react with HIV-2 gp41 or gp160 [Jiang (1998)]
- 2F5: MAbs 2G12, 2F5 and b12 are broadly neutralizing, as are some human polyconal sera, but this paper describes a set of primary isolates that are resistant to all three MAbs and 2 broadly neutralizing sera – results indicate that resistance levels of pediatric isolates might be higher than adult isolates – resistance in general did not seem to be conferred by a loss of binding affinity for gp120 or gp41, rather by a more global perturbation of oligomeric Envelope [Parren (1998b)]
- 2F5: The natural immune response to the epitope of 2F5, ELDKWA, was studied in perinatally infected children and levels of reactivity to this epitope were correlated with absolute CD4 numbers over time and health status – 3/10 children who had no antibody reactivity to ELDKWA had substitutions in the epitope (ALDKWA, ELDQWA, and KLDKWA) – 2F5 competed with the ELDKWA-reactive sera depending on the serum titer [Geffin (1998)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)

2F5 cont.

- 2F5: The complete V, J and D(H) domain was sequenced – unlike non-neutralizing anti-gp41 MAb 3D6, five neutralizing MAbs (2F5, 2G12, 1B1, 1F7, and 3D5) showed extensive somatic mutations giving evidence of persistent antigenic pressure over long periods – in contrast to Geffin98, where multiple pediatric sera were found to compete with 2F5, cross-competition was noted to be very rare in sera from HIV+ adults – Kunert *et al*. propose that because there is a binding site of human complement factor H which overlaps the 2F5 binding site, it may generally be masked from the immune system – 2F5 also has a remarkably long CDR3 loop of 22 amino acids, and this region could not be readily assigned to any described D(H) fragment, leading to the suggestion of recombination of two fragments from novel regions [Kunert (1998)]
- 2F5: Prevention of the initial infection of mucosal dendritic cells is a desirable attributes of anti-HIV-1 vaccine stimulated Abs – IgG_1b12 and a combination of 2F5 and 2G12 could neutralize viral entry into DCs [Frankel (1998)]
- 2F5: rgp120 derived from a R5X4 subtype B virus was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs [Beddows (1999)]
- 2F5: A meeting summary presented results regarding neutralization –MAbs 2G12 and 2F5 tested for their ability to neutralize primary isolate infection of genetically engineered cell lines (cMAGI and others, presented by T. Matthews, A. Trkola, J. Bradac) – an advantage of such cells lines over PBMCs is that markers (X-Gal) can be added for staining to simplify the assay – the consensus of the meeting was that these engineered cell lines did not improve the sensitivity of detection of primary isolate neutralization – D. Burton and J. Mascola presented results concerning passive immunization and protection of hu-PBL-SCID mice and macaques, respectively, and both found combinations of MAbs that were able to achieve 99% neutralization *in vitro* corresponded to efficacy *in vivo* [Montefiori & Evans(1999)]
- 2F5: Hu-PBL-SCID mice were infected with HIV-1s JRCSF and SF162 to study the effect of NAbs on an established infection – no significant differences in the initial rate of decrease in viral load or the plateau levels of viral RNA between the b12 treated and control mice were seen – in most of the Ab treated mice b12 escape mutants were observed with varying patterns of mutations – a combination of b12, 2G12 and 2F5 protected 1/3 mice, and an isolate from one of the other two was resistant to neutralization by all three MAbs [Poignard (1999)]
- 2F5: In a study of 116 HIV-1+ individuals, Ab reactivity to a peptide encompassing the ELDKWA peptide decreased in CDC stage C patients compared with stage A patients, and longitudinal studies showed a decline in 6/8 patients, while overall Ab reactivity to rec soluble gp160 stayed constant [Muhlbacher (1999)]
- 2F5: Review of the neutralizing Ab response to HIV-1 [Parren (1999)]
- 2F5: Paper uses IgG_1 form of 2F5 – a triple combination of 2F5, F105 and 2G12 effectively neutralized perinatal infection of macaque infants when challenged with SHIV-vpu+ – the plasma half-life was 4.2 +/- 0.8 days [Baba (2000)]

Table of HIV MAbs

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
2F5 cont.						

- 2F5: MAbs 98–6 and 2F5 both bind to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, and to C43 alone but not to N51 alone – 98–6 and 2F5 have comparable affinities for C43, but 98–6 has a higher affinity for the complex and 2F5 may bind to an epitope of C43 that is directly involved with complex formation –and IgG_1 rec form of the Ab was used in this study [Gorny & Zolla-Pazner(2000)]
- 2F5: 2F5 is a candidate for immunotherapy, but generally IgG_1 has a longer half life in humans than IgG_3, so the isotype was switched – rec CHO-derived MAb 2F5 $IgG_1\kappa$ and hybridoma-derived MAb 2F5 IgG3κ displayed identical specificity, *in vitro* function, and epitope (ELDKWA) – it remains to be determined if isotype switching will prolongs β-clearance [Kunert (2000)]
- 2F5: Low levels of anti-ELDKWA antibodies are observed in HIV-1+ individuals, so a C-domain P2 peptide linked to a carrier was used to immunize mice and rabbits, and stimulated a high-level anti-ELDKWA response [Liao (2000)]
- 2F5: ELDKWA peptide vaccine study [Lu (2000b)]
- 2F5: ELDKWA peptide vaccine study [Lu (2000a)]
- 2F5: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistenently to cluster II MAbs [Nyambi (2000)]
- 2F5: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i gp120 specific MAbs are 20–100-fold more efficient at neutralizing the sensitive form – gp41 MAbs bind less, and 2F5 behaves the opposite of gp120 MAbs in that it neutralizes the "sensitive" form less efficiently [Park (2000)]
- 2F5: UK Medical Research Council AIDS reagent: ARP3063
- 2F5: NIH AIDS Research and Reference Reagent Program: 1475

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
595	polyclonal	gp160(662–667)	gp41(662–667 BH10)	ELDKWA	L	chimeric influenza virus/ELDKWA	murine(IgG,IgA)
	References: [Muster (1994), Muster (1995)] • Sustained ELDKWA specific IgA response in mucosa of immunized mice [Muster (1995)]						
596	polyclonal	gp160(662–667)	gp120(669–674)	ELDKWA		multiple-epitope vaccine CG-GPGRAFY-G-ELDKWA-G-RILAVERYLKD conjugated to BSA	rabbit(Ig)
	References: [Lu (2000b), Lu (2000a)] • High titer response to ELDKWA and RILAVERYLKD was observed, weak response to GPGRAFY – immunization with CG-(ELDKWA-GPGRAFY)$_2$-K was also tried, yielding a strong Ab response to both ELDKWA and GPGRAFY – gp160 vaccination yielded strong Ab response but not to any of the peptides studied here [Lu (2000b), Lu (2000a)]						
597	B30	gp160(720–734)	gp41(720–734 BH10)	HLPIPRGPDRPEGIE		mis-folded LAI rgp160	murine(IgG$_1$)
	Donor: Gearoge Lewis **References:** [Abacioglu (1994)] • B30: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]						
598	polyclonal	gp160(724–745)	gp41(731–752 IIIB)	PRGPDRPEGIEEEGGERDRDRS		gp41 peptide expressed in chimeric cowpea mosaic virus	murine(IgA,IgG$_{2a}$)
	References: [Durrani (1998)] • Comparison of intranasal and oral immunization of HIV-1 peptide expressed in a plant viral vector – intranasal gave the better response [Durrani (1998)]						
599	41S-2	gp160(725–745)	gp160(732–750)	RGPDRPEGIEEEGGERDRDRS	yes	Peptide coupled to keyhole limpet hemocyanin	murine(BALBc mice(IgG$_{2b}\kappa$)
	References: [Hifumi (2000)] • 41S-2: BALBc mice were immunized with gp41 peptide and a MAb specific for the peptide was generated – isolated MAb light chains displayed proteolytic activity towards the peptide epitope which may be due to a catalytic triad on light chain (Asp73, Ser76, and His79) – no catalytic activity was observed for the whole antibody [Hifumi (2000)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
600	447-52D	gp160(726–729)	gp120( )	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 (1992), Buchbinder (1992), Karwowska (1992b), Gorny (1993), Keller (1993), Cavacini (1993a), Spear (1993), Conley (1994a), Laal (1994), VanCott (1994), Gorny (1994), Moore (1994a), Sattentau(1995), Fontenot (1995), Saarloos (1995), Zolla-Pazner (1995), Zolla-Pazner & Sharpe(1995), Moore (1995a), Moore & Ho(1995), Forthal (1995), Jagodzinski (1996), Trkola (1996a), Sattentau(1996), D'Souza (1997), Binley (1997a), Fouts (1997), Hioe (1997), Boots (1997), Parren (1997b), Hill (1997), Gorny (1997), Inouye (1998), Mondor (1998), Smith (1998), Parren (1998a), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Connor (1998), Gorny (1998), Nyambi (1998), Hioe (1999), Beddows (1999), Gorny (2000), Grovit-Ferbas (2000), Hioe (2000), Ly & Stamatatos(2000), Nyambi (2000), Park (2000)]

- 447-52D: Requires GPXR at the tip of the V3 loop – neutralizes a broad array of B clade lab isolates [Gorny (1992)]
- 447-52D: 60-fold increase in neutralization potency when combined 1:1 with human MAb 588-D [Buchbinder (1992)]
- 447-52D: Reacts with MN, NY5, CDC4, SF2, RF, WM52, and HXB2 [Karwowska (1992b)]
- 447-52D: Neutralizes MN and IIIB: GPGR, and binds SF2: GPGR [Gorny (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 (1993)]
- 447-52D: Additive neutralization of MN and SF2 when combined with CD4 binding site MAb F105 – supra-additive neutralization of RF [Cavacini (1993a)]
- 447-52D: Complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear (1993)]
- 447-52D: Requires GPxR at the tip of the V3 loop, common in B clade – neutralized primary isolates [Conley (1994a)]
- 447-52D: Neutralization synergy in combination with CD4 binding domain MAbs [Laal (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 (1994)]
- 447-52D: Mild oxidation of carbohydrate moieties does not alter binding [Gorny (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 (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 (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 (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 (1995)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)

447-52D cont.

- 447-52D: Serotyping study using flow-cytometry – bound only to GPGR V3 loop tips [Zolla-Pazner (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 (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 (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 (1996)]
- 447-52D: Neutralizes JR-FL – strongly inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (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'Souza97
- 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 (1997)]
- 447-52D: Tested using a resting cell neutralization assay [Hioe (1997)]
- 447-52D: Viral binding inhibition by 447-D was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- 447-52D: Neutralizes TCLA strains but not primary isolates [Parren (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 (1997)]
- 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 (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 (1997)]
- 447-52D: Used as a control for comparison to five V3 RF selected antibodies – 447-52D was reactive with A, B, and C clade peptides, but not E [Gorny (1997)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)

447-52D cont.

- 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 (1998)]
- 447-52D: Inhibits binding of Hx10 to both CD4 positive and negative HeLa cells [Mondor (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 (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 (1998a)]
- 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, IgG_1b12, 2F5 and 447-52D [Connor (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 (1998)]
- 447-52D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 447-52D was the most potent and cross-reactive of 18 human MAbs tested and was the only MAb which bound to virions from isolates CA20 (subtype F), CA13 (subtype H), and VI526 (subtype G) [Nyambi (1998)]
- 447-52D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 447-52D: MAb peptide-reactivity pattern clustered with the immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group – 447 reacted with peptides containing GPGR, but also with many lacking this sequence (GPGQ, for example), and it failed to react with 2/14 peptides containing GPGR, illustrating the importance of context [Zolla-Pazner (1999b)]
- 447-52D: The presence of leukocyte function-associated molecule 1 (LFA-1) promotes virus infectivity and hinders neutralization, and anti-LFA-1 MAbs can enhance the neutralizing effect of anti-HIV V3 MAb 447-52D and anti-HIV CD4BS MAb IgG_1b12 – non-neutralizing anti-HIV CD4BS MAb 654-D did not become neutralizing in the presence of anti-LFA-1 MAbs [Hioe (1999)]

MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)

447-52D cont.

- 447-52D: rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – TCLA strains showed enhanced 447-52D neutralization sensitivity relative to PBMC-adapted lines (32X increase between HIV-1(M2424/PBMC(p0)) and HIV-1(M2424/H9(p9)) and a >128X increase between HIV-1(W61D/PBMC) and HIV-1(W61D/SupT1) isolates) [Beddows (1999)]
- 447-52D: Binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V3 MAbs 447-52D, 838-D, and 1334 bound with a 7–10-fold preference for the oligomer [Gorny (2000)]
- 447-52D: To determine the antigenicity of virus killed by thermal and chemical inactivation, retention of conformation-dependent neutralization epitopes was examined, and exposure of CD4BS epitopes was found to be enhanced (MAbs IgG_1b12, 205-46-9, and 205-43-1) – binding to 2G12 and 447-52D epitopes was essentially unaltered – the 17b CD4i epitope was also exposed [Grovit-Ferbas (2000)]
- 447-52-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inihibited proliferative responses of gp120 specific T cells – V3 MAbs 447-52-D and 268-10-D did not effect proliferation [Hioe (2000)]
- 447-52-D: Called 447D – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG_1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447D and 391-95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]
- 447-52D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 447-52D showed the highest cross-reactivity, bound to 24/26 viruses tested, but achieved 90% neutralization only against MN, 50% against CA5, and no neutralization was observed for 3 other isolates tested [Nyambi (2000)]
- 447-52D: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100-fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
601	C8	gp160(727–732)	gp41(727–732 BH10)	PDRPEG	no	mis-folded LAI rgp160	murine(IgG_1)
	References: [Pincus & McClure(1993), Pincus (1993), Abacioglu (1994)] • C8: Immunotoxin of C8 coupled to ricin-A does not mediate cells killing, and is not affected by sCD4 [Pincus & McClure(1993)] • C8: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – C8 was used as a control – the dominant response among vaccinees was to this mid-gp41 region, but not among the infected lab workers – Abs binding this region do not neutralize, bind to infected cells, nor serve as immunotoxins [Pincus (1993)] • C8: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]					
602	B31	gp160(727–734)	gp41(727–734 BH10)	PDRPEGIE		mis-folded LAI rgp160	murine(IgG_1)
	References: [Abacioglu (1994)] • B31: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]						
603	B33	gp160(727–734)	gp41(727–734 BH10)	PDRPEGIE	no	Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	murine(IgG_1)
	References: [Abacioglu (1994), Bristow (1994)] • B33: There are two MAbs in the literature named B33. See also gp120, LAI 123–142 [Bristow (1994)] • B33: Epitope boundaries mapped by peptide scanning IgG_1 [Abacioglu (1994)]						
604	LA9 (121-134)	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	?	murine(IgM)
	References: [Evans (1989)]						
605	ED6	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	?	murine(IgM)
	References: [Evans (1989)]						
606	1575	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Poliovirus/gp41 peptide chimera	murine( )
	Donor: C. Vella, NIBSC, Potters Bar UK **References:** [Evans (1989), Vella (1993), Buratti (1997), Cleveland (2000a)] • 1575: Neutralizing activity, less broad than 1577 [Evans (1989)] • 1575: Core epitope: IEEE – neutralized IIIB, but not RF or MN [Vella (1993)] • 1575: Study shows that MAb 1575 can recognize the IEEE sequence in both gp41, and in the HPG30 region of the p17 protein – motif is conserved in both regions in different HIV-1 clades [Buratti (1997)] • 1575: Ab binding to IEEE suppresses neutralizing Ab binding to adjacent epitope ERDRD [Cleveland (2000a)]						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
607	1576	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Poliovirus/gp41 peptide chimera	murine( )
		References: [Vella (1993)] • 1576: Not neutralizing [Vella (1993)]					
608	1578	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Poliovirus/gp41 peptide chimera	murine( )
		References: [Evans (1989), Vella (1993)] • 1578: No neutralizing activity – epitope may be formed by regions from both poliovirus and HIV [Evans (1989)] • 1578: Core epitope: IEEE – in this study, neutralized IIIB, but not RF or MN [Vella (1993)]					
609	1899	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Poliovirus/gp41 peptide chimera	murine( )
		References: [Vella (1993)] • 1899: Could neutralize HIV IIIB and HIV RF [Vella (1993)]					
610	1579	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Poliovirus/gp41 peptide chimera	murine( )
		References: [Vella (1993)] • 1579: Core epitope: IEEE – neutralized IIIB, but not RF or MN [Vella (1993)]					
611	1583	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Poliovirus/gp41 peptide chimera	murine( )
		References: [Evans (1989), Vella (1993), Sattentau (1995)] • 1583: Neutralizing activity, less broad than 1577 [Evans (1989)] • 1583: Core epitope: ERDRD – Could neutralize HIV IIIB but not HIV RF [Vella (1993)] • 1583: Cytoplasmic domain, epitope not exposed at the surface of HIV-1 infected cells [Sattentau (1995)]					
612	1907	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Poliovirus/gp41 peptide chimera	murine( )
		References: [Vella (1993)] • 1907: Could not neutralize HIV IIIB, RF or MN [Vella (1993)]					

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
613	1908	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Poliovirus/gp41 peptide chimera	murine( )
		References: [Evans (1989), Vella (1993), Sattentau (1995)] • 1908: Neutralized IIIB, but not RF or MN [Vella (1993)] • 1908: Cytoplasmic domain, epitope not exposed at the surface of HIV-1 infected cells [Sattentau (1995)]					
614	1909	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Poliovirus/gp41 peptide chimera	murine( )
		References: [Vella (1993)] • 1909: Neutralized HIV IIIB but not HIV RF [Vella (1993)]					
615	41-1	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Peptide 735–752 IIIB	murine(IgMκ)
		References: [Mani (1994), Dalgleish (1988)] • 41-1: This antibody gp41(735-752 IIIB) [Dalgleish (1988)] seems to have been named the same as a different MAb to gp41(584-609) [Mani (1994)] • 41-1: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish (1988)]					
616	41-2	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Peptide 735–752 IIIB	murine(IgMκ)
		References: [Dalgleish (1988)] • 41-2: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish (1988)]					
617	41-3	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Peptide 735–752 IIIB	murine(IgMκ)
		References: [Dalgleish (1988)] • 41-3: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish (1988)]					
618	88-158/02	gp160(732–747)	gp41(732–752 IIIB)	GIEEEGGERDRDRSIR		rgp41 IIIB	murine(IgG_{2b})
		References: [Niedrig (1992a)] • 88-158/02: Mild inhibition of *in vitro* activity at high MAb concentrations – profound enhancing activity at low concentrations – significant reactivity to virion – domain non-immunogenic in humans [Niedrig (1992a)]					
619	88-158/022	gp160(732–747)	gp41(732–752 IIIB)	GIEEEGGERDRDRSIR		rgp41 IIIB	murine(IgG_{2b})
		References: [Niedrig (1992a)] • 88-158/022: Mild inhibition of *in vitro* activity at high MAb concentrations – profound enhancing activity at low concentrations – significant reactivity to virion – domain non-immunogenic in humans [Niedrig (1992a)]					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
620	88-158/079	gp160(732–747)	gp41(732–752 IIIB)	GIEEEGGERDRDRSIR		rgp41 IIIB	murine(IgG_1)

References: [Niedrig (1992a)]

- 88-158/079: Mild inhibition of HIV *in vitro* at high MAb concentrations – profound enhancing activity at low concentrations – weak binding to virion – domain non-immunogenic in humans [Niedrig (1992a)]

621	polyclonal	gp160(733–736)	gp41(735-752 IIIB)	IEEE	L	Cowpea mosaic virus chimera-HIV gp41-peptide PRGPDRPEGIEEEGGERDRDRS	murine(IgG)

References: [Cleveland (2000b)]

- When PRGPDRPEGIEEEGGERDRDRS was used as antigen an immunodominant, non-neutralizing response to IEEE was observed, but immunization GERDRDR shifts the response to ERDRD [Cleveland (2000b)]

622	B8	gp160(733–741)	gp41(733–741 BH10)	IEEEGGERD	no	mis-folded LAI rgp160	murine(IgG_1)

References: [Pincus (1993), Abacioglu (1994)]

- B8: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – B8 was used as a control – the dominant response among vaccinees was to this mid-gp41 region, but not among the infected lab workers – Abs binding this region do not neutralize, bind to infected cells, nor serve as immunotoxins [Pincus (1993)]
- B8: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

623	polyclonal	gp160(739–743)	gp41(735-752 IIIB)	ERDRD	L	Cowpea mosaic virus chimera-HIV gp41-peptide GERDRDR	murine(IgG)

References: [Cleveland (2000b)]

- ERDRD-specific IgG recognizes an externalized loop of the gp41 C-terminal tail with high affinity – neutralized HIV-1 B clade strains IIIB, NL-4.3, RF, MN and D clade virus CBL-4, but HXB-2D (clade B) was not recognized – when PRGPDRPEGIEEEGGERDRDRS was used as antigen an immunodominant, non-neutralizing response to IEEE was observed, but immunization GERDRDR shifts the response to ERDRD – NAb does not inhibit attachment of free virus, but does inhibit by an event that precedes fusion-entry [Cleveland (2000b)]

Table of HIV MAbs

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
624	1577	gp160(739–743)	gp41(735–752 IIIB)	ERDRD	no	Poliovirus/gp41 peptide chimera	murine( )
		Donor: C. Vella or Morag Ferguson (NIBSC, Potters Bar UK) **References:** [Evans (1989), D'Souza (1991), Vella (1993), Cleveland (2000a)] • 1577: Raised against IIIB peptide chimera – neutralized African and American HIV-1 lab strains [Evans (1989)] • 1577: Non-neutralizing in this multi-lab study –D'Souza91 • 1577: Core epitope: ERDRD – could neutralize HIV IIIB and HIV RF [Vella (1993)] • 1577: Ab binding to IEEE suppresses neutralizing Ab binding to adjacent epitope ERDRD [Cleveland (2000a)] • 1577: UK Medical Research Council AIDS reagent: ARP317 • 1577: NIH AIDS Research and Reference Reagent Program: 1172					
625	4E10	gp160(823–829)	gp41(824–830 BH10)	AEGTDRV	no	HIV-1 infection	human(IgG$_{3}\kappa$)
		References: [Buchacher (1992), Buchacher (1994), D'Souza (1994)] • 4E10: MAbs generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells – also binds to MHC class II proteins – anti-class II Abs are only found in HIV-1 positive people [Buchacher (1994)] • 4E10: Included in a multi-lab study for antibody characterization, binding and neutralization assay comparison –D'Souza94					
626	Chim 1	gp160(838–844)	gp120( )	KVVKEIP			humanized chimpanzee( )
		References: [Pincus & McClure(1993), Pincus (1996)] • 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 (1996)]					
627	polyclonal	Env( )	gp120( )		L	rgp120 SF2 in PLG+MF59 microparticles	murine and baboon( )
		References: [O'Hagan (2000)] • Microparticles were used as an adjuvant for entrapped HIV-1 gp120 and induced strong serum IgG responses in mice – polylactide co-glycolide polymer (PLG) microparticles in combination with MF59 had the highest response [O'Hagan (2000)]					
628	1088	Env( )	gp120(V2)				( )
		References: [Berman (1997)] • 1088: Binds weakly to 2/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)]					