Table 9: $\mathbf{gp120}$

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
223 M85	gp120(C1 30-51 LAI)	gp120(29-50)	ATEKLWVTVYYGVPVW- KEATTT	Z	451 Env	$\operatorname{murine}(\operatorname{Ig} \operatorname{G}_1)$
	 Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese et al.(1992), Moore et al.(1994c) et al.(1997), Wyatt et al.(1997)] NOTES: M85: Immunoblot and RIP reactive for strains IIIB, 451, I Marzo Veronese et al.(1992)] M85: C1 domain – mutation 40 Y/D impairs binding – the suggesting conformational component [Moore et al.(1994c)] M85: Binding inhibited by MAb 4D4#85, enhanced by con anti-18 MAbs [Moore & Sodroski(1996)] M85: Binds efficiently to sen 120 but not soluble en 120+9 	nese se et al.(1992), Mo se et al.(1992), Mo IP reactive for str [2)] tion 40 Y/D impa [1 component [Moc y MAb 4D4#85, o Sodroski(1996)] Som 120 but not s	 Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996), Ditzel et al.(1997), Wyatt et al.(1997)] NOTES: M85: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese et al.(1992)] M85: C1 domain – mutation 40 Y/D impairs binding – the relative affinity for denatured/native gp120 is < .01, suggesting conformational component [Moore et al.(1994c)] M85: Binding inhibited by MAb 4D4#85, enhanced by conformationally sensitive anti-V3 MAb 5G11, and some anti-18 MAbs [Moore & Sodroski(1996)] M85: Binds efficiently to sep120 but not soluble ep120+op41, suggesting its ep120 epitone is blocked by ep41 	1994d), Moore & UTZ – binds de ty for denaturec sensitive anti-V2	¿Sodroski(1996), Dit. eglycosylated gp120 Vnative gp120 is < .0 3 MAb 5G11, and sor	zel [di [di 01, 01, 01, 01, 01, 01, 01]
	• M85: Binds efficiently to s binding [Wyatt et al.(1997)]	sgp120 but not s	M85: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 binding [Wyatt et al.(1997)]		epitope is blocked by gp41	41
224 7E2/4	gp120(C1 31-50 LAI)	gp120(30-49)	TEKLWVTVYYGVPVWK- EATT		Env glycopro	murine(IgG)
	Donor: S. Ranjbar, NIBSC, UK References: [Moore et al.(1994c)] NOTES: • 7E2/4: C1 domain – the relative affinity for denatured/native g [Moore et al.(1994c)] • 7E2/4: UK Medical Research Council AIDS reagent: ARP3050	K (4c)] relative affinity fo urch Council AID:	or: S. Ranjbar, NIBSC, UK erences: [Moore et al.(1994c)] FES: 7E2/4: C1 domain – the relative affinity for denatured/native gp120 is .07, suggestin, [Moore et al.(1994c)] 7E2/4: UK Medical Research Council AIDS reagent: ARP3050	, suggesting cor	g conformational component	ent
225 M92	gp120(C1 31-50 LAI) g Donor: Fulvia di Marzo Veronese	gp120(40-49) nese	GVPVWKEATT	Z	451 Env	$\mathrm{rat}(\mathrm{lgG}_1)$
	 References: [di Marzo Veronese et al.(1992), Moore NOTES: M92: Immunoblot reactive, RIP negative, but pro RF, and RUTZ [di Marzo Veronese et al.(1992)] M92: The relative affinity for denatured/native g 	se et al.(1992), Me, RIP negative, but Veronese et al.(19 for denatured/nat	NOTES: • M92: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)] • M92: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]	(1994d)] 5120 – reacts wit 54c)]	:h strains IIIB, 451, M	ŢŊ,

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
226 4D4#85	gp120(C1 41-50 LAI) gp120(40-49) GVPVWI Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA References: [Moore et al.(1994c), Moore et al.(1994d), Moo NOTES: • 4D4#85: C1 domain – the relative affinity, denatured/na et al.(1994c)] • 4D4#85: Inhibits binding of C1 MAb M85, C1-C5 dis induced MAbs 48d and 17b [Moore & Sodroski(1996)] • 4D4#85: Binds efficiently to sgp120 but not soluble gp binding – does not bind to HXBc2 gp120 if the 19 C-te deleted [Wyatt et al.(1997)] • 4D4#85: A panel of MAbs were shown to bind with sin deglycosylated or variable loop deleted core gp120 pro a structure closely approximating full length folded mo	gp120(40-49) hur, NCI, Fredericl 1994c), Moore et al.(ne relative affinity, on the to HXBc2 gp120 if the to HXBc2 gp120 if the relative shown to the leop deleted con- tains affinity full length	gp120(C1 41-50 LAI) gp120(40-49) GVPVWKEATT Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA References: [Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996), Wyatt et al.(1997), Binley et al.(1998)] NOTES: • 4D4#85: C1 domain – the relative affinity, denatured/native gp120 is 0.1 – mutation 45 W/S impairs binding [Moore et al.(1994c)] • 4D4#85: Inhibits binding of C1 MAb M85, C1-C5 discontinuous epitope MAbs 181 and 212A, and CD4 binding induced MAbs 48d and 17b [Moore & Sodroski(1996)] • 4D4#85: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-50, are deleted [Wyatt et al.(1997)] • 4D4#85: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (\Delta V1, V2, and V3), thus such a core protein produces a structure closely approximation full length folded monomer (Binley et al.(1998))	-mutation 45 W/ -mutation 45 W/ e MAbs 181 and sting its gp120 ep 1 conjunction with finity and similar and V3), thus such 1 (1998)	Env 997), Binley et al.(19) S impairs binding [Ma I 212A, and CD4 bind itope is blocked by g h C1 positions 31-50, h competition profiles h a core protein produ	murine(IgG) 98)] oore ding to a to a uces
227 M86	gp120(C1 42-61 LAI) gp120(41-60) VPVWKEATTTI Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese et al.(1992), Moore et al.(1994c)] NOTES:	gp120(41-60) onese nese et al.(1992), N	20(C1 42-61 LAI) gp120(41-60) VPVWKEATTTLFCASDA- N 451 Env KAY ior: Fulvia di Marzo Veronese erences: [di Marzo Veronese et al.(1992), Moore et al.(1994c)] TES:	Z	451 Env	$\operatorname{murine}(\operatorname{IgG}_1)$
228 133/11	gp120(C1 64-78) gp] Donor: Fulvia di Marzo Veronese References: [Niedrig et al.(1992b)] NOTES: • 133/11: Region of overlap for re	gp120(63-77) onese 1992b)] ap for reactive pepti	gp120(63-77) EVHNVWATHACVPTD L IIIB gp120 or: Fulvia di Marzo Veronese erences: [Niedrig et al.(1992b)] TES: 133/11: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig et al.(1992b)]	L zation of lab strai	IIIB gp120 ins [Niedrig et al.(199:	$murine(IgG_1)$ $[2b]$

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
229 133/237	gp120(C1 51-70 LAI) gp120(60-69) YDTEVHNVWA Donor: Fulvia di Marzo Veronese References: [Niedrig et al.(1992b), Moore et al.(1994c), Moore et al.(1994d)] NOTES:	gp120(60-69) nese 992b), Moore et al	YDTEVHNVWA .(1994c), Moore et al.(1994d)	L	IIIB gp120	$\operatorname{murine}(\operatorname{IgG}_1)$
	133/237: Region of overla133/237: The relative affi et al.(1994c)]	p for reactive pept nity, denatured/na	133/237: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig et al.(1992b)] 133/237: The relative affinity, denatured/native gp120 is 1.4 – mutation of position 69 W/L impairs binding [Moore et al.(1994c)]	ization of lab stra f position 69 W/l	ins [Niedrig et al.(1992b) _ impairs binding [Moor	e)]
230 133/290	gp120(C1 61-70 LAI) g Donor: Fulvia di Marzo Veronese	gp120(60-69)	YDTEVHNVWA		IIIB gp120	$murine(IgG_1)$
	References: [Niedrig et al.(1992b), Thali et al.(1993), Moore et al.(1994c), Moore et Binley et al.(1997), Wyatt et al.(1997), Binley et al.(1998)] NOTES:	992b), Thali et al l.(1997), Binley e	(1993), Moore et al.(1994c), t al.(1998)]		al.(1994d), Wyatt et al.(1995),),
	133/290: Region of overla133/290: The relative affinet al.(1994c)]	ip for reactive pept nity for denatured/	133/290: Region of overlap for reactive peptides is WATHA—weak neutralization of lab strains [Niedrig et al.(1992b)] 133/290: The relative affinity for denatured/native gp120 is 2.2—mutation in position 69 W/L impairs binding [Moore et al.(1994c)]	ization of lab stra in position 69 W/	ins [Niedrig et al.(1992b)] L impairs binding [Moor	· e
	 133/290: Used for antige [Wyatt et al.(1995)] 	n capture assay,	133/290: Used for antigen capture assay, either to bind gp120 to the ELISA plate. [Wyatt et al.(1995)]	•	or to quantitate bound gp120	Ö
	• 133/290: Reciprocal bind is enhanced by some C5 a	ling inhibition wit and C1 binding sit	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)]	oinds to a disconski(1996)]	tinuous epitope – bindin	ŰΩ
	 133/290: A high avidity antibody as assessed by urea elution 133/290: Binds efficiently to sgp120 but not soluble gp120+; 	intibody as assesse y to sgp120 but no	133/290: A high avidity antibody as assessed by urea elution 133/290: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp1		120 epitope is blocked by gp41	<u>`</u>
	 binding [Wyatt et al.(1997)] 133/290: A panel of MAbs of deglycosylated or variable longer 	7)] os were shown to e loop deleted core	binding [Wyatt et al.(1997)] 133/290: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces	finity and similar and V3), thus suc	competition profiles to ha core protein produce	» a
231 D/3G5	gp120(C1 73-82 LAI)	gp120(72-81)	ACVPTDPNPQ	Z	Baculovirus- expressed rgp120 LAI	$\mathrm{murine}(\mathrm{IgG}_1)$
	Donor: Fulvia di Marzo Veronese References: [Bristow et al.(1994)] NOTES: • D/3G5: C1 MAb generated in	nese 994)] ed in a study of the	or: Fulvia di Marzo Veronese erences: [Bristow et al.(1994)] TES: D/3G5: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]	rgp120 and rgp	160 [Bristow et al.(1994)	<u>``</u>

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
232 D/6A11	gp120(C1 73-82 LAI)	gp120(72-81)	ACVPTDPNPQ	Z	Baculovirus- expressed rgp120 LAI	murine
	Donor: Fulvia di Marzo Veronese References: [Bristow et al.(1994)] NOTES:	onese 994)]				
	• D/6A11: C1 MAb genera	ated in a study of	D/6A11: C1 MAb generated in a study of the humoral immune response to rgp120 and		rgp160 [Bristow et al.(1994)]	
233 D/5E12	gp120(C1 73-92 LAI)	gp120(72-91)	ACVPTDPNPQEVVLVNV- TEN	Z	Baculovirus- expressed rgp120 LAI	murine
	Donor: Fulvia di Marzo Veronese References: [Bristow et al.(1994)]	onese 994)]				
	• D/5E12: C1 MAb genera	ted in a study of	D/5E12: C1 MAb generated in a study of the humoral immune response to rgp120 and		rgp160 [Bristow et al.(1994)]	
234 4A7C6	gp120(C1 81-90 LAI) Donor: R. Tedder	gp120(80-89)	gp120(80-89) PQEVVLVNVT		Env glycopro	murine(IgG)
	References: [Thiriart et al.(Moore & Sodroski(1996)] NOTES:	1989), Thali et al	References: [Thiriart et al.(1989), Thali et al.(1993), Moore & Ho(1993), Moore et al.(Moore & Sodroski(1996)] NOTES:		1994c), Moore et al.(1994d),	
	 4A7C6: Bound preferentially to denature 4A7C6: The relative affinity for denature 4A7C6: C1 region epitope (88 N/P suimpaired binding [Moore et al.(1994d)] 	ially to denatured ity for denatured pe (88 N/P subset al.(1994d)]	4A7C6: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] 4A7C6: The relative affinity for denatured/native gp120 is 7.9 – mutation 88 N/P impairs binding [Moore et al.(1994c)] 4A7C6: C1 region epitope (88 N/P substitutions abrogates binding), but substitutions 380 G/F and 420 I/R also impaired binding [Moore et al.(1994d)]	93)] 8 N/P impairs l out substitution	binding [Moore et al.(1994c)] ns 380 G/F and 420 I/R also	
	 4A7C6: Reciprocal binding inhibition with the antibody 133/192 135/9[Moore & Sodroski(1996)] 4A7C6: UK Medical Research Council AIDS reagent: ARP 360 	ing inhibition wit ((1996)] search Council A	4A7C6: Reciprocal binding inhibition with the antibody 133/192 – enhanced by anti-C' 135/9[Moore & Sodroski(1996)] 4A7C6: UK Medical Research Council AIDS reagent: ARP 360	ced by anti-C5	5 antibodies, and C1 antibody	
235 B242	gp120(C1 83-92 LAI)	gp120(82-91)	gp120(82-91) EVVLVNVTEN	Z	Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_1)$
	Donor: R. Tedder References: [Bristow et al.(1994)] NOTES: • R242: C1 MAb generated in a	994)]	or: R. Tedder erences: [Bristow et al.(1994)] TES:	ron120 and ro	m160 [Bristow et al (1994)]	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
236 1D10	gp120(C1 81-100 LAI)	gp120(80-99)	PQEVVLVNVTENFDMW- KNDM	L	IIIB-rgp120	rat
	Donor: R. Tedder References: [Dowbenko et al NOTES:	.(1988), Berman ei	Donor: R. Tedder References: [Dowbenko et al.(1988), Berman et al.(1991), Nakamura et al.(1992), Moore NOTES:		et al.(1994c)]	
	1D10: Cross-blocks 5B31D10: The relative affinit	in IIIB-rsgp160 EI y for denatured/nat	1D10: Cross-blocks 5B3 in IIIB-rsgp160 ELISA – type specific in rgp120 ELISA binding [Nakamura et al.(1992)] 1D10: The relative affinity for denatured/native gp120 is 13 – mutation 88 N/P impairs binding [Moore et al.(1994c)]	ELISA binding N/P impairs binding	[Nakamura et al.(1992)] ling [Moore et al.(1994c)]	
237 133/192	gp120(C1 91-100 LAI) Donor: Matthias Niedrig	gp120(90-99)	ENFDMWKNDM	T	IIIB gp120	$murine(IgG_1)$
	References: [Niedrig et al.(1992b), Moore et al.(196a), Binley et al.(1997), Binley et al.(1998)] NOTES:	1992b), Moore et al.(197), Binley et al.(19	References: [Niedrig et al.(1992b), Moore et al.(1993b), Moore et al.(1994c), Moore et al.(1996a), Binley et al.(1997), Binley et al.(1998)] NOTES:		& Sodroski(1996), Trkola	ñ
	 133/192: Epitope seems c 133/192: The relative affi 	complex, binds munity for denatured.	133/192: Epitope seems complex, binds multiple peptides – weak neutralization of lab 133/192: The relative affinity for denatured/native gp120 is 1.8 [Moore et al.(1994c)]	zation of lab stra al.(1994c)]	strain [Niedrig et al.(1992b)]]
	 133/192: C1 region – substitutions 76P/Y, 113 l tutions enhanced binding [Moore et al.(1994d)] 	stitutions 76P/Y, 1 [Moore et al.(1994	133/192: C1 region – substitutions 76P/Y, 113 D/A or R, 117 K/W, 420 I/R, 427 W/S tutions enhanced binding [Moore et al.(1994d)]	R, 427 W/S im	impair binding, other substi-	•
	 133/192: Reciprocal bindii [Moore & Sodroski(1996)] 	ling inhibition wit)]	133/192: Reciprocal binding inhibition with the antibody 4A7C6 – enhanced by some anti-C5 and-C1 antibodies [Moore & Sodroski(1996)]	nced by some a	inti-C5 and-C1 antibodies	S
	• 133/192: Does not neutra	lize JR-FL nor blo	133/192: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [T-k-c]a et al. (1996a)]	-5 in a MIP-1 β -	CCR-5 competition study	V
	• 133/192: A low avidity C1 antibody as assessed by urea elution	1 antibody as asse	ssed by urea elution			
	 133/192: A panel of MAI deglycosylated or variable 	os were shown to t e loop deleted core	133/192: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces	nity and similand V3), thus such	nilar competition profiles to a such a core protein produces	S
	a structure closely approx	imating full length	a structure closely approximating full length folded monomer [Binley et al.(1998)]	.(1998)]		

	241 D/4B5		240 GV4D3		239 B2		238 C6	MAb ID
NOTES: • D/4B5: C1 MAb generated in	gp120(C1 93-101 LAI)	Donor: Matthias Niedrig References: [Denisova et al.(1996)] NOTES: • GV4D3: When anti-V3 MAb N linear epitopes – MAbs GV4H4 [Denisova et al.(1996)]	3 gp120(92-100 IIIB)	Donor: Matthias Niedrig References: [Thali et al.(NOTES: B2: C1 region – epite B2: The relative affir B2: There is FNM/Fl B2: A low avidity an	gp120(C1 91-100 LAI)	Ponor: Matthias Niedrig References: [Pincus & M NOTES: C6: Also called Ch6? C6: C1 region – epito C6: The relative affin C6: There is FNM/FI C6: Called Ch6 – bincell killing – sCD4 ha C6: NIH AIDS Resea	gp120(C1 91-100 LAI)	D Location
ıl.(1994)] erated in a study of t	gp120(92-100)	al.(1996)] V3 MAb M77 was 1 bs GV4H4 and GV5	gp120(91-99)	Matthias Niedrig ces: [Thali et al.(1993), Abacioglu et al.(1994), Mo : C1 region – epitope boundaries mapped by peptide The relative affinity for denatured/native gp120 is There is FNM/FDM polymorphism in LAI-based p A low avidity antibody as assessed by urea elution	gp120(90-99)	IcClure(1993), Abace ppe boundaries mapping for denatured/namely DM polymorphism in ds to gp120 but not the day arch and Reference I	gp120(90-99)	WEAU
he humoral immune respo	gp120(92-100) FNMWKNDMV	or: Matthias Niedrig erences: [Denisova et al.(1996)] TES: GV4D3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, linear epitopes – MAbs GV4H4 and GV5F9 are homologous to GV4D3 and were gene [Denisova et al.(1996)]	NFNMWKNDM	or: Matthias Niedrig erences: [Thali et al.(1993), Abacioglu et al.(1994), Moore et al.(1994c), Moore et al.(1985: IES: B2: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacion B2: The relative affinity for denatured/native gp120 is 1.4 [Moore et al.(1994c)] B2: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. B2: A low avidity antibody as assessed by urea elution	ENFDMWKNDM	or: Matthias Niedrig Prences: [Pincus & McClure(1993), Abacioglu et al.(1994), Moore et al.(1994c) Pres: C6: Also called Ch6? C6: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [C6: The relative affinity for denatured/native gp120 is 0.9 [Moore et al.(1994c)] C6: There is FNM/FDM polymorphism in LAI-based peptides – N is essential (C6: Called Ch6 – binds to gp120 but not to infected cells – when linked to ricin / cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)] C6: NIH AIDS Research and Reference Reagent Program: 810	ENFDMWKNDM	Sequence
or: Matthias Niedrig rences: [Bristow et al.(1994)] [TES: D/4B5: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]	N Baculovirus-expressed rgp120 LAI	or: Matthias Niedrig rences: [Denisova et al.(1996)] [ES: GV4D3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV4H4 and GV5F9 are homologous to GV4D3 and were generated in the same experiment [Denisova et al.(1996)]	gp120 complexed	 Donor: Matthias Niedrig References: [Thali et al.(1993), Abacioglu et al.(1994), Moore et al.(1994c), Moore et al.(1994d), Binley et al.(1997)] NOTES: B2: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu et al.(1994)] B2: The relative affinity for denatured/native gp120 is 1.4 [Moore et al.(1994c)] B2: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.) B2: A low avidity antibody as assessed by urea elution 	mis-folded LAI rgp160	 Donor: Matthias Niedrig References: [Pincus & McClure(1993), Abacioglu et al.(1994), Moore et al.(1994c), Pincus et al.(1996)] NOTES: C6: Also called Ch6? C6: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu et al.(1994)] C6: The relative affinity for denatured/native gp120 is 0.9 [Moore et al.(1994c)] C6: There is FNM/FDM polymorphism in LAI-based peptides – N is essential (J. P. Moore, per. comm.) C6: Called Ch6 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)] C6: NIH AIDS Research and Reference Reagent Program: 810 	mis-folded LAI rgp160	Neutralizing Immunogen
4)]	murine	to	murine)]	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_{2b})$	ate	$\operatorname{murine}(\operatorname{\lg} G_1)$	Species(Isotype)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
242 D/6B2	gp120(C1 93-101 LAI)	gp120(92-100)	FNMWKNDMV	Z	Baculovirus- expressed rgp120 LAI	$\mathrm{murine}(\mathrm{IgG}_1)$
	Donor: Matthias NiedrigReferences: [Bristow et al.(1994)]NOTES:D/6B2: C1 MAb generated in	994)] ed in a study of the	nor: Matthias Niedrigerences: [Bristow et al.(1994)]TES:D/6B2: C1 MAb generated in a study of the humoral immune response to rgp120 and		rgp160 [Bristow et al.(1994)])]
243 D/5A11	gp120(C1 93-101 LAI)	gp120(92-100)	FNMWKNDMV	Z	Baculovirus- expressed rgp120 LAI	murine
	Donor: Matthias Niedrig References: [Bristow et al.(1994)] NOTES: • D/5A11: C1 MAb generated in	994)] ated in a study of the	 nor: Matthias Niedrig erences: [Bristow et al.(1994)] TES: D/5A11: C1 MAb generated in a study of the humoral immune response to rgp120 and 		rgp160 [Bristow et al.(1994)]	<u>) </u>
244 B9	gp120(C1 93-96 LAI)	gp120(92-95)	FNMW		mis-folded LAI rgp160	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: Matthias Niedrig References: [Abacioglu et al.(1994)] NOTES:	(1994)]				
	B9: C1 region – epitope	boundaries mapped	B9: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]	glu et al.(1994)]		
245 B10	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		mis-folded LAI	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: Matthias Niedrig References: [Abacioglu et al.(1994), Moore et al.(1994c)]	(1994), Moore et al	(1994c)]			
	 B10: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abace B10: The relative affinity for denatured/native gp120 is 0.4 [Moore et al.(1994c)] B10: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. 	e boundaries mapper / for denatured/nativ // polymorphism in l	B10: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu et al.(1994)] B10: The relative affinity for denatured/native gp120 is 0.4 [Moore et al.(1994c)] B10: There is FNM/FDM polymorphism in I AI based portides and N is assential (I B Moore per com	V core [Abaciogh 1994c)] essential (J. P. M	zioglu et al.(1994)] P. Moore, per. comm.)	
246 L5.1	gp120(C1 89-103 IIIB) Donor: Matthias Niedrio	on120(78-92)	LAI-based pepudes, and in is		vaccinia on160	murino(IoC)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
247 B27	gp120(C1 94-97 BH10)	gp120(92-95)	FNMW	Z	Baculovirus- expressed mis- folded rgp 160 IIIB: NL43,	$\operatorname{murine}(\operatorname{Ig} G_1)$
	Donor: Matthias Niedrig References: [Abacioglu et al.(1994), Bristow et al.(1994)] NOTES:	1994), Bristow et	al.(1994)]		MicroGenSys	
	 B27: C1 region – epitope B27: MAbs generated in et al.(1994)] 	boundaries mappe the context of a s	B27: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)] B27: MAbs generated in the context of a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]	oglu et al.(1994)] response to rgp1	 20 and rgp160 [Bristo	0W
248 B35	gp120(C1 94-99 BH10)	gp120(92-97)	FNMWKN		mis-folded LAI	$murine(IgG_1)$
	Donor: Matthias Niedrig References: [Abacioglu et al.(1994)] NOTES:	1994)]			rgp160	
	• B35: C1 region – epitope	boundaries mappe	B35: C1 region - epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]	oglu et al.(1994)		
249 489.1(961)	gp120(C1 91-100 LAI) gg Donor: C. Bruck, SKB, Belgium References: [Moore et al.(1994c)] NOTES:	gp120(90-99) .m 4c)]	ENFDMWKNDM		Env	murine(IgG)
	• 489.1(961): The relative a • 489.1(961): NIH AIDS Re	ffinity for denaturesearch and Refer	 489.1(961): The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c] 489.1(961): NIH AIDS Research and Reference Reagent Program: 961 	al.(1994c)]		
250 T1.1	gp120(C1 91-100 LAI) Donor: C. Bruck, SKB, Belgium	gp120(90-99) .m	ENFDMWKNDM		vaccinia gp160	murine(IgG)
	References: [Akerblom et al.(1990), Broliden et al.(1990), Moore et al.(1994c)] NOTES:	1990), Broliden e	t al.(1990), Moore et al.(1994c			
	 T1.1: Also reacted in solid T1.1: No ADCC activity - T1.1: C1 region - the relationship 	I phase with gp12 - reactive peptide: tive affinity for de	T1.1: Also reacted in solid phase with gp120(234-248) NGTGPCTNVSTQCT [Akerblom et al.(1990)] T1.1: No ADCC activity – reactive peptide: NVTENFNMWKNDMVEQ, IIIB [Broliden et al.(1990)] T1.1: C1 region – the relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]	QCT [Akerblom IIIB [Broliden of the control of the	et al.(1990)] et al.(1990)]	
251 T7.1	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM		Env	murine(IgG)
	Donor: C. Bruck, SKB, Belgium References: [Akerblom et al.(1990), Bolmstedt et al.(1990), Moore et al.(1994c), Moore NOTES:	ım 1990), Bolmstedt	et al.(1990), Moore et al.(1994	к), Moore et al.(et al.(1994d)]	
	• T7.1: The relative affinity	of denatured/nati	T7.1: The relative affinity of denatured/native gp120 is 4.0 [Moore et al.(1994c)]	994c)]		

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
252 T9	gp120(C1 91-100 LAI) gp120(90-99) ENFDMW Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Akerblom et al.(1990), Bolmstedt et al.(1990), M NOTES: • T9: There appear to be two T9s • T9: The relative affinity of denatured/native gp120 is 7.9 • T9: C1 region – 45 W/S, 88 N/P, 256 S/Y, 262 N/T, 475 M, tested significantly inhibited [Moore et al.(1994d)] • T9: A low avidity antibody as assessed by urea elution	gp120(90-99) Britta Wahren and Joi L(1990), Bolmstedt et two T9s v of denatured/native g 8, 88 N/P, 256 S/Y, 262 bited [Moore et al.(19 ody as assessed by ur	gp120(C1 91-100 LAI) gp120(90-99) ENFDMWKNDM Env Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Akerblom et al.(1990), Bolmstedt et al.(1990), Moore et al.(1994c), Moore et al.(1994d), Binley et al.(1997)] NOTES: T9: There appear to be two T9s T9: The relative affinity of denatured/native gp120 is 7.9 [Moore et al.(1994c)] T9: C1 region – 45 W/S, 88 N/P, 256 S/Y, 262 N/T, 475 M/S, 485 1.83, and 491 I/F enhanced binding, no substitution tested significantly inhibited [Moore et al.(1994d)] T9: A low avidity antibody as assessed by urea elution	Moore et al.(199 c)] 91 VF enhanced	Env 4d), Binley et al.(1997)] binding, no substitution	murine(IgG)
253 5B3	gp120(C1 91-100 LAI) gp120(90-99) ENFDMWKND Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Berman et al.(1991), Nakamura et al.(1992), Beretta & NOTES: • 5B3: Blocks gp120 -CD4 binding [Berman et al.(1991)] • 5B3: Cross-blocks 1D10 in competitive IIIB-rsgp160 ELISA — localized binding to residues 72-106 [Nakamura et al.(1992)] • 5B3: The relative affinity of denatured/native gp120 is 8.3 [Mo	gp120(90-99) Britta Wahren and Joi (1991), Nakamura et a 2-4 binding [Berman e 0 in competitive IIIB-esidues 72-106 [Nakaty of denatured/native	gp120(C1 91-100 LAI) gp120(90-99) ENFDMWKNDM N IIIB-rspg160 Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Berman et al.(1991), Nakamura et al.(1992), Beretta & Dalgleish(1994), Moore et al.(1994c)] • 5B3: Blocks gp120 -CD4 binding [Berman et al.(1991)] • 5B3: Cross-blocks 1D10 in competitive IIIB-rsgp160 ELISA – no neutralization – blocks IIIB-gp120 sCD4 binding – localized binding to residues 72-106 [Nakamura et al.(1992)] • 5B3: The relative affinity of denatured/native gp120 is 8.3 [Moore et al.(1994c)]	N 1994), Moore ei tion – blocks II	IIIB-rspg160 al.(1994c)] IIB-gp120 sCD4 binding	murine(IgG)
254 MF49.1	gp120(C1 91-100 LAI) gp120(90-99) ENFDMW Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Thiriart et al.(1989), Moore et al.(1994c)] NOTES: • MF49.1: The relative affinity of denatured/native gp120 is	gp120(90-99) Britta Wahren and Jon 1989), Moore et al.(1) ffinity of denatured/na	20(C1 91-100 LAI) gp120(90-99) ENFDMWKNDM nor: Lennart Akerblom, Britta Wahren and Jorma Hinkula erences: [Thiriart et al.(1989), Moore et al.(1994c)] TES: MF49.1: The relative affinity of denatured/native gp120 is 3.8 [Moore et al.(1994c)]	(1994c)]	Env	murine(IgG)
255 B20	gp120(C1 101-110 LAI) gp120(100-109) VEQMHEDIIS Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Abacioglu et al.(1994), Moore et al.(1994c)] NOTES: B20: C1 region – epitope boundaries mapped by peptide scanning – HEDII cor	gp120(100-109) Britta Wahren and Jonal. (1994), Moore et al	20(C1 101-110 LAI) gp120(100-109) VEQMHEDIIS rgp160 or: Lennart Akerblom, Britta Wahren and Jorma Hinkula erences: [Abacioglu et al.(1994), Moore et al.(1994c)] TES: B20: C1 region – epitope boundaries mapped by peptide scanning – HEDII core [Abacioglu et al.(1994)]	core [Abaciogh	mis-folded LAI rgp160	$\operatorname{murine}(\operatorname{IgG}_{2a})$

MAb ID	Location	WEAU	Sequence Neutralizing		Immunogen	Species(Isotype)
256 B18	gp120(C1 101-110 LAI)	gp120(100-109)	VEQMHEDIIS		mis-folded LAI rgp160	$\operatorname{murine}(\operatorname{IgG}_{2a})$
	Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Abacioglu et al.(1994), Moore et al.(1994c)] NOTES: • B18: C1 region – epitope boundaries mapped by peptide • B18: The relative affinity for denatured/native gp120 is 1	ritta Wahren and Joi (1994), Moore et al. boundaries mapped for denatured/nativ	nor: Lennart Akerblom, Britta Wahren and Jorma Hinkula ferences: [Abacioglu et al.(1994), Moore et al.(1994c)] TES: B18: C1 region – epitope boundaries mapped by peptide scanning, HEDII core [Abacioglu et al.(1994)] B18: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]	acioglu e	t al.(1994)]	
257 MF39.1	gp120(C1 101-110 LAI) gp120(100-109) VEQMHEDIIS Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Thiriart et al.(1989), Cook et al.(1994), Moore et al.(1994c)] NOTES:	gp120(100-109) VEQMHEDIIS ritta Wahren and Jorma Hinkula 989), Cook et al.(1994), Moore et al.(VEQMHEDIIS rma Hinkula 94), Moore et al.(1994c)]		Env	murine(IgG)
	 MF39.1: Called 39.1, and infection of normally sus of gp120 do not inhibit glet al.(1994)] MF39.1: The relative affi 	I is probably the san ceptible CD4 negati o120 binding to Galo nity of denatured/na	MF39.1: Called 39.1, and is probably the same as MF39.1 – MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)] MF39.1: The relative affinity of denatured/native gp120 is 30 [Moore et al.(1994c)]	sphingoli Abs agaii not inhib	ipid GalCer block HIV nst the N-terminal half vit MAb binding [Cook	,
258 T2.1	gp120(C1 101-120 LAI)	gp120(100-119)	gp120(100-119) VEQMHEDIISLWDQSLK- PCV		Env	murine(IgG)
	Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Akerblom et al.(1990), Bolmstedt et al.(1990), I NOTES:	ritta Wahren and Joı (1990), Bolmstedt e	Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Akerblom et al.(1990), Bolmstedt et al.(1990), Moore et al.(1994c), Moore NOTES:	e et al.(1994d)]	994d)]	
	• T2.1: The relative affinity for binding [Moore et al.(1994c)]	y for denatured/nati	T2.1: The relative affinity for denatured/native gp120 is .27 – mutations 113 D/R, binding [Moore et al.(1994c)]		106 E/A, and 117 D/A impair	
259 11/65	gp120(311-321 HXB10)	gp120(101-120)	EQMHEDIISLWDQSLKP- CVK		rgp120 BH10	$\operatorname{rat}(\operatorname{IgG}_{2b})$
	Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [McKeating et al.(1992a)] NOTES:	ritta Wahren and Joi L(1992a)]	rma Hinkula			
	 11/65: Binds only soluble gp120, not virion bound – used to qua original?) [McKeating et al.(1992a)] 11/65: UK Medical Research Council AIDS reagent: ARP3076 	al.(1992a)] arch Council AIDS	11/65: Binds only soluble gp120, not virion bound – used to quantitate gp120 shedding – (numbering is incorrect in original?) [McKeating et al.(1992a)] 11/65: UK Medical Research Council AIDS reagent: ARP3076	ing – (nu	mbering is incorrect in	

MAb ID	Location	WEAU	Sequence No	Neutralizing	Immunogen	Species(Isotype)
260 6D8	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK- PCV		IIIB-rgp120	rat
	Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Dowbenko et al.(1988), Nakamura et al.(1992), NOTES:	ritta Wahren and Jor (1988), Nakamura e	Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula References: [Dowbenko et al.(1988), Nakamura et al.(1992), Moore et al.(1994c)] NOTES:			
	 6D8: Highly cross reactiv 6D8: The relative affinity et al.(1994c)] 	e with multiple stair for denatured/native	6D8: Highly cross reactive with multiple stains by rgp120 ELISA [Nakamura et al.(1992)] 6D8: The relative affinity for denatured/native gp120 is 15 – mutations 113 D/R and 113 D/A impair binding [Moore et al.(1994c)]	et al.(1992)] and 113 D/A	impair binding [Moore	
261 M96	gp120(C1 101-120 LAI)	gp120(100-119)	gp120(100-119) VEQMHEDIISLWDQSLK- PCV	Z	451 Env	$\mathrm{rat}(\mathrm{IgG}_{2a})$
	Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese e NOTES:	nese ese et al.(1992), Moo	Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d)] NOTES:	94d)]		
	 M96: Immunoblot reactiv M96: C1 region – the rela 	e for strains IIIB, 45 tive affinity for dena	M96: C1 region – the relative affinity for denatured/native gp120 is 6 [Moore et al.(1994c)]	Veronese et al.(1994c)]	al.(1992)]	
262 37.1.1 (ARP 327)	gp120(C1 101-120 LAI)	gp120(100-119)	gp120(100-119) VEQMHEDIISLWDQSLK- PCV		Env glycopro	murine(IgG)
	Donor: Claudine Bruck References: [Thiriart et al.(1989), Moore & Ho(1993), Moore et al.(1994c)] NOTES:	89), Moore & Ho(1	993), Moore et al.(1994c)]			
	 37.1.1: Called 37.1 – bour 37.1.1: The relative affinition 	nd preferentially to c ty for denatured/nati	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	fo(1993)] D/R (not D/A	i) and 117 K/W impair	
	 binding [Moore et al.(1994c)] 37.1.1: UK Medical Research Council AIDS reagent: ARP327 	4c)] arch Council AIDS 1	reagent: ARP327			

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
263 187.2.1	gp120(C1 101-120 LAI) gp120(100-11 Donor: Claudine Bruck and Clothilde Thiriart References: [Thiriart et al.(1989), Moore & H NOTES:	gp120(100-119) Clothilde Thiriart 989), Moore & Ho(1	gp120(C1 101-120 LAI) gp120(100-119) VEQMHEDIISLWDQSLK- Env glycopro PCV Donor: Claudine Bruck and Clothilde Thiriart References: [Thiriart et al.(1989), Moore & Ho(1993), Cook et al.(1994), Moore et al.(1994c), Moore et al.(1994d)] NOTES:	et al.(1994c), I	Env glycopro Moore et al.(1994d)]	murine(IgG)
	 187.2.1: Called 187.1, and is probably the same as 187.2.1 – bo & Ho(1993)] 187.2.1: Called 187.1, and is probably the same as 187.2.1 – MA infection of normally susceptible CD4 negative cells from the b of gp120 do not inhibit gp120 binding to GalCer – binding of GalCet et al.(1994)] 187.2.1: The relative affinity for denatured/native gp120 is 7 – binding [Moore et al.(1994c)] 187.2.1: UK Medical Research Council AIDS reagent: ARP332 	nd is probably the san id is probably the san sceptible CD4 negatip120 binding to GalC inity for denatured/nip4c)]	& Ho(1993)] & Ho(1993)] 187.2.1: Called 187.1, and is probably the same as 187.2.1 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] 187.2.1: Called 187.1, and is probably the same as 187.2.1 – MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)] 187.2.1: The relative affinity for denatured/native gp120 is 7 – mutations 113 D/A (not D/R) and 117 K/W impair binding [Moore et al.(1994c)] 187.2.1: UK Medical Research Council AIDS reagent: ARP332	ntially to denature glycosphingo on – MAbs agai 20 does not inhi l3 D/A (not D/I	denatured IIIB gp120 [Moore phingolipid GalCer block HIV bs against the N-terminal half ot inhibit MAb binding [Cook not D/R) and 117 K/W impair	
264 MF58.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK- PCV		Env	murine(IgG)
	Donor: Claudine Bruck and Clothilde Thiriart References: [Thiriart et al.(1989), Moore et al.(1994c)]	Clothilde Thiriart 989), Moore et al.(19	994c)]			
265 MF77.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK- PCV		Env	murine(IgG)
	Donor: Claudine Bruck and Clothilde Thiriart References: [Thiriart et al.(1989), Moore et al.(1994c)] NOTES: • MF77.1: The relative affinity for denatured/native g	Clothilde Thiriart 989), Moore et al.(19) inity for denatured/na	 onor: Claudine Bruck and Clothilde Thiriart eferences: [Thiriart et al.(1989), Moore et al.(1994c)] OTES: MF77.1: The relative affinity for denatured/native gp120 is 11 [Moore et al.(1994c)] 	(1994c)]		
266 MF119.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK- PCV		Env	murine(IgG)
	 Donor: Claudine Bruck and Clothilde Thiriart References: [Thiriart et al.(1989), Moore et al.(1994c)] NOTES: MF119.1: The relative affinity for denatured/native binding [Moore et al.(1994c)] 	Clothilde Thiriart 989), Moore et al.(19 finity for denatured/r 94c)]	 In cor: Claudine Bruck and Clothilde Thiriart In core et al.(1989), Moore et al.(1994c) In core et al.(1989) In core et al.(1989) In core et al.(1989) In core et al.(1980) In core et al.(1994c) In core et al		.13 D/R, and 117 K/W impair	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
267 MF4.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDIISLWDQSLK- PCV		Env	murine(IgG)
	Donor: Claudine Bruck and Clothilde Thiriart References: [Thiriart et al.(1989), Moore et al.(1994c)] NOTES:	Clothilde Thiriart 989), Moore et al.(19	994c)]			
	 MF4.1: The relative affir 	ity for denatured/nat	• MF4.1: The relative affinity for denatured/native gp120 is 8 [Moore et al.(1994c)])94c)]		
268 MF53.1	gp120(C1 101-120 LAI)	gp120(100-119)	gp120(100-119) VEQMHEDIISLWDQSLK- PCV		Env	murine(IgG)
	Donor: Claudine Bruck and Clothilde Thiriart References: [Thiriart et al.(1989), Moore et al.(1994c)] NOTES:	Clothilde Thiriart 989), Moore et al.(19	994c)]			
	• MF53.1: The relative aff	inity for denatured/na	MF53.1: The relative affinity for denatured/native gp120 is 10 [Moore et al.(1994c)]	(1994c)]		
269 GV1A8	gp120(105-113 IIIB)	gp120(104-112)	HEDIISLWD		gp120 complexed with MAb M77	murine
	Donor: Claudine Bruck and Clothilde Thiriart References: [Denisova et al.(1996)]	Clothilde Thiriart (1996)]				
	• GV1A8: When anti-V3 linear epitopes – MAbs ([Denisova et al.(1996)]	MAb M77 was bour 3V7A4 and GV5H5 a	GV1A8: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV7A4 and GV5H5 are homologous to GV1A8 and were generated in the same experiment [Denisova et al.(1996)]	munogen, it stir were generated	, it stimulated many MAbs to erated in the same experiment	1

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
273 11	gp120(C1 101-120 LAI) gp120(110-119) LWI Donor: George Lewis References: [Thiriart et al.(1989), Moore et al.(1994c)] NOTES: • 11: The relative affinity for denatured/native gp120	gp120(110-119) 989), Moore et al.(19) or denatured/native §	.0(C1 101-120 LAI) gp120(110-119) LWDQSLKPCV or: George Lewis rences: [Thiriart et al.(1989), Moore et al.(1994c)] TES: 11: The relative affinity for denatured/native gp120 is 7.8 – mutation 113 D/R impairs		Env binding [Moore et al.(1994c)]	murine(IgG)
274 12G10	gp120(C1 101-120 LAI) gp120(110-119) LWI Donor: George Lewis References: [Thiriart et al.(1989), Moore et al.(1994c)] NOTES: • 12G10: The relative affinity for denatured/native et al.(1994c)]	gp120(110-119) 989), Moore et al.(19 inity for denatured	20(C1 101-120 LAI) gp120(110-119) LWDQSLKPCV Env nor: George Lewis erences: [Thiriart et al.(1989), Moore et al.(1994c)] TES: 12G10: The relative affinity for denatured/native gp120 is 17 – mutation 117 K/W impairs binding [Moore et al.(1994c)]	117 K/W im	Env npairs binding [Moore	murine(IgG)
275 7C10	gp120(C1 101-120 LAI) gp120(110-119) LWDQSLKPCV Donor: George Lewis References: [Thiriart et al.(1989), Moore et al.(1994c)] NOTES: • 7C10: The relative affinity for denatured/native gp120 is 5.8 et al.(1994c)]	gp120(110-119) 989), Moore et al.(19 nity for denatured/	gp120(110-119) LWDQSLKPCV 9), Moore et al.(1994c)] y for denatured/native gp120 is 5.8 – mutation 117	×	Env /W impairs binding [Moore	murine(IgG)
276 W1	gp120(C1 102-121 LAI) gp Donor: D. Weiner, U. Penn. References: [Moore et al.(1994c)] NOTES: • W1: The relative affinity for de [Moore et al.(1994c)]	gp120(101-120) 94c)] or denatured/native	20(C1 102-121 LAI) gp120(101-120) EQMHEDIISLWDQSLKP- Env CVK nor: D. Weiner, U. Penn. erences: [Moore et al.(1994c)] TES: W1: The relative affinity for denatured/native gp120 is 6 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore et al.(1994c)]	113 D/R, and 1		murine(IgG)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
277 T11	(C1 102-125) gp120(101-124) EQM CVK Donor: R. Doms, Univ. of Pennsylvania References: [Earl et al.(1994), Jagodzinski et al.(1996)] NOTES: • T11: Generated during a study of the influence of t the Ab response – an oligomer with no gp120/gp41 • T11: The sulfated polysaccharide, curdlan sulfate (C virus – deletion of the V3 loop from gp120 results in	gp120(101-124) ennsylvania), Jagodzinski et al.(1 study of the influen gomer with no gp120 ccharide, curdlan sul	gp120(101-124) EQMHEDIISLWDQSLKP- rec gp140 CVKLTPL nor: R. Doms, Univ. of Pennsylvania erences: [Earl et al.(1994), Jagodzinski et al.(1996)] TES: T11: Generated during a study of the influence of the oligomeric structure of Env in determining the repertoire of the Ab response – an oligomer with no gp120/gp41 cleavage site was used as the immunogen [Earl et al.(1994)] T11: The sulfated polysaccharide, curdlan sulfate (CRDS), binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop from gp120 results in more potent T11 inhibition by CRDS [Jagodzinski et al.(1996)]		rec gp140 rec gp140 1 determining the repertoire of nunogen [Earl et al.(1994)] 1-tropic viruses and neutralizes RDS [Jagodzinski et al.(1996)]	murine
278 B33	gp120(V2 123-142 LAI)	gp120(122-146)	TPLCVSLKCTDLGNATN- TNS	Z	Baculovirus- expressed mis- folded rgp160 IIIB:NL43, MicroGenSys	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_{2b\kappa})$
	 Donor: Daniels References: [Abacioglu et al.(1994), Bristow et al.(1994)] NOTES: B33: There are two MAbs in the literature named B33 B33: MAbs generated in the context of a study of the et al.(1994)] B33: UK Medical Research Council AIDS reagent: A 	.(1994), Bristow et a sin the literature na the context of a str	nor: Daniels 'erences: [Abacioglu et al.(1994), Bristow et al.(1994)] TES: B33: There are two MAbs in the literature named B33. See also gp41, LAI 123-142 [B33: MAbs generated in the context of a study of the humoral immune response to et al.(1994)] B33: UK Medical Research Council AIDS reagent: ARP304, gp160/41 binding]	[Abacioglu et al.(1994)] o rgp120 and rgp160 [Bristow	
279 6D5	gp120(V2 122-141 LAI)	gp120(121-145)	gp120(121-145) LTPLCVSLKCTDLKNDT- NTN		Env	murine(IgG)
	 Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA References: [Moore et al.(1994c), Moore et al.(1994d)] NOTES: 6D5: The relative affinity for denatured/native gp120 [Moore et al.(1994c)] 	nur, NCI, Frederick, 194c), Moore et al.(194c) for denatured/nati	 nor: S. Nigida and L. Arthur, NCI, Frederick, MD USA erences: [Moore et al.(1994c), Moore et al.(1994d)] TES: 6D5: The relative affinity for denatured/native gp120 is 15 – mutations Δ119-205 and 125 L/G impair binding [Moore et al.(1994c)] 	∆119-205 and 1	25 L/G impair binding	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen
280 C108G	gp120(V2 162-169 HXB2) gp120(166-173) STSIRGKV L IIIB infection Donor: S. Tilley, Public Health Research Institute, NY, NY References: [Warrier et al.(1994), Wu et al.(1995), Warrier et al.(1995), Ugolini et al.(1997), Mondor et al.(1998), Alsmadi & Tilley(1998)] NOTES:	gp120(166-173) h Research Institute 94), Wu et al.(1995), (1998)]	STSIRGKV .e, NY, NY), Warrier et al. (1995), Warri	L eretal.(1996), Ugol	IIIB infection ini et al.(1997), Mondo
	 C108G: High affinity, potent neutralization of HIV-1 IIIB – binding disrupted by removal of N-linked glycans – peptide et al.(1994)] C108G: Strain specificity: LAI, Bal, HXB2 – conformationa of conserved glycosylation site at 156 increased epitope exp • C108G: Characterization of MAb variable region [Warrier e C108G: Synergistic neutralization of HIV-1 when combined MAbs, 1125H and 5145A – neutralization further enhanced b • C108G: Viral binding inhibition by C108G was correlated showed some correlation except 2F5) [Ugolini et al.(1997)] • C108G: Inhibits HX10 binding to both CD4 positive and ne. • C108G: A study of 6 anti-Env MAbs and their ability to bind MN, SF-2, and RF – bound and directed lysis against only I V2 specific MAb [Alsmadi & Tilley(1998)] 	cent neutralization of oval of N-linked gl LAI, Bal, HXB2 – n site at 156 increase of MAb variable realization of HIV-1 – neutralization furuibition by C108G except 2F5) [Ugolianding to both CD4 Env MAbs and the dand directed lysis if & 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 et al.(1994)] C108G: Strain specificity: LAI, Bal, HXB2 – conformational character – glycosylation site at 160 critical – mutation of conserved glycosylation site at 156 increased epitope exposure [Wu et al.(1995)] C108G: Characterization of MAb variable region [Warrier et al.(1995)] C108G: Synergistic neutralization of HIV-1 when combined with anti-V3 MAbs 0.5β and C311E, or anti-CD4BS MAbs, 1125H and 5145A – neutralization further enhanced by presence of both 1125H and 0.5β [Warrier et al.(1996)] C108G: Viral binding inhibition by C108G was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)] C108G: Inhibits HX10 binding to both CD4 positive and negative HeLa cells[Mondor et al.(1998)] C108G: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against only IIIB – this is first demonstration of ADCC directed by a V2 specific MAb [Alsmadi & Tilley(1998)]	affected by reduct ver affinity than gly glycosylation site a glycosylation site a al.(1995)] 13 MAbs 0.5β and both 1125H and 0. ization (all other not cells[Mondor et al. DCC against target first demonstration	reduction of disulfide bonds – han glycosylated Env [Warrier n site at 160 critical – mutation β and C311E, or anti-CD4BS and 0.5β [Warrier et al.(1996)] other neutralizing MAbs tested or et al.(1998)] target cells infected with IIIB, tration of ADCC directed by a
281 10/76b	gp120(V2 162-171 BH10) gp120(166-174) STSIRG Donor: S. Tilley, Public Health Research Institute, NY, NY References: [McKeating et al.(1993b), McKeating et al.(1996)]	gp120(166-174) STSIRGKVQ th Research Institute, NY, NY al.(1993b), McKeating et al.(1993a	12-171 BH10) gp120(166-174) STSIRGKVQ L (HXF. Iley, Public Health Research Institute, NY, NY [McKeating et al.(1993b), McKeating et al.(1993a), Shotton et al.(1995),	\sim	10) BH10 rgp120 Wu et al.(1995), McKeating
	 10/76b: R to L substitution abrogated binding – human sera recognition in the properties of the properties with MAbs 10/76b and 11/4b – HXB2 mat residue 165 [Shotton et al.(1995)] 10/76b: Included in cross-competition and neutralization studies 10/76b: HX10 strain specificity – binds native, deglycosylated, or 10/76b: Neutralizes HXB2, but fails to neutralize chimeric virubackground [McKeating et al.(1996)] 10/76b: UK Medical Research Council AIDS reagent: ARP3077 	n abrogated bindin, with MAbs 10/76b; al.(1995)] -competition and notificity – binds nativ 32, but fails to neust al.(1996)] earch Council AID:	10/76b: R to L substitution abrogated binding – human sera recognize epitope [McKeating et al.(1993b)] 10/76b: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the subst at residue 165 [Shotton et al.(1995)] 10/76b: Included in cross-competition and neutralization studies [Shotton et al.(1995)] 10/76b: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)] 10/76b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in background [McKeating et al.(1996)] 10/76b: UK Medical Research Council AIDS reagent: ARP3077	pitope [McKeating ation escape mutan ation escape mutan n et al.(1995)] rred gp120 [Wu et a gp120 from prima	eating et al.(1993b)] mutant has the substitution I/T [[a] [[b] [[b] [[b] [[b] [[c] [[c] [[c] [[c

	286 6C4/S	285 RSD-33		284 11/4b		283 11/41e		282 11/4c	MAb ID
• 6C4/S: UK Medica	gp120(V2 162-170 BH10) gp Donor: S. Ranjbar (NIBSC, UK) References: [Moore et al.(1993a)] NOTES:	gp120(V2 162-171 BH10) gp Donor: R. Daniels (NIMR, UK) References: [Moore et al.(1993a)]	 11/4b: A change from R to L abroga 11/4b: Cross-competes with MAbs 1 at residue 165 [Shotton et al.(1995)] 11/4b: HXB10 strain specificity – bi 11/4b: Linear V2 epitope – reciproca to BAT085) and CD4 inducible antibodoes not inhibit 11/4b [Moore & Sod 	gp120(V2 162-171) Donor: S. Tilley, Public References: [McKeatir NOTES:	 11/41e: R to L abro 11/41e: Included in 11/41e: HX10 strain 	gp120(V2 162-171) Donor: S. Tilley, Public References: [McKeatir NOTES:	 11/4c: R to L substitution abrogated 11/4c: HX10 strain specificity – bine 11/4c: Cross-competes with MAbs 1 at residue 165 [Shotton et al.(1995)] 11/4c: UK Medical Research Counc 	gp120(V2 162-171) Donor: S. Tilley, Public References: [McKeatir NOTES:	D Location
6C4/S: UK Medical Research Council AIDS reagent: ARP3049	gp120(166-173) ;, UK) 1993a)]	10) gp120(166-174) STSIRGKVQ MR, UK) al.(1993a)]	11/4b: A change from R to L abrogated binding – human sera recognize epitope [McKeating et al.(1993b)] 11/4b: Cross-competes with MAbs 10/76b and 11/4c – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton et al.(1995)] 11/4b: HXB10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)] 11/4b: Linear V2 epitope – reciprocal binding enhancement of anti-V2 discontinuous epitope antibodies (in contrast to BAT085) and CD4 inducible antibody 48d. Reciprocal inhibits BAT085 binding – inhibits CRA-3 binding CRA-3 does not inhibit 11/4b [Moore & Sodroski(1996)]	gp120(V2 162-171) gp120(166-174) STSIRGKVQ L (HXI Donor: S. Tilley, Public Health Research Institute, NY, NY References: [McKeating et al.(1993b), Shotton et al.(1995), Wu et al.(1995), Moore & S. NOTES:	11/41e: R to L abrogated binding – human sera recognize the epitope [McKeating et al.(19 11/41e: Included in cross-competition and neutralization studies [Shotton et al.(1995)] 11/41e: HX10 strain specificity – binds native and deglycosylated gp120 [Wu et al.(1995)]	gp120(V2 162-171) gp120(166-174) STSIRGKVQ Donor: S. Tilley, Public Health Research Institute, NY, NY References: [McKeating et al.(1993b), Shotton et al.(1995), Wu et al.(1995)] NOTES:	11/4c: R to L substitution abrogated binding – human sera recognize epitope [McKeating et al.(1993b)] 11/4c: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)] 11/4c: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton et al.(1995)] 11/4c: UK Medical Research Council AIDS reagent: ARP3035	gp120(V2 162-171) gp120(166-174) STSIRGKVQ Donor: S. Tilley, Public Health Research Institute, NY, NY References: [McKeating et al.(1993b), Wu et al.(1995), Shotton et al.(1995)] NOTES:	WEAU
eagent: ARP3049	STSIRGKV	STSIRGKVQ	ig – human sera recognize of 111/4c – HXB2 neutralizated, deglycosylated, or denatuenhancement of anti-V2 dia Reciprocal inhibits BAT085 [6]]	STSIRGKVQ NY, NY al.(1995), Wu et al.(1995),	a recognize the epitope [Mutralization studies [Shotton and deglycosylated gp120	STSIRGKVQ NY, NY al.(1995), Wu et al.(1995)]	human sera recognize epit deglycosylated, or denature 1 11/4b – HXB2 neutralizat agent: ARP3035	STSIRGKVQ , NY, NY 995), Shotton et al.(1995)]	Sequence
			pitope [McKeatiion escape mutan red gp120 [Wu et continuous epitop binding – inhibit			L (HXB10)	ope [McKeating e d gp120 [Wu et a ion escape mutan	L (HXB2)	Neutralizing
	BH10 gp120	BH10 gp120	Keating et al.(1993b)] mutant has the substitution I/T wu et al.(1995)] epitope antibodies (in contrast hhibits CRA-3 binding CRA-3	310) rgp120 LAI:BH10 odroski(1996)]	al.(1993b)])] 995)]	rgp120 LAI:BH10	at al.(1993b)] L.(1995)] It has the substitution I/	BH10 rgp120	Immunogen
			. Ω	$\operatorname{rat}(\operatorname{IgG}_{2a})$		$\operatorname{rat}(\operatorname{IgG}_1)$	H	$\operatorname{rat}(\operatorname{IgG}_{2a})$	Species(Isotype)

	287 G3-4	MAb]
References: [Ho et al.(1991a et al.(1993), Sattentau et al.	gp120(V2 170-180 BH10) gp120(174-184) QKEYA Donor: Tanox Biosystems Inc and David Ho, ADARC, NY	MAb ID Location
a), Ho et al.(1992), F (1993), Thali et al.(gp120(174-184) nc and David Ho, Al	WEAU
References: [Ho et al.(1991a), Ho et al.(1992), Fung et al.(1992), McKeating et al.(1992a), et al.(1993), Sattentau et al.(1993), Thali et al.(1993), Moore et al.(1993a), Moore et al.	gp120(174-184) QKEYAFFYKLD Inc and David Ho, ADARC, NY	Sequence
et al.(1992a), Moo Moore et al.(199	L	Neutralizing
, Moore & Ho(1993), Sullivar .(1994b), Gorny et al.(1994)	IIIB gp120	Immunogen
m),	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_{2b\kappa})$	Species(Isotype)

NOTES:

Parren et al.(1998)]

- G3-4: Also called G3.4
- G3-4: Binding is sensitive to removal of glycans by endo H 50% neutralization of 4/9 primary isolates has conformational features [Ho et al.(1991a)]

Sodroski (1996), Poignard et al. (1996a), Binley et al. (1997), Stamatatos et al. (1997), Ditzel et al. (1997), Wyatt et al. (1997),

Thali et al.(1994), Yoshiyama et al.(1994), Wu et al.(1995), Sattentau & Moore(1995), Jagodzinski et al.(1996), Moore &

- G3-4: Neutralizes IIIB and RF, not MN blocks sCD4-gp120, not as potent as MAb 15e V2 binding MAbs BAT085
- G3-4: Substitutions in residues 176 to 184 affect MAb recognition substitutions in V2 can result in gp120-gp41 and G3-136 block G3-4 gp120 binding – sensitive to reduction of gp120 by DTT [Ho et al.(1992)]
- G3-4: Increased binding in the presence of sCD4 [Sattentau et al.(1993)]

dissociation [Sullivan et al.(1993)]

- G3-4: Conformational, does not bind well to denatured gp120 not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- G3-4: V2 region, marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore et al.(1993a)]
- G3-4: Conformationally sensitive sporadic cross-reactivity among, and outside, B clade gp120s [Moore et al.(1994b)]
- G3-4: Weakly neutralizing, $IC_{50} = 53 \mu g/ml$ [Gorny et al.(1994)]
- G3-4: gp41 mutation (582 A/T) that reduces neutralization of anti-CD4 binding site MAbs does not alter G3-4s ability to neutralize [Thali et al.(1994)]

MAb ID	
Location	
WEAU	
Sequence	
Neutralizing	
Immunogen	
Species(Isotype)	

28 / cont.

NOTES:

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

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
288 BAT085	gp120(V2 170-180 IIIB)	gp120(175-184) KEYAFFYKLD	KEYAFFYKLD	L	Inact IIIB	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY	c and David Ho, AL)ARC, NY			
	References: [Fung et al.(1987), Fung et al.(1992), Moore & Ho(1993), Pirofski et al.(1993)	7). Fung et al.(1992), Moore & Ho(1993), Piro	ifski et al.(1993). T	3). Thali et al.(1993). Moor	ore

et al.(1993a), D'Souza et al.(1994), Moore et al.(1994d), Gorny et al.(1994), Yoshiyama et al.(1994), Wu et al.(1995),

Sattentau & Moore (1995), Moore & Sodroski (1996), Poignard et al. (1996a), Binley et al. (1997), Ditzel et al. (1997),

NOTES:

Parren et al.(1998)]

- BAT085: Also called BAT-085
- BAT085: V2 region sCD4 does not block neutralizes IIIB and some primary isolates, but not MN or RF binds MN - deglycosylation or DDT reduction of gp120 does not diminish reactivity [Fung et al.(1992)]
- BAT085: Called BAT-85 conformational, does not bind well to denatured gp120 not reactive with SF-2 gp120 and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- BAT085: 7/8 V2 murine MAbs required gp120 native structure to bind, but BAT085 was the exception type-specific [Moore et al.(1993a)]
- BAT085: Peptide affinities of G3-136 and G3-4 are 100-fold less than BAT085, but BAT085 has lower affinity for BH10 gp120 and is weaker at neutralization [Moore et al.(1993a)]
- BAT085: Multi-lab study for antibody characterization and assay comparison did not bind MN or SF2 [D'Souza
- BAT085: Interacts with two overlapping peptides with region of overlap KEYAFFYKLD [Gorny et al. (1994)]
- BAT085: Neutralizes RF substitution 177 Y/H in the V2 loop of RF does not inhibit neutralization, in contrast to MAbs G3-4 and SC258 [Yoshiyama et al.(1994)]
- BAT085: HXB10 strain specificity binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)]
- BAT085: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 neutralizes cell free Hx 10 [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 contrast to anti-V3 MAbs [Poignard et al.(1996a)] BAT123 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50-69, in
- BAT085: An antibody with moderate avidity as assessed by urea elution
- BAT085: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]

	289 G3-136 gp Do Re Yo Sta N (MAb ID Lo
gp120(V2 172-191 HXB2) gp120(176-195) EYAFF' TSY Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: (Wit et al. (1995))	pp120(V2 170-180 IIIB) gp120(174-184) QKEYAFFYKLD L purified IIIB gp120 Donor: Tanox Biosystems Inc and David Ho, ADARC, NY Yoshiyama et al.(1994), Sattentau & Moore (1995), Moore & Sodroski(1996), Poignand et al.(1993), Moore et al.(1997), Stamatatos et al.(1997), Ditzel et al.(1997), Wyatt et al.(1997), Parren et al.(1998)] NOTES: • G3-136: V2 region – binds and neutralizes IIIB and RF in CEM-SS cells, but not MN – neutralization activity against a few primary isolates in PBMC – sCD4 binding inhibits binding (contrast with BAT085) – deglycosylation or reduction of gp120 by DTT diminishes reactivity [Fung et al.(1992)] • G3-136: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore et al.(1993a)] • G3-136: Marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore et al.(1993a)] • G3-136: Marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore et al.(1993a)] • G3-136: Marginal binding to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)] • G3-136: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)] • G3-136: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)] • G3-136: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)] • G3-136: Galed G3.136 – does not mediate gp120 gp120 point of the gp41 pinope of MAb 50-69, in contrast to anti-V2 MAbs [Poignard et a	Location
gp120(176-195) , and David Ho, ADA	gp120(174-184) QKEYAFFYKLD and David Ho, ADARC, NY 2), Pirofski et al.(1993), Thali et al.(1997), Pirofski et al.(1995), Moore & Sodroski et al.(1997), Wyatt et al.(1997), Parren ds and neutralizes IIIB and RF in CE ates in PBMC – sCD4 binding inhibits DTT diminishes reactivity [Fung et al.(1997)] to peptide, binding inhibited by 183/1 to peptide, binding inhibited by 183/1 dy selected antibodies to C1, C4, C5 and anti-CD4 binding site MAbs [Mostitutions 177 Y/H and 179 L/P in the V2 ially to the monomeric rather than olig (1995)] e as STSIRGKVKEYAFFYKLDI – bin cantly alter gp120 dissociation from vi [Poignard et al.(1996a)] fibody as assessed by urea elution – does not mediate gp120 virion diss SF128A in either primary macrophages) and soluble gp120+gp41 complex effial.(1997)] ab binding to the oligomeric form of galization is determined by the fraction of galization is determined by the	WEAU
EYAFFYKLDIIPIDNDT- TSY ARC, NY	purified IIIB gp120(174-184) QKEYAFFYKLD L purified IIIB gp120 for: Tanox Biosystems Inc and David Ho, ADARC, NY forences: [Fung et al.(1992), Pirofski et al.(1993), Thali et al.(1993), Moore & Ho(1993), Moore et al.(1993a), hiyama et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), natatos et al.(1997), Ditzel et al.(1997), Wyatt et al.(1997), Parren et al.(1998)] TES: G3-136: V2 region – binds and neutralizes IIIB and RF in CEM-SS cells, but not MN – neutralization activity against a few primary isolates in PBMC – sCD4 binding inhibits binding (contrast with BAT085) – deglycosylation or reduction of gp120 by DTT diminishes reactivity [Fung et al.(1992)] G3-136: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)] G3-136: Marginal binding to Epitide, binding inhibited by 183/184 PI/SG substitution [Moore et al.(1993a)] G3-136: Marginal binding to Epitide, binding site MAbs [Moore et al.(1993a)] G3-136: HIV-1 RFV2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity [Yoshiyama et al.(1993a)] G3-136: Bonda preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)] G3-136: Bonda preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)] G3-136: Allow avidity antibody as assessed by urea elution G3-136: Allow avidity antibody as assessed by urea elution G3-136: Binding to SF162 or SF128A in either primary macrophages or PBMC [Stamatatos et al.(1997)] G3-136: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	Sequence
	L L Ore & Ho(1993) ignard et al.(1998) ignard et al.(1998) ignard et al.(1998) ignard et al.(1999) s, but not MN – contrast with BAT ve with SF-2 gp1 ubstitution [Moc nnti-CD4 binding 1993a)] reduce affinity [reduce affinity	Neutralizing
BH10 gp120	purified IIIB gp120 (1993), Moore et al.(1993a), Ll.(1996a), Binley et al.(1997), MN – neutralization activity th BAT085) – deglycosylation [Moore et al.(1993a)] sinding site MAbs – enhances finity [Yoshiyama et al.(1994)] gp120 – neutralizes cell free 1g of V2 MAbs G3-136, G3-4941 epitope of MAb 50-69, in to anti-V2 MAb G3-4 – not tos et al.(1997)] s gp120 epitope is not blocked ation were highly correlated – on a virion irrespective of the	Immunogen
rat	murine(IgG)	Species(Isotype)

293 3	292 1:		291 6	7
38/60b	12b		697-D	MAb ID
gp120(V2 17 Donor: Cella References: NOTES: • 38/60b:	gp120(V2 162-181) Donor: Cellular Pro References: [Shotto NOTES: • 12b: V2 MAb 1 [Shotton et al.(] • 12b: Neutralize ground [McKea	References: [Goret al.(1997), Parret al.(1997), Parret NOTES: • 697-D: Also ce 697-D: Confo isolates, but ne YSL/GSS abret moieties inhille 697-D: Not ne 697-D: Reviere 697-D: Partial et al.(1996a)] • 697-D: Study with oligomer 697D: Does ne 697D: Does ne 697D: Does ne 697-D: Study	gp120(V2 161-180 IIIB)	Location
gp120(V2 172-191 HXB2) gp120(17) Donor: Cellular Products Inc, Buffalo NY References: [Wu et al.(1995)] NOTES: • 38/60b: Strain specificity: HXB2 – bi	gp120(V2 162-181) Donor: Cellular Products Inc, Buffalo NY References: [Shotton et al.(1995), McKea NOTES: • 12b: V2 MAb neutralized HXB2 – pc [Shotton et al.(1995)] • 12b: Neutralizes HXB2, but fails to ne ground [McKeating et al.(1996)]	Donor: Cellular Products Inc, Buffalo NY References: [Gorny et al.(1994), Forthal et et al.(1997), Parren et al.(1997b)] NOTES: • 697-D: Also called 697D • 697-D: Conformational with weak read isolates, but none of 4 lab strains – V2 YSL/GSS abrogate binding – anti-C4 moieties inhibits binding [Gorny et al. 697-D: Not neutralizing, no ADCC ac • 697-D: Review: called 697/30D – neu • 697-D: Partial inhibition of gp120 et al.(1996a)] • 697-D: A low avidity antibody as asse • 697-D: Study shows neutralization is with oligomeric Env binding – 697-D1	1-180 IIIB)	
gp120(176-195) c, Buffalo NY)] v: HXB2 – binds na	gp120(V2 162-181) gp120(166-185) LDI Donor: Cellular Products Inc, Buffalo NY References: [Shotton et al.(1995), McKeating et al.(1996)] NOTES: 12b: V2 MAb neutralized HXB2 – position 179-180 L [Shotton et al.(1995)] 12b: Neutralizes HXB2, but fails to neutralize chimeri ground [McKeating et al.(1996)]	or: Cellular Products Inc, Buffalo NY erences: [Gorny et al.(1994), Forthal et al.(1995), Moore & .(1997), Parren et al.(1997b)] FES: 697-D: Also called 697D 697-D: Conformational with weak reactivity to V2 peptide isolates, but none of 4 lab strains – V2 substitutions 176/1 YSL/GSS abrogate binding – anti-C4 MAbs G3-536 and (moieties inhibits binding [Gorny et al.(1994)] 697-D: Not neutralizing, no ADCC activity, and no viral e 697-D: Review: called 697/30D – neutralizes some primat 697-D: Partial inhibition of gp120 interaction with C et al.(1996a)] 697-D: A low avidity antibody as assessed by urea elution 697-D: Study shows neutralization is not predicted by M with oligomeric Env binding – 697-D bound monomer, did 697D: Does not neutralize TCLA strains but neutralizes so	gp120(165-184)	WEAU
195) EYAFFY TSY s native and de	185) STSIRG LDI 1g et al.(1996)] tion 179-180 LI ttralize chimeric	l.(1995), Moore l.(1995), Moore vity to V2 pepti ubstitutions 176 Abs G3-536 an 994)] vity, and no vira alizes some printeraction with ed by urea elution teraction with ed by urea for predicted by und monomer, of but neutralizes some printeraction with the control of the cont		Sequence
20(V2 172-191 HXB2) gp120(176-195) EYAFFYKLDIIPIDNDT- TSY INV INV INV INV INV INV INV IN	STSIRGKVQKEYAFFYK-LDI(1996)] /9-180 LD to DL abrogates chimeric virus with gp120	rerences: [Gorny et al.(1994), Forthal et al.(1995), Moore & Ho(1995), Trkola et al.(1996a), Binley et al.(1997) res. (1997), Parren et al.(1997b)] res. 697-D: Also called 697D 697-D: Conformational with weak reactivity to V2 peptide ISTSIRGKVQKEYAFFYKLD – neutralized 3/4 p isolates, but none of 4 lab strains – V2 substitutions 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 19 YSL/GSS abrogate binding – anti-C4 MAbs G3-536 and G45-60 enhance binding – mild oxidation of carbol moieties inhibits binding [Gorny et al.(1994)] 697-D: Not neutralizing, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] 697-D: Review: called 697/30D – neutralizes some primary, but not lab adapted strains [Moore & Ho(1995)] 697-D: Partial inhibition of gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [et al.(1995a)] 697-D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is assowith oligomeric Env binding – 697-D bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997b)] 697D: Does not neutralize TCLA strains but neutralizes some primary isolates weakly [Parren et al.(1997b)]	ISTSIRGKVQKEYAFFY- KLD	e
) [Wu et al.(1995]	L (HXB10) binding – compe from primary isc	la et al.(1996a), E EYAFFYKLD – 80 LD/DL, 183/ binding – mild ox y [Forthal et al.(1) apted strains [Mα -1β-CCR-5 con P-1β-CCR-5 con er or neutralize J ates weakly [Pan	P (weak)	Neutralizing
BH10 gp120	or: Cellular Products Inc, Buffalo NY rences: [Shotton et al.(1995), McKeating et al.(1996)] 12b: V2 MAb neutralized HXB2 – position 179-180 LD to DL abrogates binding – competes with 60b, but not 74 [Shotton et al.(1995)] 12b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]	 Donor: Cellular Products Inc, Buffalo NY References: [Gorny et al.(1994), Forthal et al.(1995), Moore & Ho(1995), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997), Parren et al.(1997b)] NOTES: 697-D: Also called 697D 697-D: Conformational with weak reactivity to V2 peptide ISTSIRGKVQKEYAFFYKLD – neutralized 3/4 primary isolates, but none of 4 lab strains – V2 substitutions 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS abrogate binding – anti-C4 MAbs G3-536 and G45-60 enhance binding – mild oxidation of carbohydrate moieties inhibits binding [Gorny et al.(1994)] 697-D: Not neutralizing, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] 697-D: Review: called 697/30D – neutralizes some primary, but not lab adapted strains [Moore & Ho(1995)] 697-D: Partial inhibition of gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)] 697-D: Study shows neutralization is not predicted by urea elution 697-D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 697-D bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)] 697D: Does not neutralize TCLA strains but neutralizes some primary isolates weakly [Parren et al.(1997b)] 	HIV-1 infection	Immunogen
rat	$\operatorname{rat}(\operatorname{IgG}_{2a})$ 74	ary ary ola ted 7)]	$\operatorname{human}(\operatorname{IgG}_{1\lambda})$	Species(Isotype)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
294 60b	gp120(V2 172-181 HXB2) gp120(17) Donor: Cellular Products Inc, Buffalo NY References: [Shotton et al.(1995)] NOTES: • 60b: V2 MAb did not neutralize HXF YSL/GSS abrogate binding, as do cha	gp120(176-185) Buffalo NY 95)] tralize HXB2 – bou g, as do changes ou	20(V2 172-181 HXB2) gp120(176-185) EYAFFYKLDI N nor: Cellular Products Inc, Buffalo NY 'erences: [Shotton et al.(1995)] TES: 60b: V2 MAb did not neutralize HXB2 – bound to rgp120 in ELISA – substitutions YSL/GSS abrogate binding, as do changes outside the minimum epitope – competes		BH10 rgp120 179-180 LD/DL and 191-193 with 12b, but not 74 [Shotton	$\operatorname{rat}(\operatorname{IgG}_{2b})$
295 74	gp120(V2 172-181) gp120(176- Donor: Cellular Products Inc, Buffalo NY References: [Shotton et al.(1995)] NOTES: • 74: V2 MAb did not neutralize HXB2 – as do changes outside the minimum epite dependent MAbs [Shotton et al.(1995)]	gp120(176-185) Buffalo NY 95)] alize HXB2 – did nc ninimum epitope – d et al.(1995)]	20(V2 172-181) gp120(176-185) EYAFFYKLDI N BH10 rgp120 nor: Cellular Products Inc, Buffalo NY erences: [Shotton et al.(1995)] TES: 74: V2 MAb did not neutralize HXB2 – did not bind rgp120 ELISA – position 179-180 LD to DL abrogates binding, as do changes outside the minimum epitope – does not compete with 60b or 12b, and is enhanced by two conformation dependent MAbs [Shotton et al.(1995)]	N n 179-180 LD to b, and is enhanc	BH10 rgp120 30 LD to DL abrogates binding, enhanced by two conformation	$\operatorname{rat}(\operatorname{IgG}_1)$
296 1088	gp120(V2) gp120 Donor: Cellular Products Inc, Buffalo NY References: [Berman et al.(1997)] NOTES: • 1088: Binds weakly to 2/7 isolates fro	gp120 Buffalo NY 97)] 'isolates from break	gp120 20(V2) nor: Cellular Products Inc, Buffalo NY erences: [Berman et al.(1997)] TES: 1088: Binds weakly to 2/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)]	20 vaccine trial	[Berman et al.(1997)]	
297 3D3.B8	gp120(211-220 LAI) gp120(215-225) EPIPIF Donor: Cellular Products Inc, Buffalo NY References: [Bolmstedt et al.(1990), Moore et al.(1994c)] NOTES: • 3D3.B8: The relative affinity denatured/native gp120	gp120(215-225) Buffalo NY 1990), Moore et al.(20(211-220 LAI) gp120(215-225) EPIPIHYCAPA tor: Cellular Products Inc, Buffalo NY erences: [Bolmstedt et al.(1990), Moore et al.(1994c)] TES: 3D3.B8: The relative affinity denatured/native gp120 is greater than 10 [Moore et al.] Dre et al.(1994c)]	Env glycopro	murine(IgG)
298 4C11.D8	gp120(211-220 LAI) gp120(215-225) EPIPIH Donor: Cellular Products Inc, Buffalo NY References: [Bolmstedt et al.(1990), Moore et al.(1994c)] NOTES: • 4C11.D8: The relative affinity denatured/native gp120	gp120(215-225) Buffalo NY 1990), Moore et al.(onor: Cellular Products Inc, Buffalo NY eferences: [Bolmstedt et al.(1990), Moore et al.(1994c)] OTES: 4C11.D8: The relative affinity denatured/native gp120 is greater than 10 [Moore et al.	E pore et al.(1994c)]	Env glycopro	murine(IgM)
299 322-151	gp120(201-220 LAI) gp120(215-225) EPII Donor: G. Robey, Abbot Labs References: [Moore et al.(1994c), Moore et al.(1994d)] NOTES: • 322-151: The relative affinity denatured/native gp1.	gp120(215-225) 4c), Moore et al.(19 ity denatured/natiw	20(201-220 LAI) gp120(215-225) EPIPIHYCAPA 1007: G. Robey, Abbot Labs 110994c, Moore et al.(1994d) 1111 1112 1112 1112 1112 1112 1112 11	94c)]	Env glycopro	murine(IgG)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
300 110.1	gp120(200-217) gp120(216-225) PIPIHYC Donor: G. Robey, Abbot Labs References: [Pincus & McClure(1993), Pincus et al.(1996), NOTES: • 110.1: There is another antibody with this ID that binds to 110.1: A panel of immunotoxins were generated by lin killing, but killing was not directly proportional to binding no effect [Pincus & McClure(1993), Pincus et al.(1996)]	gp120(216-225) abs Clure(1993), Pincus et antibody with this ID antibody with the ID	gp120(200-217) gp120(216-225) PIPIHYCAPA? N Env glycoprotein Donor: G. Robey, Abbot Labs References: [Pincus & McClure(1993), Pincus et al.(1996), Valenzuela et al.(1998)] NOTES: • 110.1: There is another antibody with this ID that binds to Env at positions 491-500 in LAI, see [Gosting et al.(1987)] • 110.1: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding – 110.1-RAC did not mediate cell killing, and sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)]	N 998)] 91-500 in LAI, so ricin A – immud not mediate ce	Env glycoprotein AI, see [Gosting et al.(1987)] immunotoxins mediated cell ate cell killing, and sCD4 has	human
301 493-156	gp120(211-230 LAI)	gp120(215-234)	EPIPIHYCAPAGFAILK- CNN		Env glycopro	murine(IgG)
	Donor: G. Robey, Abbot LabsReferences: [Moore et al.(1994c)]NOTES:493-156: The relative affinity	abs [994c)] Iffinity denatured/nativ	 onor: G. Robey, Abbot Labs eferences: [Moore et al.(1994c)] OTES: 493-156: The relative affinity denatured/native gp120 is >10 [Moore et al.(1994c)] 	1994c)]		
302 GV4H3	gp120(219-226 IIIB)	(223-230)	APAGFAIL		gp120 complexed with MAb M77	murine
	 Donor: G. Robey, Abbot Labs References: [Denisova et al.(1996)] NOTES: GV4H3: When anti-V3 MAb M77 w. linear epitopes [Denisova et al.(1996)] 	abs l.(1996)] 3 MAb M77 was bour va et al.(1996)]	nor: G. Robey, Abbot Labs ferences: [Denisova et al.(1996)] YTES: GV4H3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, linear epitopes [Denisova et al.(1996)]		it stimulated many MAbs to	
303 J1	gp120(222-231 LAI) gp120(226-235) GFAILKCNNK Donor: J. Hoxie, U. Penn. References: [Moore et al.(1994c), Moore et al.(1994d), Cook et al.(1994)] NOTES:	gp120(226-235) (1994c), Moore et al.(19	GFAILKCNNK 994d), Cook et al.(1994)]		peptide	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_1)$
	 J1: The relative affinity denatured/native gp120 is 30 [Moore et al.(1994c)] J1: MAbs against the glycosphingolipid GalCer block HIV infection of nor from the brain and colon – MAbs against the N-terminal half of gp120 do n binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)] 	denatured/native gp1: glycosphingolipid Gal on – MAbs against th o120 does not inhibit N	J1: The relative affinity denatured/native gp120 is 30 [Moore et al.(1994c)] J1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]	ormally suscept onot inhibit gp1	ble CD4 negative cells 20 binding to GalCer –	

MAb ID	Location	WEAU	Sequence Neu	Neutralizing	Immunogen	Species(Isotype)
304 J3	gp120(222-231 LAI) gp120(226-235) Gi Donor: J. Hoxie, U. Penn. References: [Moore et al.(1994c), Cook et al.(1994)] NOTES:	gp120(226-235) 94c), Cook et al.(199	GFAILKCNNK 94)]		peptide	$murine(IgG_1)$
	 J3: The relative affinity of J3: MAbs against the glassing from the brain and color binding of GalCer to gp1 	lenatured/native gp12 ycosphingolipid Gal n – MAbs against the 20 does not inhibit N	J3: The relative affinity denatured/native gp120 is 30 [Moore et al.(1994c)] J3: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]	lly suscepti nhibit gp12	ble CD4 negative cells 20 binding to GalCer –	
305 MF87.1	gp120(242-261 LAI) gp120(256-265) RPV Donor: J. Hoxie, U. Penn. References: [Thiriart et al.(1989), Moore et al.(1994c)]	gp120(256-265) 989), Moore et al.(19	RPVVSTQLLL 994c)]		Env	murine(IgG)
	 MF87.1: The relative affinity binding [Moore et al.(1994c)] 	finity denatured/nati 94c)]	MF87.1: The relative affinity denatured/native gp120 is 10 – mutations 252 R/W, binding [Moore et al.(1994c)]	W, 257 T/	257 T/G, and 257 T/R impair	
306 MF169.1	gp120(242-261 LAI) gp120(256-265) RPVVSTQLLL Donor: J. Hoxie, U. Penn. References: [Thiriart et al.(1989), Moore et al.(1994c), Moore et al.(1994d)]	gp120(256-265) 989), Moore et al.(19	RPVVSTQLLL 994c), Moore et al.(1994d)]		Env	murine(IgG)
	• MF169.1: The relative affinit binding [Moore et al.(1994c)]	ffinity denatured/nat 94c)]	MF169.1: The relative affinity denatured/native gp120 is 11 – mutations 252 R/W, binding [Moore et al.(1994c)]		257 T/G, and 257 T/R impair	
307 MF170.1	gp120(242-261 LAI) Donor: J. Hoxie, U. Penn.	gp120(256-265)	RPVVSTQLLL		Env	murine(IgG)
	 References: [Thiriart et al.(1989), Moore et al.(1994c), Moore et al.(1994d)] NOTES: MF170.1: The relative affinity denatured/native gp120 is 15 – mutation 	989), Moore et al.(19 iffinity denatured/nat	 erences: [Thiriart et al.(1989), Moore et al.(1994c), Moore et al.(1994d)] TES: MF170.1: The relative affinity denatured/native gp120 is 15 – mutations 252 R/W, 	/W, 257 T/	257 T/G, and 257 T/R impair	
308 213.1	gp120(242-261 LAI)	gp120(256-265)	20(242-261 LAI) gp120(256-265) RPVVSTQLLL Env glycopro	2	Env glycopro	murine(IgG1)
	References: [Thiriart et al.(1989), Moore & Ho(1993), Moore et al.(1994c)]	989), Moore & Ho(1	993), Moore et al.(1994c)]			
	 213.1: Bound preferentia 213.1: The relative affin 	ully to denatured IIIB ity denatured/native	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	3)] V, 257 T/G	or T/R impair binding	
	• 213.1: UK Medical Research Council AIDS reagent: ARP334	arch Council AIDS 1	reagent: ARP334			

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
309 M89	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQLLLNGSLAEE- EVV	N	451 Env	$murine(IgG_1)$
	Ponor: Fulvia di Marzo Veronese References: [di Marzo Veronese e NOTES: • M89: Immunoblot reactive, Rl • M89: C2 region – the relative	nese lese et al.(1992), Moo ve, RIP negative, for s lative affinity for den	 Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d)] NOTES: M89: Immunoblot reactive, RIP negative, for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)] M89: C2 region – the relative affinity for denatured/native gp120 is >30 – mutations 257 T/R and 269 E/L impair 	1994d)] RUTZ [di Marzo mutations 257 T	o Veronese et al.(1992)] '⁄R and 269 E/L impair	
310 B12	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQLLLNGSLAEE- EVV		mis-folded LAI rgp160	murine(IgG)
	Donor: Fulvia di Marzo Veronese References: [Moore et al.(1994c)]	mese 94c)]				
	 B12: C2 region – the relative binding [Moore et al.(1994c)] 	lative affinity for der 94c)]	B12: C2 region – the relative affinity for denatured/native gp120 is 27 – mutations binding [Moore et al.(1994c)]		257 T/R and 262 N/T impair	
311 B13	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQLLLNGSLAEE- EVV		mis-folded LAI rgp160	$murine(\mathrm{Ig}G_{2a})$
	Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Pincus & McClure(1993), Moore & Ho(1993), Moore e et al.(1994d), Pincus et al.(1996), Connor et al.(1998)] NOTES:	ite of Human Virolog Clure(1993), Moore 96), Connor et al.(19	Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Pincus & McClure(1993), Moore & Ho(1993), Moore et al.(1994c), Abacioglu et al.(1994), Moore et al.(1994d), Pincus et al.(1996), Connor et al.(1998)] NOTES:	994c), Abaciog	lu et al.(1994), Moore	
	 B13: Also called Bh13 B13: Bound preferentiall B13: The relative affinity 	y to denatured IIIB g for denatured/native	B13: Also called Bh13 B13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] B13: The relative affinity for denatured/native gp120 is 30 – mutations 257 T/R and 269 E/L impair binding [Moore et al (1904.61)	[/R and 269 E/L	impair binding [Moore	
	 B13: C2 region – epitope B13: Called Bh13 – bin mediate cell killing – sCI 	boundaries mapped ds to gp120 but not O4 has no effect [Pin	B13: C2 region – epitope boundaries mapped by peptide scanning, core epitope: TQLLLN [Abacioglu et al.(1994)] B13: Called Bh13 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)]	ope: TQLLLN [3d to ricin A, the et al.(1996)]	Abacioglu et al.(1994)] e immunotoxin did not	
312 B24	gp120(C2 257-262 BH10)	gp120(261-266) TQLLLN	TQLLLN		mis-folded LAI rgp160	$\mathrm{murine}(\mathrm{IgG}_{2a})$
	Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Abacioglu et al.(1994)] NOTES:	ıte of Human Virolog .(1994)]	gy, Baltimore MD, USA		OF 3 C	
	B24: C2 region, epitope	boundaries mapped b	B24: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]	ı et al.(1994)]		

MAb ID	Location	WEAU	Sequence Neutralizing	Immunogen	Species(Isotype)
313 B3	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLLN	mis-folded LAI rgp160	$murine(IgG_1)$
	Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Abacioglu et al.(1994)] NOTES:	e of Human Virolog 1994)]	onor: George Lewis, Institute of Human Virology, Baltimore MD, USA eferences: [Abacioglu et al.(1994)] OTES:		
314 B21	gp120(C2 257-262 BH10)	gp120(261-266) TQLLLN	TQLLLN	mis-folded LAI	$murine(IgG_1)$
	Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Abacioglu et al.(1994)] NOTES: • R21: C2 region enitone boundaries manned by pentide scanning [Ab	e of Human Virolog 1994)]	onor: George Lewis, Institute of Human Virology, Baltimore MD, USA eferences: [Abacioglu et al.(1994)] OTES: R21: C2 region enitone boundaries manned by pentide scanning [Abacioglu et al.(1994)]	001d31	
	0 - 2 - 0 - 0 - 0 - 0 - 0 - 0 - 0 - 0 -	Jane of Janes Contraction	9 For		
315 B23	gp120(C2 257-262 BH10)	gp120(261-266) TQLLLN	TQLLLN	mis-folded LAI rgp160	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_{2a})$
	Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Abacioglu et al.(1994)] NOTES:	e of Human Virolog 1994)]	gy, Baltimore MD, USA		
	• B23: C2 region, epitope b	oundaries mapped b	• B23: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]		
316 B25	gp120(C2 257-262 BH10)	gp120(261-266) TQLLLN	TQLLLN	mis-folded LAI rgp160	$murine(IgG_1)$
	Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Abacioglu et al.(1994)] NOTES:	e of Human Virolog 1994)]	gy, Baltimore MD, USA		
	• B25: C2 region, epitope b	oundaries mapped b	• B25: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]		
317 B29	gp120(C2 257-263 BH10)	gp120(261-267) TQLLLNG	TQLLLNG	mis-folded LAI rgp160	$\operatorname{murine}(\operatorname{IgG}_{2a})$
	Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Abacioglu et al.(1994)] NOTES:	e of Human Virolog 1994)]	gy, Baltimore MD, USA		
	B29: C2 region, epitope b	oundaries mapped b	B29: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]		

318 B26 gp120						
	gp120(C2 257-263 BH10)	gp120(261-267)	TQLLLNG	mis-	ded LAI	$murine(IgG_1)$
Donor: (Reference NOTES: B26:	 Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Abacioglu et al.(1994)] NOTES: B26: C2 region, epitope boundaries mapped by peptide scanning [Ab 	of Human Virology 994)] undaries mapped by	 or: George Lewis, Institute of Human Virology, Baltimore MD, USA erences: [Abacioglu et al.(1994)] B26: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)] 	rgp160 994)]	60	
319 B36 gp120	gp120(C2 257-263 BH10)	gp120(261-267)	TQLLLNG	mis-	ded LAI	$murine(IgG_1)$
Donor: (Reference NOTES: B36:	 Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA References: [Abacioglu et al.(1994)] NOTES: B36: C2 region, epitope boundaries mapped by peptide scanning [Ab 	of Human Virology 994)] undaries mapped by	or: George Lewis, Institute of Human Virology, Baltimore MD, USA rences: [Abacioglu et al.(1994)] FES: B36: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]	rgp160 994)]	60	
320 C13 gp120	gp120(C2 252-271 LAI) Donor: George Lewis	gp120(256-275)	RPVVSTQLLLNGSLAEE- EVV	mis-fol rgp160	ded LAI	$murine(IgG_1)$
Reference NOTES:	rences: [Moore & Ho(1993) ES:	3), Moore et al.(199	References: [Moore & Ho(1993), Moore et al.(1994c), Abacioglu et al.(1994)] NOTES:			
• •	C13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] C13: The relative affinity for denatured/native gp120 is 36 – mutations 25' [Moore et al (1994c)]	r denatured/native g	C13: Bound preferentially to denatured 1118 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, and 269 E/L impair binding	
)	C13: Epitope boundary extended to RPVVSTQLLLNGSLAEEF mutation [Abacioglu et al.(1994)]	tended to RPVVST	C13: Epitope boundary extended to RPVVSTQLLLNGSLAEEEVVIR, to take into mutation [Abacioglu et al.(1994)]	account the	account the effect of a point	
321 110.E gp120	gp120(C2 262-281 LAI)	gp120(266-285)	NGSLAEEEVVIRSVNFT-	Env	Env glycopro	murine(IgG)
Donor: I Referenc NOTES:	Donor: F. Traincard References: [Moore et al.(1994c), Moore et al.(1994d)] NOTES:	c), Moore et al.(199	DNA 94d)]			
	·		CI			
322 110.C gp120 Dono	gp120(C2 261-280 LAI) gp120(275-284) Donor: F. Traincard, Hydridolabs, Institut Pasteur	gp120(275-284) bs, Institut Pasteur	VIRSVNFTDN	Env	Env glycopro	murine(IgG)
NOTES: • 110.0	ES: 110.C: The relative affinity 110.C: Only slightly reduce	for denatured/natives LAI viral binding	NOTES: • 110.C: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)] • 110.C: Only slightly reduces LAI viral binding or entry into CEM cells [Valenzuela et al.(1998)]	et al.(1998)]		

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
323 IIIB-V3-21	gp120(V3 299-304 IIIB) gp120(298-303) INCTRP Donor: J. Laman References: [Laman et al.(1992), Laman et al.(1993), Valenzuela et al.(1998)] NOTES:	gp120(298-303) INCTRP 992), Laman et al.(1993), Valen	INCTRP 93), Valenzuela et al.(1998)]	Z	Peptide	$\operatorname{murine}(\operatorname{IgG}_1)$
	 IIIB-V3-21: Also called V3-21 IIIB-V3-21: Binds to the base IIIB-V3-21: Binds to NP40 	V3-21 base of the V3 loop NP40 treated gp120,	IIIB-V3-21: Also called V3-21 IIIB-V3-21: Binds to the base of the V3 loop on denatured gp120 [Laman et al.(1992)] IIIB-V3-21: Binds to NP40 treated gp120, and epitope is probably obscured by local glycosylation [Laman	et al.(1992)]	l glycosylation [Lam:	an
	et al.(1993)] • IIIB-V3-21: Does not bl	ock HIV-1 LAI bindi	et al.(1993)] IIIB-V3-21: Does not block HIV-1 LAI binding or entry into CEM cells [Valenzuela	alenzuela et al.(et al.(1998)]	
	 IIIB-V3-21: UK Medical Research Council AIDS reagent: ARP3048 IIIB-V3-21: NIH AIDS Research and Reference Reagent Program: 1 	ll Research Council A Research and Refere	IIIB-V3-21: UK Medical Research Council AIDS reagent: ARP3048 IIIB-V3-21: NIH AIDS Research and Reference Reagent Program: 1725			
324 IIIB-V3-26	gp120(V3 299-304 IIIB) gg Donor: J. Laman References: [Laman et al.(1992)] NOTES: • IIIB-V3-26: Binds to the base	gp120(295-311) 992)] base of the V3 loop	20(V3 299-304 IIIB) gp120(295-311) SVEINCTRPNNNTRKSI N nor: J. Laman et al.(1992)] TES: IIIB-V3-26: Binds to the base of the V3 loop on denatured gp120 [Laman et al.(1992)]	N et al.(1992)]	Peptide	$\mathrm{murine}(\mathrm{IgG}_1)$
325 MO97/V3	gp120(V3 299-308 IIIB)	gp120(303-312) PNNNTRKSIR	PNNNTRKSIR	Z	rpB1 (IIIB Env 286-467)	human(IgM)
	Donor: J. Laman References: [Ohlin et al.(1992)] NOTES: • MO97: Generated through <i>ii</i>	92)] gh <i>in vitro</i> "immuniz:	 I. Laman Frences: [Ohlin et al.(1992)] TES: MO97: Generated through in vitro "immunization" of uninfected-donor lymphocytes 		[Ohlin et al.(1992)]	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
326 8/38c	gp120(V3 300-315 HXB10) gp120(304-317) NNNTRKRIRIQRGPG Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [McKeating et al.(1992a), Sattentau & Moore(1995), Jeffs et a NOTES: • 8/38c: Also called 8/38/1c • 8/38c: Binds to virion gp120 and neutralizes only in the presence of sC • 8/38c: Binds equally well to monomer and oligomer, less rapid associan associated less potent neutralization of lab strains [Sattentau & Moo • 8/38c: Deletion of the V1V2 regions did not affect anti-V3 Abs ability [Jeffs et al.(1996)] • 8/38c: The MAb and Fab binding to the oligomeric form of gp120 and not suggest that neutralization is determined by the fraction of Ab sites occ [Parren et al.(1998)] • 8/38c: UK Medical Research Council AIDS reagent: ARP3039	tton, Institute for Canal. (1992a), Sattentau al. (1992a), Sattentau	gp120(V3 300-315 HXB10) gp120(304-317) NNNTRKRIRIQRGPGR L rBH10 gp120 Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [McKeating et al.(1992a), Sattentau & Moore(1995), Jeffs et al.(1996), Parren et al.(1998)] NOTES: 8/38c: Also called 8/38/1c 8/38c: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating et al.(1992a)] 8/38c: Binds equally well to monomer and oligomer, less rapid association rate than other anti-V3 antibodies, and an associated less potent neutralization of lab strains [Sattentau & Moore(1995)] 8/38c: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)] 8/38c: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)] 8/38c: UK Medical Research Council AIDS reagent: ARP3039	L McKeating et al McKeating et al rate than other 95)] ind when comp itation were high d on a virion irr	rBH10 gp120 n et al.(1998)] g et al.(1992a)] other anti-V3 antibodies, and compared to intact rec gp120 ere highly correlated – authors ion irrespective of the epitope	$\operatorname{rat}(\operatorname{IgG}_{2a})$
327 8/64b	gp120(V3 300-315 HXB10) gp120(304-317) NNNTRKRIRIQRGPG Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [McKeating et al.(1992a)] NOTES: • 8/64b: Binds to virion gp120 and neutralizes only in the presence of sC • 8/64b: UK Medical Research Council AIDS reagent: ARP3036	tton, Institute for Candal.(1992a)] gp120 and neutralizes (gearch Council AIDS r	20(V3 300-315 HXB10) gp120(304-317) NNNTRKRIRIQRGPGR L rBH10 g 100 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 110 erences: [McKeating et al.(1992a)] 111 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 111 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 111 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 111 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 111 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 112 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 113 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 113 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 114 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 115 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 115 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 116 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 117 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK 118 cor: C. Dean and C. Shotton, Institute	L McKeating et al	rBH10 gp120	rat(IgM)
328 polyclonal	gp120(V3 IIIB) gp120(305-328) NNTRKSIRIQRGPGR VTIGKIGN Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [Bukawa et al.(1995)] NOTES: • Polyclonal secretory IgA antibody raised by mucosal immunization is a neutralization may be due to V3, CD4 or HPG30 component of the met al.(1995)]	gp120(305-328) tton, Institute for Canc (1995)] A antibody raised by mue to V3, CD4 or HP0	20(V3 IIIB) gp120(305-328) NNTRKSIRIQRGPGRAF- VTIGKIGN peptide plus cholera toxin adjuvant toxin adjuvant research; Surrey, UK research; Surrey, UK research periode immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa et al.(1995)]	L neutralize IIIB mponent peptid	oral immunization – peptide plus cholera toxin adjuvant toxin adjuvant e immunogen [Bukawa	murine(IgA)

329 polyclonal gp120(V3 IIIB) gp120(305-328) NNTRKSIRIQRGPGRAF- L DNA vaccine IIIIB murine(IgA22a) Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [Sasaki et al.(1998)] NOTES: • The env response is what is being sought, but co-expression of Rev is required – intramuscular versus nasal vaccination with DNA vaccine with a QS-21 adjuvant was studied – QS-21 enhanced the IgG2a response mediated via Th1 cytokines IFN7 and IL-2 [Sasaki et al.(1998)] 330 polyclonal gp120(V3 MN) gp120(304-328) CNYNKRKRIHIGPGRAF- L rabbit(IgA and Ig YTTKNIIGTIC Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [FitzGerald et al.(1998)] • Polyclonal response to MN, or Thai E V3 loop inserted into Pseudomonus Exotoxin for vaccination – inserts of 14 or 26 amino acids were used from MN or a Thai E strain, constrained by disulfide bond – sera from vaccinated rabbit were reactive with strain-specific gp120 – adminstration to mucosal surfaces elicits IgA [FitzGerald et al.(1998)] Bonor: D. Bolognesi and T. Matthews, Duke University References: [Allaway et al.(1993)] NOTES: • Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)]	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
 Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [Sasaki et al.(1998)] NOTES: The env response is what is being sought, but co-expression of Rev is required – intramuscular versus nasal vaccination with DNA vaccine with a QS-21 adjuvant was studied – QS-21 enhanced the IgG2a response mediated via Th1 cytokines IFNγ and IL-2 [Sasaki et al.(1998)] gp120(V3 MN) gp120(304-328) CNYNKRKRIHIGPGRAF- L	329 polyclonal	gp120(V3 IIIB)	gp120(305-328)	NNTRKSIRIQRGPGRAF- VTIGKIGN	T	cine IIIB	murine(IgA22a)
 The env response is what is being sought, but co-expression of Rev is required – intramuscular versus nasal vaccination with DNA vaccine with a QS-21 adjuvant was studied – QS-21 enhanced the IgG2a response mediated via Th1 cytokines IFNγ and IL-2 [Sasaki et al.(1998)] gp120(V3 MN) gp120(304-328) CNYNKRKRIHIGPGRAF- L YTTKNIIGTIC Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [FitzGerald et al.(1998)] Polyclonal response to MN, or Thai E V3 loop inserted into Pseudomonas Exotoxin for vaccination – inserts of 14 or 26 amino acids were used from MN or a Thai E strain, constrained by disulfide bond – sera from vaccinated rabbit were reactive with strain-specific gp120 – administration to mucosal surfaces elicits IgA [FitzGerald et al.(1998)] gp120(V3 IIIB) gp120(305-325) CNNTRKSIRIQRGPGRA- L ? FVTIGK Donor: D. Bolognesi and T. Matthews, Duke University References: [Allaway et al.(1993)] Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)] 		Donor: C. Dean and C. Shot References: [Sasaki et al.(19 NOTES:	ton, Institute for Can 198)]	cer Research, Surrey, UK			
gp120(V3 MN) gp120(304-328) CNYNKRKIHIGPGRAF- YTTKNIIGTIC Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [FitzGerald et al.(1998)] NOTES: • Polyclonal response to MN, or Thai E V3 loop inserted into Pseudomonas Exotoxin for vaccination – inserts of 14 or 26 amino acids were used from MN or a Thai E strain, constrained by disulfide bond – sera from vaccinated rabbit were reactive with strain-specific gp120 – administration to mucosal surfaces elicits IgA [FitzGerald et al.(1998)] gp120(V3 IIIB) gp120(V3 IIIB) gp120(305-325) GNNTRKSIRIQRGPGRA- FVTIGK Donor: D. Bolognesi and T. Matthews, Duke University References: [Allaway et al.(1993)] NOTES: • Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)]		 The env response is what with DNA vaccine with cytokines IFNγ and IL-2 	is being sought, but co a QS-21 adjuvant w [Sasaki et al.(1998)]	o-expression of Rev is required vas studied – QS-21 enhanced l	– intramuscular the IgG2a resp	versus nasal vaccination onse mediated via Th1	
Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK References: [FitzGerald et al.(1998)] NOTES: • Polyclonal response to MN, or Thai E V3 loop inserted into <i>Pseudomonas</i> Exotoxin for vaccination – inserts of 14 or 26 amino acids were used from MN or a Thai E strain, constrained by disulfide bond – sera from vaccinated rabbit were reactive with strain-specific gp120 – administration to mucosal surfaces elicits IgA [FitzGerald et al.(1998)] gp120(V3 IIIB) gp120(V3 IIIB) gp120(305-325) EVTIGK Donor: D. Bolognesi and T. Matthews, Duke University References: [Allaway et al.(1993)] NOTES: • Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)]	330 polyclonal	gp120(V3 MN)	gp120(304-328)	CNYNKRKRIHIGPGRAF- YTTKNIIGTIC	Т		rabbit(IgA and IgG)
gp120(V3 IIIB) gp120(305-325) CNNTRKSIRIQRGPGRA- L ? FVTIGK Donor: D. Bolognesi and T. Matthews, Duke University References: [Allaway et al.(1993)] NOTES: • Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)]		 Donor: C. Dean and C. Shot References: [FitzGerald et a NOTES: Polyclonal response to N or 26 amino acids were u were reactive with strain 	ton, Institute for Can 1.(1998)] IN, or Thai E V3 loo sed from MN or a Th specific gp120 – adn	cer Research, Surrey, UK p inserted into <i>Pseudomonas</i> ai E strain, constrained by disuninistration to mucosal surface	Exotoxin for vac ılfide bond – ser s elicits IgA [Fi	cination – inserts of 14 a from vaccinated rabbit tzGerald et al.(1998)]	
ws, Duke University CD4-based molecules in inhibition of HIV-1 Env	331 polyclonal	gp120(V3 IIIB)		CNNTRKSIRIQRGPGRA- FVTIGK	Т		Guinea pig IgG
		Donor: D. Bolognesi and T.References: [Allaway et al.(NOTES:Synergy with combinati et al.(1993)]	Matthews, Duke Uni 1993)] ons of CD4-based m	versity nolecules in inhibition of HIV		d cell fusion [Allaway	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
332 1324E	gp120(V3 E clade)	gp120(307-312)	TRTSVR	T	HIV-1 E clade infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	 Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Gorny et al.(1998)] NOTES: A hu MAb derived from an HIV-1 E subtype reactive with V3 peptides, rgp120, and mon C-clade primary isolate, but not with B and 	(NYU Med. Center) (98)] an HIV-1 E subtype s, rgp120, and mone but not with B and I	or: Susan Zolla-Pazner (NYU Med. Center) erences: [Gorny et al.(1998)] TES: A hu MAb derived from an HIV-1 E subtype infected service man from the US who had served in Thailand – cross-reactive with V3 peptides, rgp120, and monomeric gp120 from E, C and A clades, as well as cells infected with a C-clade primary isolate, but not with B and D clade V3 peptides or rgp120 – neutralizes E clade virus adapted for	US who had ser a clades, as well — neutralizes E	ved in Thailand – cross- as cells infected with a clade virus adapted for	
	measured, and the association rates were similar, but dis 1324E was comparable to 447-52D [Gorny et al.(1998)]	iation rates were sim o 447-52D [Gorny et	measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 1324E was comparable to 447-52D [Gorny et al.(1998)]	stants were quite	variable for V3 MAbs,	
333 MO99/V3	gp120(V3 304-308 IIIB)	gp120(308-312)	RKSIR	Z	rpB1 (IIIB Env 286-467)	human(IgM)
	 Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Ohlin et al.(1992)] NOTES: MO99: Generated through <i>in vitro</i> "immuniz 	(NYU Med. Center) 92)] gh <i>in vitro</i> "immuniz	 onor: Susan Zolla-Pazner (NYU Med. Center) eferences: [Ohlin et al.(1992)] OTES: MO99: Generated through in vitro "immunization" of uninfected-donor lymphocytes 		[Ohlin et al.(1992)]	
334 DO 142-10	gp120(V3 MN) gp120(309-322) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Seligman et al.(1996)]	gp120(309-322) (NYU Med. Center) (1996)]	KRIHIGPGRAFYTT		HIV-1 infection	human
	• DO 142-10: Fab fragme [Seligman et al.(1996)]	ent – competition EI	DO 142-10: Fab fragment – competition ELISAs with serial deletions defined the epitope KRIHIGPGRAFYTT [Seligman et al.(1996)]	fined the epitop	e KRIHIGPGRAFYTT	
335 polyclonal	gp120(V3 C subtype) gp120(310-321) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Ahluwalia et al.(1997)]	gp120(310-321) (NYU Med. Center) (.(1997)]	CKRKIHIGPGQAFYT		Peptide-ISCOM	$murine(IgG_{2a,b})$
	 A V3 loop peptide modified to resemble an Indian form (C complexes) or liposomes, and used to immunize mice – presentation in the ISCOM suggestive of a Th1 response 	ed to resemble an Inc s, and used to immu M suggestive of a TI	A V3 loop peptide modified to resemble an Indian form (GPGQ) was incorporated into complexes) or liposomes, and used to immunize mice – the IgG2a/IgG2b antibody presentation in the ISCOM suggestive of a Th1 response		ISCOMS (immune stimulating response was enhanced by the	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
336 polycional	gp120(V3 MN) gp120(308-322) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Spear et al.(1994)] NOTES: • 40% of antibody in serum that can bind to na RKRIHIGPGRAFYTT, which can also bloc et al.(1994)]	gp120(308-322) (NYU Med. Center) 94)] m that can bind to na which can also bloc	20(V3 MN) gp120(308-322) RKRIHIGPGRAFYTT L (MN A nor: Susan Zolla-Pazner (NYU Med. Center) ferences: [Spear et al.(1994)] 7TES: 40% of antibody in serum that can bind to native viral proteins on MN-infected cells RKRIHIGPGRAFYTT, which can also block 75-95% of the complement activation et al.(1994)]		L (MN ALA-1) HIV-1 infection ted cells can be blocked by the peptide activation on HIV infected cells [Spear	human
337 polyclonal	gp120(V3 MN)	gp120(313-324)	gp120(313-324) IGPGRAFYTTKN	L (MN ALA-1)	L (MN ALA-1) IGPGRAFYTTKN HRV14: HIV-1 chimera	guinea pigs
	 Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Smith et al.(1998)] NOTES: The tip of the MN V3 loop was inserted into elected, and chimeric viruses were neutralizagainst ALA-1 and MN [Smith et al.(1998)] 	(NYU Med. Center) (98)] op was inserted into coruses were neutralize [Smith et al.(1998)]	or: Susan Zolla-Pazner (NYU Med. Center) erences: [Smith et al.(1998)] TES: 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 – chimeric viruses elicited potent NAbs against ALA-1 and MN [Smith et al.(1998)]	s 14 (HRV14) – ch s – chimeric viruse	14) – chimeras were immunos- c viruses elicited potent NAbs	
338 TH1	gp120(V3) unk Donor: Michael Fung, Tanox Biosystem, USA References: [D'Souza et al.(1995), Yang et al.(1998)] NOTES:	x Biosystem, USA (1995), Yang et al.(19	unk 998)]	L (MN,JRCSF)		$\operatorname{human}(\operatorname{Ig} G_{1\lambda})$
	 TH1: Found to neutralize MN and JRCSF, but not two by most labs in a multi-laboratory study involving 11 la TH1: A neutralization assay was developed based on h HNPCR consistently revealed HIV DNA and was showr on tests with 6 MAbs and 5 isolates [Yang et al.(1998)] 	we MN and JRCSF, but aboratory study invo- assay was developed lealed HIV DNA and d 5 isolates [Yang et]	TH1: Found to neutralize MN and JRCSF, but not two B subtype primary isolates, nor a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs [D'Souza et al.(1995)] TH1: A neutralization assay was developed based on heminested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang et al.(1998)]	isolates, nor a D st 1995)] aplification of the I ific and reliable nev	or a D subtype primary isolate, of the LTR (HNPCR) – LTR- able neutralization assay based	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
339 D47	(V3 IIIB)		unk		IIIB vaccinia ex- pressed Env	murine
	Donor: Michael Fung, Tanox Biosystem, USA References: [Richardson Jr et al.(1996), Wyatt et al.(1997), Earl et al.(1997), Otteken et. NOTES:	x Biosystem, USA et al.(1996), Wyatt et	al.(1997), Earl et al.(1997),	Otteken et al.(1996)]	96)]	
	 D47: Used for capture c was not blocked by hum et al.(1996)] D47: Binds both gp120 	f oligomeric Env for a nan sera from the US, and soluble gp120+g	D47: Used for capture of oligomeric Env for antigen capture ELISA – binding of this antibody to oligomeric Env IIIB was not blocked by human sera from the US, consistent with a low prevalence of IIIB-like V3 strains [Richardson Jr et al.(1996)] D47: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by	ing of this antiboence of IIIB-like gesting its gp120	dy to oligomeric Env IIIB V3 strains [Richardson Ji epitope is not blocked by	
	 D47: Binds both gp120 and solubl gp41 binding [Wyatt et al.(1997)] D47: Used for comparison in a st Env than any of 38 conformation of D47: Pulse label experiments of N diately and binding stayed constar 	and soluble gp120+g al.(1997)] son in a study of gp4 ormation dependent aments of MAb bindin ed constant through c	D47: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)] gp41 binding [Wyatt et al.(1997)] D47: Used for comparison in a study of gp41 antibodies – D47 binds to a greater extent to cell surface expressed Env than any of 38 conformation dependent anti-gp41 MAbs [Earl et al.(1997)] D47: Pulse label experiments of MAb binding to noncleavable gp160 revealed that this anti-V3 MAb bound immediately and binding stayed constant through chase period [Otteken et al.(1996)]	gesting its gp120 a greater extent [997)] ealed that this an 996)]	epitope is not blocked by to cell surface expressed ti-V3 MAb bound imme-	. 1
340 F19.48-3	gp120(V3 312-324 LAI)	gp120(309-321)	gp120(309-321) IRIQRGPGRAFVT	L	IIIB rgp120 294- 474	$\mathrm{murine}(\mathrm{Ig}G_{2a\kappa})$
	Donor: ? References: [Boudet et al.(1994)] NOTES: • F19.48-3: Strain specific – use	(1994)] c – used to raise anti-	 ior: ? ierences: [Boudet et al.(1994)] TES: F19.48-3: Strain specific – used to raise anti-idiotype antibodies [Boudet et al.(1994)] 	et al.(1994)]		
341 F19.26-4	gp120(V3 312-324 LAI)	gp120(309-321)	gp120(309-321) IRIQRGPGRAFVT	Т	IIIB rgp120 294- 474	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_{2a\kappa})$
	Donor: ? References: [Boudet et al.(1994)] NOTES:	[994)]				

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
342 F19.57-11	gp120(V3 312-324 LAI)	gp120(309-321)	IRIQRGPGRAFVT	L (LAI)	IIIB rgp120 294- 474	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_{1\kappa})$
	Donor:? References: [Boudet et al.(1991), Boudet et al.(1994), Boudet et al.(1995)] NOTES: • F19.57-11: MAb F19.57-11 is strain specific for LAI – used to raise	91), Boudet et al.(19 -11 is strain specifi	nor:? ferences: [Boudet et al.(1991), Boudet et al.(1994), Boudet et al.(1995)] TES: F19.57-11: MAb F19.57-11 is strain specific for LAI – used to raise anti-idiotype		rabbit antibodies (called 57-B	
	Ab2) [Boudet et al.(1994)] • F19.57-11: Anti-anti-idiot than the original F19.57-11 [Boudet et al.(1995)])] I (Ab3 could also re	Ab2) [Boudet et al.(1994)] F19.57-11: Anti-anti-idiotypic antibodies (Ab3) were raised in BALB/c mice that had greater breadth of reactivity than the original F19.57-11 (Ab3 could also recognize 1282 and SF2, with aa TRK(R or S)IYIGPGRA(WY or FH)T) [Boudet et al.(1995)]	ice that had grea	ter breadth of reactivity IGPGRA(WY or FH)T)	
343 M096/V3	gp120(309-318 + 329-338)	gp120	IQRGPGRAFV + AHCN- ISRAKW		rIIIB Env 286-467	human(IgM)
	Donor: ? References: [Ohlin et al.(1992)] NOTES: • M096: Generated through <i>in</i>	2)] 1 <i>in vitro</i> "immuniza	nor: ? ferences: [Ohlin et al.(1992)] YTES: M096: Generated through <i>in vitro</i> "immunization" of uninfected-donor lymphocytes		Ohlin et al.(1992)]	
344 MO101/- V3,C4	gp120(314-323 + 494-503)	gp120	GRAFVTIGKI + LGVA- PTKAKR		rIIIB Env 286-467	human(IgM)
	Donor: ? References: [Ohlin et al.(1992)] NOTES: • MO101: generated through <i>in vitro</i> "immuthe V3 and C4 regions [Ohlin et al.(1992)]	2)] gh <i>in vitro</i> "immuni hlin et al.(1992)]	nor:? ferences: [Ohlin et al.(1992)] TES: MO101: generated through <i>in vitro</i> "immunization" of uninfected-donor lymphocytes – reacts with peptides from the V3 and C4 regions [Ohlin et al.(1992)]	ymphocytes – re	acts with peptides from	
345 N70-1.9b	gp120(V3 316-322) gp120(315-320) PGRA Donor: ? References: [Robinson et al.(1990), Scott Jr et al.(1990)] NOTES: NOTES: N70-1.9b: Type specificity [Robinson et al.(1990)] N70-1.9b: Type specific neutralization, ADCC direct	gp120(315-320) 1990), Scott Jr et al. 1980) g [Robinson et al.(1) 1990 eutralization, ADC	or: ? prences: [Robinson et al.(1990), Scott Jr et al.(1990)] res: N70-1.9b: Type specific neutralization, ADCC directed against MN infected cells [Scott Jr et al.(1990)]	L d cells [Scott Jr	HIV-1 infection et al.(1990)]	$\operatorname{human}(\operatorname{IgG}_1)$

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
346 MAG 49	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRAFV- TIG	T	sCD4-(rHXB2 gp120)-complex	murine
	Donor: ? References: [Kang et al.(1994), Moore & Sodroski(1996)] NOTES:), Moore & Sodrosl	દાં(1996)]			
	 MAG 49: Binds a V3 loop peptide – sensitive to bo loop structure (120/121 VK/LE) [Kang et al.(1994)] 	p peptide – sensitiv K/LE) [Kang et al.(MAG 49: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]	nd a mutation a	t the base of the V1/V2	, c
	• MAG 49: Called #49 in thi site MAbs – reciprocal enh & Sodroski(1996)]	Is text. Binding enhalancement of some a	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)]), 135/9, and by ing inhibition o	nd by many anti-CD4 binding tion of anti-V3 MAbs [Moore	
347 MAG 53	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRAFV- TIG	L	sCD4-(rHXB2 gp120)-complex	murine
	Donor: ? References: [Kang et al.(1994)] NOTES:	[(4				
	 MAG 53: Binds a V3 loop peptide – sensitive to be loop structure (120/121 VK/LE) [Kang et al.(1994)] 	p peptide – sensitiv K/LE) [Kang et al.(MAG 53: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]	nd a mutation a	t the base of the V1/V2	
348 MAG 56	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRAFV- TIG	L	sCD4-(rHXB2 gp120)-complex	murine
	Donor: ? References: [Kang et al.(1994)] NOTES:	9)				
	• MAG 56: Binds a V3 loop peptide – sensitive to be loop structure (120/121 VK/LE) [Kang et al.(1994)]	p peptide – sensitiv K/LE) [Kang et al.(MAG 56: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]	nd a mutation a	t the base of the V1/V2	
349 MAG 109	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRAFV- TIG	L	sCD4-(rHXB2 gp120)-complex	murine
	Donor: ? References: [Kang et al.(1994)] NOTES: • MAG 109: Binds a V3 loop peptide – sensitive to b loop structure (120/121 VK/LE) [Kang et al.(1994)]	l)] op peptide – sensitiv K/LE) [Kang et al.(or: ? erences: [Kang et al.(1994)] TES: MAG 109: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]	nd a mutation a	t the base of the V1/V2	, -

MAb ID	Location	WEAU	Sequence	Neutralizing	Neutralizing Immunogen	Species(Isotype)
350 polyclonal	gp120(V3 306-338 BH10)	gp120(303-334)	PNNNTRKSIRIQRGPGR- AFVTIGKIGNMRQAHC	L	Peptide	rabbit(IgG)
	Donor: ? References: [Neurath & Strick(1990)] NOTES:	((1990)]				
	• 21 V3 loop variant peptide: [Neurath & Strick(1990)]	spanning this regio	21 V3 loop variant peptides spanning this region were tested and serological cross-reactivity correlated with divergence [Neurath & Strick(1990)]	oss-reactivity co	orrelated with divergen	<u>i</u> ce
351 1026	gp120(V3 tip MN) Donor: ?	gp120(314-319) GPGRAF?	GPGRAF?	L	rgp120 MN	murine(IgG)
	References: [Nakamura et al.(1993), Bou-Habib et al.(1994)] NOTES:	1993), Bou-Habib	et al.(1994)]			
	 1026: Bound diverse strai 1026: Greater affinity fo [Bou-Habib et al.(1994)] 	ns, neutralizing acti	1026: Bound diverse strains, neutralizing activity against MN, close to GPGRAF [Nakamura et al.(1993)] 1026: Greater affinity for T cell-tropic strain T-CSF, derived from JR-CSF, than to the primary isolate [Bou-Habib et al.(1994)]	RAF [Nakamu F, than to the	[akamura et al.(1993)] to the primary isolate JR-CSF	SF
352 9284	gp120(V3 307-318 IIIB)	gp120(305-316)	gp120(305-316) NNTRKSIRIQRG	T	disrupted IIIB virion	$murine(IgG_1)$
	Danor: Dimont de Nemoire Les Illis France or Wilmington Delaware	es Illis France or	Wilmington Delawara			

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

- 9284: IIIB type-specific binding and neutralization [Skinner et al.(1988b)]
- 9284: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 9284: Single amino acid substitutions in the C4 region (427 W/V or W/S) or at the base of the V3 loop (298 R/G) binding site MAbs [Wyatt et al.(1992)] can significantly increase binding and neutralization-position 427 is also important for CD4 binding and anti-CD4
- 9284: Increased binding in the presence of sCD4 [Sattentau et al.(1993)]
- 9284: Inhibits C4 region antibodies (G3-299, G3-519) which have conformational requirements [Moore et al. (1993b)]
- 9284: Peptide RIQRGPGRAFVTIGKIGNMRQA Reacts with three human brain proteins of 35, 55, 110 kDa called NEA-9284 [Trujillo et al.(1993)]

MAb ID
Location
WEAU S
Sequence
Neutralizing
Immunogen
Species(Isotype)

352 (cont.)

NOTES:

- 9284: Does not bind MN gp120, just IIIB [VanCott et al.(1994)]
- 9284: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali et al.(1994)]
- 9284: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer in vitro [Cook et al.(1994)]
- 9284: Binding domain aa 301-310: TRKSIRIQRG mutations in the V3 loop from basic residues can destroy virus different binding sites: 9284 and 0.5β – called NEA9284 [Okada et al.(1994)] infectivity and syncytium formation: 306 R/T,309 R/T and 313 R/G can also reduce binding of V3 MAbs with two
- 9284: Did not neutralize infection of HIV/HTLV-I pseudotype [Sorensen et al.(1994)]
- 9284: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains neutralizes cell-free virus Hx10 [Sattentau & Moore(1995)]
- et al.(1995)] 9284: Used to monitor HIV-1 Env expression in infected H9 cells, binds native and reduced gp 120s similarly [VanCott
- 9284: Binds V3 loop anti-C1 MAbs 133/290 and 135/9 enhance binding reciprocal binding inhibition of other anti-V3 MAbs [Moore & Sodroski(1996)]
- 9284: V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)]
- 9284: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao et al.(1997)]
- 9284: A high avidity antibody as assessed by urea elution
- suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope 9284: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors [Parren et al.(1998)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
353 1034	gp120(V3 tip MN) gp120(314-319) GPGRAF? Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [Bou-Habib et al.(1994), Berman et al.(1997)] NOTES: • 1034: Greater affinity for T cell tropic T-CSF, derived from JR-CS GPGRAF [Bou-Habib et al.(1994)] • 1034: Binds to 5/7 isolates from breakthrough cases from a MN gp1	gp120(314-319) Les Ulis, France or V (1994), Berman et a (1994) T cell tropic T-CSF (1(1994)) Infrom breakthrough	120(V3 tip MN) gp120(314-319) GPGRAF? In rgp120 MN gp120(314-319) GPGRAF? In rgp120 MN gp120(314-319) GPGRAF? In rgp120 MN gp120 MN gp120(314-319) GPGRAF? L rgp120 MN L rgp120 MN L rgp120 MN rgp120 MN primary isolate JR-CSF, close to GPGRAF [Bou-Habib et al.(1994)] 1034: Binds to 5/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)]	L o the primary ii	rgp120 MN mary isolate JR-CSF, close to Berman et al.(1997)]	murine(IgG)
354 polyclonal	 gp120(V3 304-318 LAI) gp120(306-320) RKSIRIQRGPGRAF Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [Chin et al.(1995)] NOTES: • Mimicking the humoral immune response <i>in vitro</i> supports isotype from naive donors [Chin et al.(1995)] 	gp120(306-320) Les Ulis, France or V mune response <i>in</i> 1 al.(1995)]	20(V3 304-318 LAI) gp120(306-320) RKSIRIQRGPGRAFV ? nor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware erences: [Chin et al.(1995)] TES: Mimicking the humoral immune response in vitro supports isotype switching – human IgG MAbs were generated from naive donors [Chin et al.(1995)]	ıg – human IgG	? MAbs were generated	human(IgG, IgM)
355 polyclonal	gp120(V3 304-318 LAI) gp120(306-320) RKSIRIQRGPGRAF Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [Andersson et al.(1997)] NOTES: • IgG to IgM isotype switching in response to primary and secondary pe contained a V3 loop fragment and a tetanus toxin helper epitope [Ar	gp120(306-320) es Ulis, France or V 1997)] ng in response to prir ent and a tetanus to:	20(V3 304-318 LAI) gp120(306-320) RKSIRIQRGPGRAFV peptide or: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware erences: [Andersson et al.(1997)] TES: IgG to IgM isotype switching in response to primary and secondary peptide vaccinations was studied—the immunogen contained a V3 loop fragment and a tetanus toxin helper epitope [Andersson et al.(1997)]	et al.(1997)]	peptide udied – the immunogen	human(IgG, IgM)
356 Aw	gp120(V3 tip, Gun-1wt) gp120(309-322) KSITIGPGRAFHAI Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [McKnight et al.(1995)] NOTES: • Aw: Rat antibodies were raised against V3 peptides that represent eit (v) of the isolate Gun-1 – Aw gives weak neutralization of both wt a	gp120(309-322) es Ulis, France or V 1995)] iised against V3 pepi	20(V3 tip, Gun-1wt) gp120(309-322) KSITIGPGRAFHAI L V3 peptide nor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware erences: [McKnight et al.(1995)] TES: Aw: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – Aw gives weak neutralization of both wt and v strains [McKnight et al.(1995)]	L ildtype (wt), or ins [McKnight.	V3 peptide brain-cell tropic variant at al.(1995)]	rat t
357 Bw	gp120(V3 tip, Gun-1wt) gp120(309-322) KSITIGPGRAFHAI Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [McKnight et al.(1995)] NOTES: • Bw: Rat antibodies were raised against V3 peptides that represent eit (v) of the isolate Gun-1 – Bw gives weak neutralization of only the et al.(1995)]	gp120(309-322) Les Ulis, France or V [1995] uised against V3 pep [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995] [1995]	20(V3 tip, Gun-1wt) gp120(309-322) KSITIGPGRAFHAI Lor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware erences: [McKnight et al.(1995)] TES: Bw: Rat antibodies were raised against V3 peptides that represent either the wildtype (v) of the isolate Gun-1 – Bw gives weak neutralization of only the wt strain, does et al.(1995)]	` · · · · ·	V3 peptide (wt), or brain-cell tropic variant not bind to variant [McKnight	rat t

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
358 Dv	gp120(V3 tip, Gun-1v) gp120(309-322) KSITIGSGRAFHAI Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [McKnight et al.(1995)] NOTES: • Dv: Rat antibodies were raised against V3 peptides that represent eitl (v) of the isolate Gun-1 – neutralization of only the variant strain, do	gp120(309-322) es Ulis, France or W [1995] ised against V3 pept [1995] [1995]	120(V3 tip, Gun-1v) gp120(309-322) KSITIGSGRAFHAI L V3 peptide nor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware ferences: [McKnight et al.(1995)] TES: Dv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]	L vildtype (wt), or nd to wildtype	V3 peptide brain-cell tropic variant [McKnight et al.(1995)]	rat
359 Fv	gp120(V3 tip, Gun-1v) gp120(309-322) KSITIGSGRAFHAI Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [McKnight et al.(1995)] NOTES: • Fv: Rat antibodies were raised against V3 peptides that represent eith (v) of the isolate Gun-1 – neutralization of only the variant strain, do	gp120(309-322) es Ulis, France or W 1995)] ised against V3 pepti eutralization of only	20(V3 tip, Gun-1v) gp120(309-322) KSITIGSGRAFHAI L V3 peptide nor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware erences: [McKnight et al.(1995)] TES: Fv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]	L 'ildtype (wt), or nd to wildtype [wt]	V3 peptide brain-cell tropic variant [McKnight et al.(1995)]	rat
360 Gv	gp120(V3 tip, Gun-1v) gp120(309-322) KSITIGSGRAFHAI Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [McKnight et al.(1995)] NOTES:	gp120(309-322) .es Ulis, France or W 1995)]	gp120(309-322) KSITIGSGRAFHAI ss Ulis, France or Wilmington, Delaware 995)]	T	V3 peptide	rat
	(v) of the isolate Gun-1 – r	eutralization of only	(v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]	nd to wildtype	McKnight et al.(1995)]	
361 Hv	gp120(V3 tip, Gun-1v) gp120(309-322) KSITIGSGRAFHAI Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [McKnight et al.(1995)] NOTES: • Hv: Rat antibodies were raised against V3 peptides that represent eitl (v) of the isolate Gun-1 – neutralization of only the variant strain, do	gp120(309-322) es Ulis, France or W 1995)] ised against V3 pept eutralization of only	20(V3 tip, Gun-1v) gp120(309-322) KSITIGSGRAFHAI L V3 peptide nor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware erences: [McKnight et al.(1995)] TES: Hv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]	L rildtype (wt), or nd to wildtype	V3 peptide brain-cell tropic variant [McKnight et al.(1995)]	rat
362 polyclonal	gp120(V3 304-318 LAI) gp120(310-321) RIHIGPGRAFYT Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware References: [Langedijk et al.(1995)] NOTES: Polylconal sera from six individuals tested for reactivity against a paper provide evidence for immunological escape mutations in the tip of the state o	gp120(310-321) RIHIGPGRAFYT es Ulis, France or Wilmington, Delaw [1995)] adividuals tested for reactivity against a nological escape mutations in the tip c	20(V3 304-318 LAI) gp120(310-321) RIHIGPGRAFYT? 10r: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware 12rences: [Langedijk et al.(1995)] 12rences: [Langedijk et al.(1995)] 12rences: [Langedijk et al.(1995)] 13rences: [Langedijk et al.(1995)] 13rences: [Langedijk et al.(1995)]	eptides based o op [Langedijk e	? n autologous sequences t al.(1995)]	human(IgG, IgM)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
363 C311E	gp120 (V3 309-316 MN) Donor: ? References: [Warrier et al.(19	gp120(308-315) 96). Alsmadi & Till	RKRIHIGP ev(1998)]	T	IIIB infection	chimpanzee(IgG1)
	 References: [Warrier et al.(1996), Alsmadi & Tilley(1998)] NOTES: C311E: Synergistic neutralization of HIV-1 when comb C311E: A study of 6 anti-Env MAbs and their ability to MN, SF-2, and RF – C311E bound and directed lysis ag 	96), Alsmadi & Till lization of HIV-1 w Env MAbs and their E bound and directed	TES: C311E: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier et al.(1996)] C311E: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – C311E bound and directed lysis against all four strains [Alsmadi & Tilley(1998)]	MAb C108G [Wa CC against target Is [Alsmadi & Ti]	rrier et al.(1996)] cells infected with IIIB ley(1998)]	•
364 5G11	gp120(V3 loop) gp120 ? Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA References: [Moore & Sodroski(1996)] NOTES:	gp120 .ur, NCI, Frederick, I .ki(1996)]	? MD USA		?	
	 5G11: Binds to conformation sensitive epitope in the V2 reciprocal enhancement of some C1-C5 MAbs (unusual f enhances binding of V2 MAbs [Moore & Sodroski(1996)] 	ation sensitive epito f some C1-C5 MAI lAbs [Moore & Sod	5G11: Binds to conformation sensitive epitope in the V3 loop – reciprocal inhibition of other V3 loop MAbs – reciprocal enhancement of some C1-C5 MAbs (unusual for an anti-V3 MAb) and CD4 binding site MAbs – and enhances binding of V2 MAbs [Moore & Sodroski(1996)]	cal inhibition of IAb) and CD4 bi	other V3 loop MAbs - nding site MAbs - and	· +
365 110.3	gp120(V3 308-328 BRU)	gp120(312-319) QRGPGRAF	QRGPGRAF	L	BRU infected cell lysates	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_{1\kappa})$
	Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA References: [Kinney Thomas et al.(1988), Evans et al.(1989), Langedijk et al.(1992), et al.(1994)] NOTES:	ır, NCI, Frederick, I et al.(1988), Evans	MD USA s et al.(1989), Langedijk et a		Pirofski et al.(1993), Connelly	y
	 110.3: Included as a control [Evans et al.(1989)] 110.3: MAb variable region sequenced – heavy [Pirofski et al.(1993)] 	ol [Evans et al.(198 on sequenced – hea	110.3: Included as a control [Evans et al.(1989)] 110.3: MAb variable region sequenced – heavy chain: V 7138(40), D deletion, J_H4 – light chain: $V_\kappa 21(47)$, $J_\kappa 2$ [Pirofski et al.(1993)]	letion, $\mathrm{J}_H 4 - \mathrm{ligh}$	t chain: $V_{\kappa}21(47)$, $J_{\kappa}2$	2
	 110.3: An anti-idiotypic MAb generated against may associated with itself [Connelly et al.(1994)] 	MAb generated agai [Connelly et al.(199	110.3: An anti-idiotypic MAb generated against 110.3 both mimics and binds to V3, suggesting that the V3 loop may associated with itself [Connelly et al.(1994)]	pinds to V3, sugg	esting that the V3 loop	73

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
366 110.4	gp120(V3 308-328 BRU)	gp120(312-319) QRGPGRAF	QRGPGRAF	Т	BRU infected cell lysates	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_{1\kappa})$
	Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas References: [Kinney Thomas et al.(1988), Thali et al.(1992b), Langedijk et al.(1992), Thali et al.(1993), Pirofski et al.(1993), Arendrup et al.(1993), Thali et al.(1994), Boudet et al.(1994), Connelly et al.(1994), McDougal et al.(1996), Valenzuela et al.(1998), Cao et al.(1997)] NOTES:	p, Seattle WA, E. Ki as et al.(1988), Tha 993), Thali et al.(199 et al.(1997)]	inney-Thomas ıli et al.(1992b), Lange 94), Boudet et al.(1994),	dijk et al.(1992), Thal Connelly et al.(1994),	i et al.(1993), Pirofski McDougal et al.(1996),	
	 110.4: 313 P/S substitution in the V3 region disrupts binding [Thali et al.(1992b)] 110.4: MAb variable region sequenced – heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J_H2 – light 	on in the V3 region of ion sequenced – hea	disrupts binding [Thali e	et al.(1992b)] 2, D closest to DSP2.3,	2.4 and .6, $J_H 2 - light$	
	• 110.4: Primary isolates from different time points from one individual were not susceptible to neutralization by 110.4	om different time po	ints from one individual	were not susceptible to	neutralization by 110.4	
	[Arendrup et al.(1993)] • 110.4: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce	t confers resistance	to neutralization by ant	i-CD4 binding site anti	bodies does not reduce	
	neutralizing efficiency of this V3 region MAb [Thali et al.(1994)] • 110.4: An anti-idiotypic MAb generated against 110.3 also blocks binding of 110.4 [Connelly et al.(1994)]	this V3 region MAt MAb generated agai	o [Thali et al.(1994)] Inst 110.3 also blocks bi	nding of 110.4 [Connel	ly et al.(1994)]	
	 110.4: Neutralizes HIV-1 LAI [McDougal et al.(1996)] 110.4: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11-20 is through inhibition of viral binding 	LAI [McDougal et AI in CFM cells by	al.(1996)] anti-V3 MAbs 110 4 an	d N11-20 is through inl	hihition of viral hinding	
	to the cell [Valenzuela et al.(1998)] 110 A: Virus with the VI V2 Lond deleted was visible and more successible to neutralization by CDA; MAb 17b, and	al.(1998)]	e viable and more custom	atible to poutrolization !	by CD4: MAB 17b and	
	anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao et al.(1997)]	4, and 110.4, but not	t to and CD4BS MAb F	105 or sCD4 [Cao et al.	.(1997)]	

MAb ID Location	Location	WEAU	Sequence	Neutralizing Immunogen	Immunogen	Species(Isotype)
367 110.5	gp120(V3 308-328 BRU)	gp120(312-319) QRGPGRAF	QRGPGRAF	L	BRU infected cell	$\mathrm{murine}(\mathrm{IgG}_{1\kappa})$
	Donor: E. Kinney-Thomas or Genetic Systems, Seattle WA	r Genetic Systems, S	seattle WA		1) buco	
)			

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

NOLES

- 110.5: Did not induce dissociation of gp120, as sCD4 did discrepancy with [Poignard et al.(1996a)], that was suggested to be due to MAb interference with detection, as the gpl 20-MAb complex was denatured in the Poignard
- 110.5: Binding insensitive to gp120 reduction [Cordell et al.(1991)]
- 110.5: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 110.5: Variable region sequenced heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J_H2 light chain: $V_{\kappa}21, J_{\kappa}2$ [Pirofski et al.(1993)]
- 110.5: Thrombin cleavage of V3 loop between R-315 and A-316 abrogates binding can inhibit C4 region antibody [Moore et al.(1993b)] which has conformational requirements (G3-299) - binding to native gp120 100-300 fold greater than to denatured
- 110.5: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization Jr. et al.(1988), Klasse et al.(1993a)] resistance to conformationally sensitive neutralizing MAbs - neutralization efficiency of 110.5 is not affected [Reitz
- 110.5: Pretreatment of HX10-infected H9 cells with sCD4 decreases signal from 110.5 at 37 degrees due to dissociation of gp120-gp41 [Sattentau et al.(1995)]
- 110.5: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strains neutralizes cell-free Hx10 [Sattentau & Moore(1995)]
- 110.5: Reciprocal binding inhibition with other anti-V3 MAbs enhances binding of some anti-V2 MAbs binding enhanced by some CD4 binding site MAbs [Moore & Sodroski(1996)]
- 110.5: V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus. mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)]
- 110.5: Neutralizes HIV-1 LAI [McDougal et al.(1996)]
- 110.5: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)]
- 110.5: A high avidity antibody as assessed by urea elution
- 110.5: Viral binding inhibition by 110.5 was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 110.5: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
368 5023A	gp120(V3 311-317 BH10)	gp120(313-319)	m RgPGRAF	Т	15 mer synthetic BH10 V3 peptide	murine(IgG)
	 Donor: Paul Durda, Du Pont de Nemours and Co References: [Langedijk et al.(1991), D'Souza et: NOTES: 5023A: Generation and Fine mapping of mur 5023A: Called 5023 – Langedijk also has an later 1700013 	de Nemours and Co (1991), D'Souza et : ine mapping of mur ngedijk also has an l	 Donor: Paul Durda, Du Pont de Nemours and Co References: [Langedijk et al.(1991), D'Souza et al.(1991), Back et al.(1993), Rovinski et al.(1995)] NOTES: 5023A: Generation and Fine mapping of murine MAbs [Langedijk et al.(1991)] 5023A: Called 5023 – Langedijk also has an MAb called 5023B – strong cross-reactive neutralizing MAb [D'Souza 	ovinski et al.(19 91)] ss-reactive neut	95)] ralizing MAb [D'Souza	
	et al.(1991)] • 5023A: Called 5023 – La 675 (I/M) in gp41 interfe et al.(1993)]	angedijk also has ar ere with 5023s neut	et al.(1991)] 5023A: Called 5023 – Langedijk also has an MAb called 5023B – gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662-675 is ELDKWANLWNWFNI [Back et al.(1993)]	mino acid subs	titutions 668 (N/S) and WANLWNWFNI [Back	
	et al.(1993)]5023A: Called 5023 in thiusing unprocessed gp160	is paper – Used to pr glycoprotein as an i	et al.(1993)] 5023A: Called 5023 in this paper – Used to precipitate gp160 in immunoblots in a study examining the feasibility of using unprocessed gp160 glycoprotein as an immunogen [Rovinski et al.(1995)]	s in a study exa 95)]	mining the feasibility of	
369 178.1	gp120(V3 305-309 BH10) gp120(309-313) KSiRI Donor: C. Thiriart, Smith Kline and MRC AIDS reagent project References: [Thiriart et al.(1989), Back et al.(1993), Moore & HNOTES:	gp120(309-313) ne and MRC AIDS: 989), Back et al.(199	gp120(V3 305-309 BH10) gp120(309-313) KSiRI L Donor: C. Thiriart, Smith Kline and MRC AIDS reagent project References: [Thiriart et al.(1989), Back et al.(1993), Moore & Ho(1993), Cook et al.(1994)] NOTES:	L et al.(1994)]	yeast rgp160 IIIB	$\operatorname{murine}(\operatorname{Ig} \operatorname{G}_{2a})$
	 178.1: Reacts to gp120 ar 178.1: Called 178.1.1 – co 	nd gp160 in RIPA E onformational, does	178.1: Reacts to gp120 and gp160 in RIPA EIA and immunoblot [Thiriart et al.(1989)] 178.1: Called 178.1.1 – conformational, does not bind well to denatured gp120 [Moore & Ho(1993)]	al.(1989)] 20 [Moore & F	lo(1993)]	
	 178.1: gp41 amino acid substitutions 668 (N/S) and 675 (I/M) region 662-675 is ELDKWANLWNWFNI [Back et al.(1993)] 178.1: MAbs against the glycosphingolipid GalCer block HIV from the brain and colon – this MAb can inhibit gp120 bind inhibited but did not completely block MAb binding[Cook et al. 178 of the LIW Medical Because Company ADS records (ABB221) 	ubstitutions 668 (N. WANLWNWFNI [B. WANLWNWFNI] (B. glycosphingolipid C. – this MAb can in pletely block MAb I (MAB) (MAB) (MAB) (MAB)	178.1: gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662-675 is ELDKWANLWNWFNI [Back et al.(1993)] 178.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer <i>in vitro</i> – binding of GalCer to gp120 inhibited but did not completely block MAb binding[Cook et al.(1994)]	fere with 5023s fere with 5023s formally suscep in vitro – bind	1 5023s neutralization potency, susceptible CD4 negative cells – binding of GalCer to gp120	
370 5042A	gp120(V3 310-315 BH10)	gp120(312-317)	QrGPGR	T	15 mer synthetic BH10 V3 peptide	murine(IgG)
	Donor: C. Thiriart, Smith Kline and MRC AIDS reagent project References: [Langedijk et al.(1991)]	ne and MRC AIDS (1991)]	reagent project		'n	
	• 5042A: Generation and fi	ne mapping of muri	5042A: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]	1)]		

MAb ID	Location	WEAU	Sequence Neu	Neutralizing	Immunogen	Species(Isotype)
371 5025A	gp120(V3 313-317 BH10)	gp120(315-319)	pgRAF	L	15 mer synthetic BH10 V3 peptide	murine(IgG)
	Donor: Paul Durda, Du Pont de Nemours and Co References: [Langedijk et al.(1991), D'Souza et al.(1991)] NOTES:	le Nemours and Co 1991), D'Souza et a	1.(1991)]			
	5025A: Generation and fir5025: Called 5025 – strair	e mapping of murir specific weakly ne	5025A: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)] 5025: Called 5025 – strain specific weakly neutralizing [D'Souza et al.(1991)]			
372 5020	gp120(V3 311-316 BH10)	gp120(313-318)	RGPGRA	Z	15 mer synthetic BH10 V3 peptide	murine(IgG)
	 Donor: Paul Durda, Du Pont de Nemours and Co References: [Langedijk et al.(1991)] NOTES: 5020: Generation and fine mapping of murino 	e Nemours and Co 1991)] mapping of murine	 onor: Paul Durda, Du Pont de Nemours and Co eferences: [Langedijk et al.(1991)] OTES: 5020: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)] 		, r	
373 5042B	gp120(V3 310-315 BH10)	gp120(312-317)	QRGPGr	Z	15 mer synthetic BH10 V3 peptide	murine(IgG)
	 Donor: Paul Durda, Du Pont de Nemours and Co References: [Langedijk et al.(1991)] NOTES: 5042B: Generation and fine mapping of muri 	e Nemours and Co 1991)] e mapping of murin	or: Paul Durda, Du Pont de Nemours and Co erences: [Langedijk et al.(1991)] FES: 5042B: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]			
374 5025B	gp120(V3 310-316 BH10)	gp120(312-318)	QRGPGra	Z	15 mer synthetic BH10 V3 peptide	murine(IgG)
	Donor: Paul Durda, Du Pont de Nemours and Co References: [Langedijk et al.(1991)] NOTES:	e Nemours and Co 1991)]	nor: Paul Durda, Du Pont de Nemours and Co erences: [Langedijk et al.(1991)] TES:		;	
375 5023B	gp120(V3 309-316 BH10)	gp120(311-318)	IQRGPGra	Z	15 mer synthetic	murine(IgG)
	 Donor: Paul Durda, Du Pont de Nemours and Co References: [Langedijk et al.(1991)] NOTES: 5023B: Generation and fine mapping of muri 	e Nemours and Co 1991)] e mapping of murin	or: Paul Durda, Du Pont de Nemours and Co erences: [Langedijk et al.(1991)] TES: 5023B: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]		nino es popular	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
376 110.1	gp120(V3 316-322) gp120(318-324) A Donor: F. Traincard, Pasteur Institute, France References: [Moore et al.(1993b), Moore et al.(199aet al.(1996a), Wyatt et al.(1997), Parren et al.(1998)] NOTES:	gp120(318-324) ur Institute, France 1993b), Moore et al.(1'997), Parren et al.(1998)	gp120(V3 316-322) gp120(318-324) AFVTIGK L Donor: F. Traincard, Pasteur Institute, France References: [Moore et al.(1993b), Moore et al.(1994c), Sattentau & Moore(1995), Moore et al.(1996a), Wyatt et al.(1997), Parren et al.(1998)] NOTES:		recombinant gp120 & Sodroski(1996), Poignard	murine
	 110.I: Binds to carboxy-terminal side of the 110.I: Binds equally well to monomer and c& Moore(1995)] 110.I: Reciprocal binding inhibition with MAbs – binding enhanced by some anti-C 110.I: Epitope suggested to be RAFVTIC increase gp120 dissociation from virus, not on anti-V2 MAbs [Poignard et al.(1996a)] 110.I: Binds both gp120 and soluble gp12 by gp41 binding [Wyatt et al.(1997)] 110.I: The MAb and Fab binding to the oli suggest that neutralization is determined to parren et al.(1998)] 	terminal side of the V3 I to monomer and oligo ng inhibition with othe sed by some anti-CD4 d to be RAFVTIGK - dition from virus, mimi nard et al.(1996a)] 0 and soluble gp120+; et al.(1997)] b binding to the oligor ion is determined by the	110.I: Binds to carboxy-terminal side of the V3 loop – inhibits binding of C4 region MAb G3-299 [Moore et al.(1993b)] 110.I: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains [Sattentau & Moore(1995)] 110.I: Reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and enhances binding of some anti-V2 MAbs – binding enhanced by some anti-CD4 binding site MAbs [Moore & Sodroski(1996)] 110.I: Epitope suggested to be RAFVTIGK – V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)] 110.I: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)] 110.I: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	gion MAb G3-299 nt neutralization of and enhances bi Sodroski(1996)] 0.5, and 110.1 co p41 epitope for No p41 epitope for No p541 epitope for No p	of IMoore et al.(1993b)] of lab strains [Sattentau nding of some anti-V2 nuld each significantly Ab 50-69, in contrast epitope is not blocked ly correlated – authors spective of the epitope	
377 110.J	gp120(V3 loop) gp120 Donor: F. Traincard, Pasteur Institute, France	gp120 r Institute, France	?		?	
	References: [Thali et al.(1993), Moore & Sodroski(1996)] NOTES: • 110.J: Inhibits sCD4-inducible anti-CD4 binding site N • 110.J: Binds to carboxy-terminal side of the V3 loop C4 MAbs – and reciprocal enhanced binding of some Sodroski(1996)]	93), Moore & Sodrosl ducible anti-CD4 bind y-terminal side of the ocal enhanced binding	 eferences: [Thali et al.(1993), Moore & Sodroski(1996)] OTES: 110.J: Inhibits sCD4-inducible anti-CD4 binding site MAb 48d [Thali et al.(1993)] 110.J: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs [Moore & Sodroski(1996)] 	[1993)] inhibition with c inti-CD4 binding	with other anti-V3 and anti- inding site MAbs [Moore &	
378 G3-1472	gp120(V3 loop) gp120 Donor: M. Fung References: [Moore & Sodroski(1996)] NOTES:	gp120 roski(1996)]	3		.9	
	• G3-1472: Binds to carboxy-terminal side of the V3 l anti-C4 MAbs – reciprocal enhanced binding of some inhibited by anti-C4 MAbs [Moore & Sodroski(1996)]	boxy-terminal side of ocal enhanced binding Abs [Moore & Sodrosl	G3-1472: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs – binding inhibited by anti-C4 MAbs [Moore & Sodroski(1996)]	ding inhibition v anti-CD4 binding	vith other anti-V3 and site MAbs – binding	

MAb ID	Location	WEAU	Sequence	Neutralizing	Neutralizing Immunogen	Species(Isotype)
379 AG1121	gp120(V3 loop) gp120 ? Donor: AGMED, Inc, Bedford MA, commercial References: [Sullivan et al.(1995), Cao et al.(1997)] NOTES: AG1121: Also called 1121 AG1121: Recognizes monomeric gp120 from T-89.6 was three-fold less sensitive to neutralizatio. AG1121: Called 1121 – Virus with the V1-V2 loom MAb 17b, and anti-V3 MAbs 1121, 9284, and 1	gp120 3edford MA, commerct al.(1995), Cao et al.(1995) ad 1121 es monomeric gp120 less sensitive to neutr	20(V3 loop) gp120 ? nor: AGMED, Inc, Bedford MA, commercial erences: [Sullivan et al.(1995), Cao et al.(1997)] TES: AG1121: Also called 1121 AG1121: Recognizes monomeric gp120 from T-cell adapted line HXBc2 and primary isolate 89.6 equally well, but	L HXBc2 and primary isol	? ate 89.6 equally well, bu	1
380 110.6	gp120(V3 BRU) gp120(313-320) RGPGRAFV L (w Donor: AGMED, Inc, Bedford MA, commercial References: [Kinney Thomas et al.(1988), Pirofski et al.(1993), Langedijk et al.(1992)]	V3 MAbs 1121, 9284	89.6 was three-fold less sensitive to neutralization by AG1121 than HXBc2 [Sullivan et al.(1995)] AG1121: Called 1121 – Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao et al.(1997)]	n HXBc2 [Sullivan et al. ble and more susceptible ad CD4BS MAb F105 or	ptible to neutralization by CD4i 105 or sCD4 [Cao et al.(1997)]	1

MAb ID	Location	WEAU	Sequence	Neutralizing	Neutralizing Immunogen	Species(Isotype)
381 BAT123	gp120(V3 308-322 HXB2) gp120(308-324) RIRIQRO Donor: Tanox Biosystems Inc and David Ho, ADARC, NY	gp120(308-324) and David Ho, AD	gp120(308-324) RIRIQRGPGRAFVTIGK and David Ho, ADARC, NY	L	Inact IIIB	$\mathrm{murine}(\mathrm{Ig} G_{1\kappa})$
	References: [Fung et al.(198	(7), Liou et al.(198	References: [Fung et al.(1987), Liou et al.(1989), Fung et al.(1990), Moore & Ho(1993), Safrit et al.(1993), Thali	& Ho(1993), S	Safrit et al.(1993), Thali	
	et al.(1993), Pirofski et al.(1993), Gauduin et al.(1995), Sattentau & Moore(1995), Poignard et al.(1996a), Andrus	993), Gauduin et a	l.(1995), Sattentau & Moore(1995), Poignar	d et al.(1996a), Andrus	
	et al.(1998), Parren et al.(1998), Gauduin et al.(1998)] NOTES:), Gauduin et al.(19	98)]			
	 BAT123: Also called BAT 	F-123 – CGP 47 439	 BAT123: Also called BAT-123 − CGP 47 439 is a BAT123 chimera that has a human IgG₁ Fc domain 	a human IgG_1	Fc domain	
	 BAT123: Anti-idiotypic N 	1Ab, AB19-4i, stim	BAT123: Anti-idiotypic MAb, AB19-4i, stimulates anti-anti-ID which neutralizes MN and IIIB [Fung et al.(1990)]	ralizes MN and	IIIB [Fung et al.(1990)]	
	 BAT123: Called BAT-123 	– conformational,	BAT123: Called BAT-123 - conformational, does not bind well to denatured gp120 -	d gp120 – not r	not reactive with SF-2 gp120	
	 does not inhibit HIV-1 s 	era from binding to	- does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]	3)]		
	 BAT123: Passive transfer 	to Hu-PBS-SCID	BAT123: Passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free	st challenge wi	th homologous cell-free	

- virus [Safrit et al.(1993)]
- BAT123: Variable region sequenced heavy chain: V 3660-SB32, D unknown, J_H3 light chain: $V_{\kappa}21$, $J_{\kappa}2$ [Pirofski et al.(1993)]
- BAT123: Passive transfer of BAT123 to hu-PBL-SCID mice 1 hour prior to inoculation with HIV-1 LAI, or up to strain LAI [Gauduin et al.(1995)] four hours post-exposure, could protect mice from infection – the protection, like the MAb, was specific for the viral
- BAT123: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strair [Sattentau & Moore(1995)]
- BAT123: Epitope described as RGPGRAFVTIGK V3 MAbs 9284, BAT123, 110.5, and 110.I could each signifgp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)] icantly increase gp120 dissociation from virus (BAT123 less so than the others), mimicking sCD4, and expose the
- BAT123: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIVinfection [Andrus et al.(1998)] have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post 1 LAI in hu-PBL-SCID mice, but declined to 50% if delivered 60 min post-exposure, and similar time constraints
- BAT123: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated epitope [Parren et al.(1998)] authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the
- BAT123: Post-exposure passive transfer of murine BAT123 can confer protection to hu-PBL-SCID mice challenged suggesting that the protection is mediated by complement - the protective ability of BAT123 is lost when mice were an IgG₂ MAb might perform better [Gauduin et al.(1998)] treated with cobra venom factor, which inactivates serum complement – IgG₁ does not fix complement efficiently so with HIV-1 LAI – this protection is not elicited by CGP 47 439, a BAT123 chimera that has a human IgG₁ Fc domain,

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
382 CGP 47 439	gp120(V3 tip)	gp120(308-324)	?	Т	IIIB gp120	BAT123-human Ig chimera
	 Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Liou et al.(1989), Safrit et al.(1993), Gunthard NOTES: CGP 47 439: passive transfer to Hu-PBS-SCID mice covirus – BAT123-human Ig chimera [Safrit et al.(1993)] CGP 47 439: PhaseI/IIA clinical trial studying multidose – GP 47 439 was well tolerated, serum t_{1/2} was 8-16 da [Gunthard et al.(1994)] CGP 47 439: Post-exposure passive transfer of murine lenged with HIV-1 LAI – this protection is not elicited be complement – the protective ability of BAT123 is lost w tivates serum complement – in this circumstance complet al.(1998)] CGP 47 439: Review of passive immunotherapy, sumr [Jacobson(1998)] 	or: Tanox Biosystems Inc and David Ho, ADARC, NY rences: [Liou et al.(1989), Safrit et al.(1993), Gunthar IES: CGP 47 439: passive transfer to Hu-PBS-SCID mice co virus – BAT123-human Ig chimera [Safrit et al.(1993)] CGP 47 439: PhaseI/IIA clinical trial studying multidose – GP 47 439 was well tolerated, serum $t_{1/2}$ was 8-16 de [Gunthard et al.(1994)] CGP 47 439: Post-exposure passive transfer of murine lenged with HIV-1 LAI – this protection is not elicited by complement – the protective ability of BAT123 is lost witvates serum complement – in this circumstance complet al.(1998)] CGP 47 439: Review of passive immunotherapy, sum [Jacobson(1998)]	 Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Liou et al.(1989), Safrit et al.(1993), Gunthard et al.(1994), Gauduin et al.(1998), Jacobson(1998)] NOTES: CGP 47 439: passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus – BAT123-human Ig chimera [Safrit et al.(1993)] CGP 47 439: PhaseI/IIA clinical trial studying multidose tolerability, immunogenicity and pharmacokinetic responses – GP 47 439 was well tolerated, serum t_{1/2} was 8-16 days, and a virus burden reduction was noted in some patients [Gunthard et al.(1994)] CGP 47 439: Post-exposure passive transfer of murine BAT123 can confer protection to hu-PBL-SCID mice challenged with HIV-1 LAI – this protection is not elicited by CGP 47 439, suggesting that the protection is mediated by complement – the protective ability of BAT123 is lost when mice were treated with cobra venom factor, which inactivates serum complement – in this circumstance complement activation provided a protective advantage [Gauduin et al.(1998)] CGP 47 439: Review of passive immunotherapy, summarizing [Gunthard et al.(1994)] in relation to other studies [Jacobson(1998)] 	duin et al.(1998), ainst challenge w nogenicity and ph rden reduction water protection to he ggesting that the pated with cobrave provided a protection detal.(1994)] in detal.(1994)] in	[1998], Jacobson(1998)] enge with homologous cell-free and pharmacokinetic responses tion was noted in some patients on to hu-PBL-SCID mice chalat the protection is mediated by obra venom factor, which inacprotective advantage [Gauduin protective advantage studies	
383 10F10	gp120(V3 MN) gp120(308-322) RKRIHI Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Duarte et al.(1994)] NOTES: • 2C4: Putative epitope lies within IHIGPGRAFYT – gene MN and SC (TRSIHIGPGRAFYTT) peptides, lower af	gp120(308-322) ns Inc and David Ho, AI L.(1994)] e lies within IHIGPGRAI IGPGRAFYTT) peptide	gp120(308-322) RKRIHIGPGRAFYTT L Peptide nor: Tanox Biosystems Inc and David Ho, ADARC, NY erences: [Duarte et al.(1994)] TES: 2C4: Putative epitope lies within IHIGPGRAFYT – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAFYTT) peptides, lower affinity for SF2 [Duarte et al.(1994)]	L tope polypeptide in arte et al.(1994)]	Peptide nmunization-recognize	$\operatorname{murine}(\operatorname{Ig} G_1)$
384 2C4	gp120(V3 MN) pp120(308-322) RKRIHI Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Duarte et al.(1994)] NOTES: 2C4: Putative epitope lies within IHIGPGRAFYT – ner polypeptide immunization – recognize MN and SC (TR et al.(1994)]	gp120(308-322) ns Inc and David Ho, AI ul.(1994)] se lies within IHIGPGR A zation – recognize MN a	gp120(308-322) RKRIHIGPGRAFYTT L (MN) Peptide or: Tanox Biosystems Inc and David Ho, ADARC, NY erences: [Duarte et al.(1994)] TES: 2C4: Putative epitope lies within IHIGPGRAFYT – neutralizes MN, not IIIB and SF2 – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAFYTT) peptides, lower affinity for SF2 [Duarte et al.(1994)]	L (MN) IIIB and SF2 – ge T) peptides, lower	Peptide nerated by multi-epitope affinity for SF2 [Duarte	$\operatorname{murine}(\operatorname{Ig} \operatorname{G}_{2a})$

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
385 19b	gp120(V3) gp120(310-322) -I—-G- Donor: James Robinson, University of Connecticut, Storrs	gp120(310-322) -I—G-FY-T iversity of Connecticut, Storrs	-I—-G–FY-T cut, Storrs	L	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} \operatorname{G}_1)$

NOTES:

et al.(1998), Trkola et al.(1998)]

• 19b: V3 loop binding MAb that is more broadly clade cross-reactive than most (binds to 19/29 clade B and 10/12 clade E gp120s) [Moore et al.(1994b)]

et al.(1997), Fouts et al.(1997), Ugolini et al.(1997), Boots et al.(1997), Parren et al.(1997b), Mondor et al.(1998), Parren

et al. (1995a), Moore & Ho(1995), Gauduin et al. (1996), Wu et al. (1996), Trkola et al. (1996a), D'Souza et al. (1997), Binley **References:** [Scott Jr et al. (1990), Moore et al. (1994b), Moore et al. (1994a), Sattentau (1995), Moore et al. (1995b), Moore

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

MAb ID	
Location	
WEAU	
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Neutralizi	
izing Immunogen	
Species(Isotype)	

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NOTES:

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

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
386 G3-523	gp120(V3 308-322) gp120(310-322) RIQRGPGRAFV Donor: James Robinson, University of Connecticut, Storrs References: [Matsushita et al.(1988), Jagodzinski et al.(1996)] NOTES: • G3-523: The sulfated polysaccharide curdlan sulfate (CRDS) bind virus – CRDS inhibits G3-523 binding [Jagodzinski et al.(1996)]	gp120(310-322) versity of Connecticu .(1988), Jagodzinski 'saccharide curdlan sı -523 binding [Jagod:	gp120(310-322) gp120(310-322) RIQRGPGRAFVTIGK ? nor: James Robinson, University of Connecticut, Storrs ferences: [Matsushita et al.(1988), Jagodzinski et al.(1996)] YTES: G3-523: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits G3-523 binding [Jagodzinski et al.(1996)]	elope of T-tropi	? viruses and neutralizes	murine
387 4G10	gp120(V3 308-322 LAI)	gp120(310-322)	gp120(310-322) RIQRGPGRAFVTGK		V3-loop HBcAg hybrid	murine
	 Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany References: [von Brunn et al.(1993)] NOTES: 4G10: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn et al.(1993) 4G10: NIH AIDS Research and Reference Reagent Program: 2534 	nn, Max-von-Pettenk (1993)] (3-loop sequence fusch and Reference Re	 nor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany ferences: [von Brunn et al.(1993)] YTES: 4G10: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn et al.(1993)] 4G10: NIH AIDS Research and Reference Reagent Program: 2534 	lians-Universita munogenicity [t Munchen, Germany on Brunn et al.(1993)]	
388 5F7	gp120(V3 308-322 LAI)	gp120(310-322)	gp120(310-322) RIQRGPGRAFVTGK		V3-loop HBcAg hybrid	murine
	 Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany References: [von Brunn et al.(1993)] NOTES: 5F7: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn et al.(1993)] 5F7: NIH AIDS Research and Reference Reagent Program: 2533 	nn, Max-von-Pettenk (1993)] -loop sequence fusec	 nor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany erences: [von Brunn et al.(1993)] TES: 5F7: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn et al.(1993)] 5F7: NIH AIDS Research and Reference Reagent Program: 2533 	lians-Universita	t Munchen, Germany n Brunn et al.(1993)]	
389 10/54	gp120(V3 311-321 HXB10) gp120(313-323) RGPGRAFVTIG L (HXB10) rgp120 BH10 Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany References: [McKeating et al.(1992a), McKeating et al.(1993a), McKeating et al.(1993b)] NOTES:	gp120(313-323) RGPGRAFVTIGnn, Max-von-Pettenkofer-Institut, Lud-(1992a), McKeating et al.(1993a), Mc	RGPGRAFVTIG cofer-Institut, Ludwig-Maximi g et al.(1993a), McKeating et a	L (HXB10) lians-Universita d.(1993b)]	rgp120 BH10 t Munchen, Germany	$\operatorname{rat}(\operatorname{IgG}_1)$
	10/54: Binding to virion §10/54: Studied in the con	gp120 enhanced by stext of a neutralization	10/54: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)] 10/54: Studied in the context of a neutralization escape mutant [McKeating et al.(1993a)])] et al.(1993a)]		
390 10/36e	gp120(V3 311-321 HXB10) gp120(313-323) RGPGRAFVTIG L (HXB10) rgp120 BH10 Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany References: [McKeating et al.(1992a), McKeating et al.(1993b)] NOTES: • 10/36e: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)]	gp120(313-323) RGPGRAFVTIGnn, Max-von-Pettenkofer-Institut, Ludv(1992a), McKeating et al.(1993b)] gp120 enhanced by sCD4 [McKeating	10(V3 311-321 HXB10) gp120(313-323) RGPGRAFVTIG I or: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilia rences: [McKeating et al.(1992a), McKeating et al.(1993b)] [TES: 10/36e: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)]	L (HXB10) lians-Universita	rgp120 BH10 t Munchen, Germany	$\operatorname{rat}(\operatorname{IgG}_{2a})$

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
391 11/85b	gp120(V3 311-321 HXB10) gp120(313-323) RGPGRAFV7 Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, I References: [McKeating et al.(1992a), McKeating et al.(1993b)] NOTES: • 11/85b: Binding to virion gp120 enhanced by sCD4 [McKea	gp120(313-323) n, Max-von-Petten (1992a), McKeatin gp120 enhanced by	gp120(V3 311-321 HXB10) gp120(313-323) RGPGRAFVTIG L (HXB2) rgp120 BH10 Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany References: [McKeating et al.(1992a), McKeating et al.(1993b)] NOTES: • 11/85b: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)]	L (HXB2) ians-Universita		$\operatorname{rat}(\operatorname{IgG}_{2b})$
392 loop 2	gp120(V3) gp120(311-322) SISGPG Donor: D. Burton, Scripps Research Institute, La Jolla, CA References: [Barbas III et al.(1993), Moore et al.(1994b), Parren et al.(1997b), Parren & Burton(1997), Mondor et al.(NOTES:	gp120(311-322) search Institute, La (1993), Moore et a Burton(1997), Mor	gp120(V3) gp120(311-322) SISGPGRAFYTG L HIV-1 infection Donor: D. Burton, Scripps Research Institute, La Jolla, CA References: [Barbas III et al.(1993), Moore et al.(1994b), Wu et al.(1996), Ditzel et al.(1997), Ugolini et al.(1997), Parren et al.(1997b), Parren & Burton(1997), Mondor et al.(1998), Parren et al.(1998)] NOTES:	L izel et al.(1997) 998)]		human Fab
	 loop2: Also known as Loo loop 2: Sequences of the h loop 2: Called Loop 2 – et al.(1994b)1 	p 2, IgG1 Loop 2 veavy and light chair shows modest creations.	loop2: Also known as Loop 2, IgG1 Loop 2 was a obtained by engineering Fab loop2 into an IgG1 molecule loop 2: Sequences of the heavy and light chain Fab variable regions were generated [Barbas III et al.(1993)] loop 2: Called Loop 2 – shows modest cross-reactivity among B clade gp120s, little outside B clade [et al.(1994b)]	ab loop2 into an herated [Barbas pl 20s, little o	l into an IgG1 molecule Barbas III et al.(1993)] little outside B clade [Moore	
	 loop 2: MIP-1α binding to inhibition [Wu et al.(1996)] loop 2: Binds to gp120 from 	CCR-5 expressing MN and SF2 bu	loop 2: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of loop 2 blocks this inhibition [Wu et al.(1996)] loop 2: Binds to gp120 from MN and SF2 but not LAI [Ditzel et al.(1997)])-sCD4 – bindi	ng of loop 2 blocks this	
	 loop 2: Viral binding inhi MAbs tested showed some loop 2: Fnitone is probable 	bition by loop 2 N correlation except	loop 2: Viral binding inhibition by loop 2 MAb or Fab was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)] loop 2: Enitone is probably GPGRAF – binds to 10/17 US clade R monomeric on 120s – IoG1 form can neutralize	n neutralization	lization (all other neutralizing	
	MN and 2 primary isolates tested [Parren & Burton(1997)] • loop 2: Neutralizes TCLA strains but not primary isolates	tested [Parren & F strains but not prir	MN and 2 primary isolates tested [Parren & Burton(1997)] loop 2: Neutralizes TCLA strains but not primary isolates [Parren et al.(1997b)]	(p)]		
	• loop 2: The rank order of l b14 > b13 > D0142-10 >	FAb binding affinit DA48 > L17) was	loop 2: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = D08i > b11 > b3 > b14 > b13 > D0142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (2B2 > b12 > D0142 10 > 1 cm 3 > b11 > 117 > b6 > D08i > b14 > DA48 > b2 > b12 > cm 4 binding to	> 3B3 > b12 nding affinity to	> b12 = DO8i > b11 > b3 > finity to the mature oligomeric	
	oligomeric form and neutrandetermined by the fraction	alization were corn of Ab sites occupi	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 [act 1] loon 2 is only 2-fold greater than monovalent Fab loon 2 is only 2-fold greater than monovalent Fab loon 2 is only 2-fold greater than monovalent Fab loon 2.	- authors sugge e epitope – bind he IoG1 form r	s suggest that neutralization is b – binding affinity of divalent form may bind with only one	
	arm [Parren et al.(1998)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
393 41.1	pp120(V3 dis HXB10) Donor: J. Cordell, Institute for Cancer Research, Sutton, Surrey, UK References: [McKeating et al.(1992a), McKeating et al.(1993b), J Armstrong & Dimmock(1996), Armstrong et al.(1996), Jeffs et al.(19 NOTES: 41.1: Also called ICR41.1i and ICR41 41.1: The gp41 mutation 582(Ala to Thr) results in conformat resistance to conformationally sensitive neutralizing MAbs – net Jr. et al.(1988), Klasse et al.(1993a)] 41.1: Called ICR41.1i – Kinetics of neutralization studied – no la lags of 5 and 15 min respectively – neutralization mediated by neutralization of the three MAbs studied – acts with multi-hit kir etlls at 24 degrees C or below [Armstrong & Dimmock(1996)] 41.1: Called ICR41.1i – Neutralization occurs by blocking a po F58 [Armstrong et al.(1996)] 41.1: Deletion of the V1V2 regions did not affect anti-V3 Abs [Jeffs et al.(1996)] 41.1: Viral binding inhibition by 41.1 was weakly correlated with showed some correlation except 2F5) [Ugolini et al.(1997)]	gp120 or Cancer Rese al.(1992a), M), Armstrong 6 li and ICR41 li and ICR41 li and IS2(Ala to 7 nally sensitive al.(1993a)] Kinetics of neu pectively – ne pectively – ne pectively – ne MAbs studies IGG _{2c} ? – Neut elow [Armstro Neutralization 96)] V2 regions did v2 regions did ition by 41.1 w	pgp120(V3 dis HXB10) gp120 DISCONTINUOUS L (HXB2) rgp120 BH10 r ponor: J. Cordell. Institute for Cancer Research, Sutton, Surrey, UK References: [McKeating et al.(1992a), McKeating et al.(1993b), Klasse et al.(1993a), McLain & Dimmock(1994), Armstrong & Dimmock(1996), Armstrong et al.(1996), Jeffs et al.(1996), Ugolini et al.(1997)] NOTES: • 41.1: Also called ICR41.1i and ICR41 • 41.1: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralization MAbs – neutralization efficiency of 41.1 is not affected [Reitz Jr. et al.(1988), Klasse et al.(1993a)] • 41.1: Called ICR41.1i – Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – neutralization mediated by 3 molecules of IgG per virion – most efficient at neutralization of the three MAbs studied – acts with multi-hit kinetics [McLain & Dimmock(1994)] • 41.1: Called ICR41.1i – Neutralization was affected if the Ab was added after the virus bound to the host cells at 24 degrees C or below [Armstrong & Dimmock(1996)] • 41.1: Called ICR41.1i – Neutralization occurs by blocking a post-fusion internalization event, in contrast to MAb F58 [Armstrong et al.(1996)] • 41.1: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)] • 41.1: Viral binding inhibition by 41.1 was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]	L (HXB2) et al.(1993a), l golini et al.(199 changes in gp12 ation efficiency ation efficiency ation efficiency flecules of IgG p lecules of IgG p lecules after b was added after b was added after to bind when c ralization (all other	rgp120 BH10 r McLain & Dimmock(1994), 997)] 120 that confer neutralization y of 41.1 is not affected [Reitz CR 39.13g and ICR 41.1i have per virion – most efficient at nmock(1994)] ter the virus bound to the host ion event, in contrast to MAb compared to intact rec gp120 other neutralizing MAbs tested	rat(IgG _{2a}) 394), 394), ation Reitz have nt at host host host p120 ested
394 DO142-10	 gp120(V3 MN) gp120 SISGPGRAFYTG Donor: J. Cordell, Institute for Cancer Research, Sutton, Surrey, UK References: [Ditzel et al.(1997), Parren et al.(1997b), Parren & Burt NOTES: D0142-10: Phage expression libraries panned against MN pepti MN gp120, but not a primary isolate rec gp120 [Ditzel et al.(199 D0142-10: Neutralizes TCLA strains but not primary isolates [F D0142-10: Binds to gp120 MN and an MN V3 peptide with e JRCSF less well, and to IIIB gp120 not at all [Parren & Burton(1 of 100) D0142-10: The rank order of FAb binding affinity to monomeric b14 > b13 > D0142-10 > DA48 > L17) was markedly different form (3B3 > b12 > D0142-10 > Loop 2 > b11 > L17 > b6 > oligomeric form and neutralization were correlated for both Fabs determined by the fraction of Ab sites occupied on a virion irres 	gp120 or Cancer Rese 77), Parren et a ssion libraries nary isolate rec CLA strains b 20 MN and a 11B gp120 not er of FAb bindi > DA48 > L1 42-10 > Loop ralization wer n of Ab sites o	gp120(V3 MN) gp120 SISGPGRAFYTG L HIV-1 infection https: References: [Ditzel et al.(1997), Parren et al.(1997b), Parren & Burton(1997), Parren et al.(1998)] NOTES: DO142-10: Phage expression libraries panned against MN peptide were used to select Fab DO142-10 – Fab binds MN gp120, but not a primary isolate rec gp120 [Ditzel et al.(1997)] DO142-10: Neutralizes TCLA strains but not primary isolates [Parren et al.(1997b)] DO142-10: Binds to gp120 MN and an MN V3 peptide with equal affinity, but binds a consensus B peptide and JRCSF less well, and to IIIB gp120 not at all [Parren & Burton(1997)] DO142-10: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b14 > b13 > DO142-10 > DA48 > L17) was markedly different that FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	L 97), Parren et al. ere used to select et al.(1997b)] affinity, but binds (Loop 2 > 3B3) (Loop 2 > 3B3 FAb binding affin b b14 > DA48 MAbs – authors e of the epitope [HIV-1 infection 1 ul.(1998)] cct Fab DO142-10 – Fab binds ids a consensus B peptide and 3 > b12 = DO8i > b11 > b3 > finity to the mature oligomeric 8 > b3 > b13) and binding to s suggest that neutralization is [Parren et al.(1998)]	human Fab(IgG ₁) binds and and b3 > neric neric on is

		395 257-D	MAb ID
References: [Gorny et al.(19	Donor: Susan Zolla-Pazner (NYU Med. Center)	gp120(V3 MN)	Location
91), D'Souza et al.(19	(NYU Med. Center)	gp120(309-313)	WEAU
References: [Gorny et al. (1991), D'Souza et al. (1991), Karwowska et al. (1992b), Gorny et al.		KRIHI	Sequence
		L	Neutralizing
(1993), Cavacini et al.(1993a		HIV-1 infection	Neutralizing Immunogen
3a) ,		$\operatorname{human}(\operatorname{IgG}_{1\lambda})$	Species(Isotype)

• 257-D: Also called 257, 257-2-D-IV and 257-D-IV

• 257-D: Called 257-2-D-IV – potent neutralizing MAb [D'Souza et al.(1991)]

et al.(1997), Stamatatos et al.(1997), Hill et al.(1997), LaCasse et al.(1998), Yang et al.(1998), Gorny et al.(1998)] ten et al.(1995a), Schutten et al.(1995b), Fontenot et al.(1995), Wisnewski et al.(1996), Schutten et al.(1996), Schutten Spear et al. (1993), D'Souza et al. (1994), VanCott et al. (1994), D'Souza et al. (1995), Zolla-Pazner et al. (1995), Schut-

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

MAb ID
Location
WEAU Sequence
e Neutralizin
ing Immunogen
Species(Isotype)

395 cont.

NOTES:

- 257-D: Comparable affinity for SI and NSI viruses, in contrast to MAb MN215 [Schutten et al.(1995b)]
- 257-D: 257-D is $V_H S V$ -region heavy chain usage was examined and a bias of enhanced $V_H I$ and $V_H I$ 4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- 257-D: IIIB neutralizing MAbs in vitro fail to neutralize in a mouse model in vivo [Schutten et al.(1996)]
- et al.(1997)] 257-D: Neutralized (>90%) an SI-env chimeric virus and enhanced (>200%) an NSI-env chimeric virus [Schutten
- et al.(1997)] 257-D: Binds less extensively than MAb 391-95D on the surface of HIV-1 isolates SF162 and SF128A - neutralizes less potently than 391-95D - stronger neutralization of primary macrophage targets than PBMC [Stamatatos
- 257-D: Called 257 gp120 can inhibit MIP-1 α from binding to CCR5, but this inhibitory effect is blocked by effect [Hill et al.(1997)] pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 - MAb 670 which binds in the C5 region had no
- 257-D: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse et al.(1998)] anti-V3 MAbs. The primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was
- 257-D: A neutralization assay was developed based on heminested PCR amplification of the LTR (HNPCR) LTRon tests with 6 MAbs and 5 isolates [Yang et al.(1998)] HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based
- 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
- 257-D: UK Medical Research Council AIDS reagent: ARP3023
- 257-D: NIH AIDS Research and Reference Reagent Program: 1510

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
396 4117C	gp120(V3) gp120(311-317) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Tilley et al.(1991a), Tilley et al. et al.(1993b), Alsmadi & Tilley(1998)]	gp120(311-317) her (NYU Med. Center) d.(1991a), Tilley et al.(Tilley(1998)]	gp120(311-317) IXIGPGR L n Zolla-Pazner (NYU Med. Center) [Tilley et al.(1991a), Tilley et al.(1992), di Marzo Veronese et al.(1993) Alsmadi & Tilley(1998)]	L t al.(1993), Pint	HIV-1 infection, Pinter et al.(1993a), Pinter	$\operatorname{human}(\operatorname{IgG}_{1\lambda})$
	 4117C: Potent neutralizing activet al.(1991a)] 4117C: Neutralizes SF2 and MN et al.(1993a), Tilley et al.(1992)] 4117C: Binds V3 loop – does not et al.(1993b)] 4117C: A study of 6 anti-Env MAMN, SF-2, and RF – bound and 6 	alizing activity against l F2 and MN synergistica t al.(1992)] p – does not immunopre anti-Env MAbs and their bound and directed lysis	 4117C: Potent neutralizing activity against MN, SF-2, and NY-5 – synergy with CD4BS MAb 1125H [Tilley et al.(1991a)] 4117C: Neutralizes SF2 and MN synergistically combined with anti-CD4 binding site discontinuous MAb [Pinter et al.(1993a), Tilley et al.(1992)] 4117C: Binds V3 loop – does not immunoprecipitate soluble gp120, does react with gp120 on intact virions [Pinter et al.(1993b)] 4117C: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against MN and SF2, but not IIIB and RF [Alsmadi & Tilley(1998)] 		CD4BS MAb 1125H [Tilley te discontinuous MAb [Pinter gp120 on intact virions [Pinter target cells infected with IIIB, RF [Alsmadi & Tillev(1998)]	
397 41148D	gp120(V3 MN)	gp120(309-315)	KRIHIGP	T		human(IgG1)
	Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Pinter et al.(1993b), Alsmadi & Tilley(1998)] NOTES:	er (NYU Med. Center) (1993b), Alsmadi & Tille	ey(1998)]		i i i i i i i i i i i i i i i i i i i	1101111111(45(04)
	 41148D: Neutralizes 41148D: A study of IIIB, MN, SF-2, and 41148D is 10x less e Tilley(1998)] 	less potently than 4117C 6 anti-Env MAbs and th RF – bound and directed fficient at neutralization,	41148D: Neutralizes less potently than 4117C, reacts with MN, IIIB, SF2 [Pinter et al.(1993b)] 41148D: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against strains IIIB, MN, SF-2, comparable to 4117C, however 41148D is 10x less efficient at neutralization, showing ADCC and neutralization don't always correlate [Alsmadi & Tilley(1998)]	Pinter et al.(1993b)] ADCC against target N, SF-2, comparable ation don't always c	(3b)] urget cells infected with able to 4117C, however ys correlate [Alsmadi &	
398 453-D	gp120(V3 MN) gp120(311-317) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Gorny et al.(1993), VanCott et al.(gp120(311-317) her (NYU Med. Center) .(1993), VanCott et al.(19	gp120(V3 MN) gp120(311-317) IHIGPGR Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Gorny et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)] NOTES:	L	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\lambda})$
	 453-D: Neutralizes N 453-D: Moderate hor 453-D: Called 453, higher valency con 	IN – binds SF2: IYIGPC nologous neutralization, pitope described as KRI elates with stronger affin	453-D: Neutralizes MN – binds SF2: IYIGPGR – specificity: MN, SF2, NY5, RF [Gorny et al.(1993)] 453-D: Moderate homologous neutralization, moderately slow dissociation rate [VanCott et al.(1994)] 453-D: Called 453, epitope described as KRIHIGPGR – the tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot et al.(1995)]	Y5, RF [Gorny e rate [VanCott et loop was present 995)]	t al.(1993)] al.(1994)] ed in a mucin backbone	
399 504-D	gp120(V3) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Gorny et al.(1993)] NOTES: • 504-D – Neutralizes MN – binds SF2: IYIG	gp120(V3) gp120(311-317) IHIGPGR or: Susan Zolla-Pazner (NYU Med. Center) erences: [Gorny et al.(1993)] TES: 504-D – Neutralizes MN – binds SF2: IYIGPGR [Gorny et al.(1993)]	IHIGPGR 'GR [Gorny et al.(1993)]	L	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
400 418-D	gp120(V3) gp120(312-318) HIGPGF Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Karwowska et al.(1992b), Gorny et al.(1993)] NOTES: • 418-D: MN strain specific, does not cross-react with SF2, • 418-D: Neutralizes MN, does not bind to SF2 or HXB2	gp120(312-318) (NYU Med. Center) al.(1992b), Gorny et c, does not cross-react does not bind to SF2	20(V3) gp120(312-318) HIGPGRA L HIV-1 infection nor: Susan Zolla-Pazner (NYU Med. Center) erences: [Karwowska et al.(1992b), Gorny et al.(1993)] TES: 418-D: MN strain specific, does not cross-react with SF2, NY5, RF, CDC4 WM52 or HXB2 [Karwowska et al.(1992b)] 418-D: Neutralizes MN, does not bind to SF2 or HXB2 [Gorny et al.(1993)]	L 52 or HXB2 [K	HIV-1 infection arwowska et al.(1992b)]	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
401 311-11-D	gp120(V3) gp120(309-315) KRIHIGP Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Gorny et al.(1993), Spear et al.(1993), Gorny et al.(1998)] NOTES:	gp120(309-315) KRIHIGP (NYU Med. Center) 993), Spear et al.(1993), Gorny et	KRIHIGP 3), Gorny et al.(1998)]	L	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\lambda})$
	 311-11-D: Also called 311-11D 311-11-D: Neutralizes MN – binds SF2: K 311-11-D: Mediated deposition of complen rabbit anti-human IgG [Spear et al.(1993)] 	11-11D AN – binds SF2: KST osition of complemen spear et al.(1993)]	311-11-D: Also called 311-11D 311-11-D: Neutralizes MN – binds SF2: KSIYIGP [Gorny et al.(1993)] 311-11-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear et al.(1993)]	cells, enhance	d by second Ab binding,	
402 391/95-D	gp120(V3) gp120(308-322) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Gorny et al.(1993), Fontenot et al.(NOTES:	gp120(308-322) (NYU Med. Center) 993), Fontenot et al.(1	gp120(V3) gp120(308-322) RKRIHIGPGRAFYTT L Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Gorny et al.(1993), Fontenot et al.(1995), Seligman et al.(1996), Stamatatos NOTES:		HIV-1 infection et al.(1997)]	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	 391/95-D: Also called 391-95D 391/95-D: Neutralizes MN – binds to SF2, not IIIB [Gorny et 391/95-D: Competition ELISAs with serial deletions estimate peptide had higher affinity than cyclic [Seligman et al.(1996)] 391/95-D: Called 391-95D – binds more extensively than M_I SF128A – neutralizes more potently than 257-D – stronger neu – binding post-gp120-sCD4 association is related to anti-V3 / 	91-95D IN – binds to SF2, no IN – binds to SF2, no ELISAs with serial de ty than cyclic [Selign 5D – binds more exte ore potently than 257- D4 association is rela	391/95-D: Also called 391-95D 391/95-D: Neutralizes MN – binds to SF2, not IIIB [Gorny et al.(1993)] 391/95-D: Competition ELISAs with serial deletions estimated the epitope to be KRIHIGPGRAFY – unconstrained peptide had higher affinity than cyclic [Seligman et al.(1996)] 391/95-D: Called 391-95D – binds more extensively than MAb 257-D on the surface of HIV-1 isolates SF162 and SF128A – neutralizes more potently than 257-D – stronger neutralization of primary macrophage targets than PBMC – binding post-gp120-sCD4 association is related to anti-V3 Abs neutralizing capacity [Stamatatos et al.(1997)]	be KRIHIGPG e surface of HI imary macroph capacity [Stan	IIGPGRAFY – unconstrained of HIV-1 isolates SF162 and acrophage targets than PBMC [Stamatatos et al.(1997)]	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
403 412-D	gp120(V3 MN) gp120(308-322) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Gorny et al.(1993), Spear et al.(1998) NOTES: 412-D: Also called 412-10D and 412 412-D: Neutralizes MN, does not bind SF2 et al.(1993)] 412-D: Mediated deposition of complement rabbit anti-human IgG [Spear et al.(1993)] 412-D: Called 412-10D – relatively rapid dis	gp120(308-322) (NYU Med. Center 993), Spear et al.(19 10D and 412 , does not bind SF2 ition of complement Spear et al.(1993)] – relatively rapid di	gp120(V3 MN) gp120(308-322) RKRIHIGPGRAFYTT L HIV-1 infection Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Gorny et al.(1993), Spear et al.(1993), VanCott et al.(1994), Fontenot et al.(1995), Gorny et al.(1998)] NOTES: 412-D: Also called 412-10D and 412 412-D: Neutralizes MN, does not bind SF2 or HXB2 – not reactive with hexa or heptapeptides by Pepscan [Gorny et al.(1993)] 412-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear et al.(1993)] 412-D: Called 412-10D – relatively rapid dissociation and weak homologous neutralization [VanCott et al.(1994)]	L Enot et al.(1995), C exa or heptapeptid d cells, enhanced b us neutralization [HIV-1 infection forny et al.(1998)] es by Pepscan [Gorny y second Ab binding, VanCott et al.(1994)]	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
404 MN215	gp120(V3 MN) gp120(310-324) RIHIGPGRAFYT Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Schutten et al.(1995b)] NOTES: • MN215: Minimum epitope for MAB using the Dutch conser generated by EBV transformation of PBMC – displayed highe acids HIGP were essential for binding [Schutten et al.(1995b)]	gp120(310-324) (NYU Med. Center (1995b)] tope for MAB using formation of PBMC ial for binding [Schu	RIHIGPGRAFYTTKN the Dutch consensus is AFY displayed higher affinity for ten et al.(1995b)]	NEUTRALIZING; HIV-1 infection TTGE, different than defined for M or NSI than for SI glycoproteins — an	IZING; HIV-1 infection erent than defined for MN – for SI glycoproteins – amino	$\operatorname{human}(\operatorname{IgG}_1)$
405 SPBAL114	gp1 Dor Ref NO	gp120(310-319) SIHIGPGRAF (NYU Med. Center)(1995)] ring <i>in vivo</i> immunoselection of escendrup et al.(1995)]	20(V3 BAL) gp120(310-319) SIHIGPGRAF L ? nor: Susan Zolla-Pazner (NYU Med. Center) ferences: [Arendrup et al.(1995)] TES: Authors suggest that during <i>in vivo</i> immunoselection of escape virus, the V3 domain gains increasing resemblance to that of lab strains [Arendrup et al.(1995)]	L /3 domain gains ir	? creasing resemblance	murine?($\operatorname{IgG}_{2a\kappa}$)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
406 SP.SF2:104	gp120(V3 SF2) gp120(310-319) SIYIGP Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Arendrup et al.(1993), Arendrup et al.(1995)] NOTES: • SP.SF2:104: Anti-V3 antibody that could neutralize resistance of autologous virus [Arendrup et al.(1993)] • SP.SF2:104: Authors suggest that during <i>in vivo</i> immu resemblance to lab strains [Arendrup et al.(1995)]	gp120(310-319) (NYU Med. Center) .(1993), Arendrup et a untibody that could not virus [Arendrup et al. (1998) as [Arendrup et al. (1998)]	20(V3 SF2) gp120(310-319) SIYIGPGRAF L HIV-1 infection nor: Susan Zolla-Pazner (NYU Med. Center) erences: [Arendrup et al.(1993), Arendrup et al.(1995)] TES: SP.SF2:104: Anti-V3 antibody that could neutralize primary virus isolated from a time point of neutralization resistance of autologous virus [Arendrup et al.(1993)] SP.SF2:104: Authors suggest that during in vivo immunoselection of escape virus, the V3 domain gains increasing resemblance to lab strains [Arendrup et al.(1995)]	L ted from a time se virus, the V3 c	HIV-1 infection time point of neutralization e V3 domain gains increasing	$(\mathrm{IgG}_{2a\kappa})$
407 A47/B1	gp120(V3 307-316 IIIB) gp120(311-320) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Akerblom et al.(1990)]	gp120(311-320) (NYU Med. Center) L.(1990)]	IQRGPGRAFV	L	IIIB gp120	$\operatorname{murine}(\operatorname{IgG})$
408 G44/H7	gp120(V3 307-316 IIIB) gp120(311-320) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Akerblom et al.(1990)]	gp120(311-320) (NYU Med. Center) l.(1990)]	IQRGPGRAFV	L	IIIB gp120	murine(IgG)
409 D59/A2	gp120(V3 307-316 IIIB) gp120(311-320) Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Akerblom et al.(1990)]	gp120(311-320) (NYU Med. Center) l.(1990)]	IQRGPGRAFV	L	IIIB gp120	$\operatorname{murine}(\operatorname{Ig} G)$
410 IIIB-34 V3	gp120(V3 308-316 IIIB) gp120(311-319) IQI Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Laman et al.(1992), Laman et al.(1993)] NOTES:	gp120(311-319) (NYU Med. Center) 992), Laman et al.(19	IQRGPGRAF 93)]	L	Peptide	$\operatorname{murine}(\operatorname{Ig} G_1)$
	 IIIB-34 V3: Neutralizes et al.(1992)] IIIB-34 V3: Called III SDS-DTT, enhanced by IIIB-34 V3: UK Medica 	IIIB but not MN – QX B-V3-34 – IIIB strain NP40, but binds to na l Research Council A	IIIB-34 V3: Neutralizes IIIB but not MN – QXGPG are critical amino acids for binding by Pepscan analysis [Laman et al.(1992)] IIIB-34 V3: Called IIIB-V3-34 – IIIB strain specific neutralization – binding is reduced somewhat by DTT or SDS-DTT, enhanced by NP40, but binds to native and denatured gp120 [Laman et al.(1993)] IIIB-34 V3: UK Medical Research Council AIDS reagent: ARP3047	for binding by P nding is reduced aman et al.(1993)	epscan analysis [Laman l somewhat by DTT or	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
411 IIIB-13 V3	gp120(V3 308-316 IIIB) gp120(311-319) IQRGPGRAF Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Laman et al.(1992), Laman et al.(1993), D'Souza et al.(1998) NOTES: • IIIB-13 V3: Also known as 1044-13 and as IIIB-V3-13 (J. P. Moore, • IIIB-13 V3: Neutralizes IIIB but not MN [Laman et al.(1992)] • IIIB-13 V3: Included in a panel of antibodies used in a multi-lab ecomparison, some neutralization of strains other than IIIB [D'Souza et al.(1992)] • IIIB-13 V3: Called IIIB-V3-13 – a neutralization escape mutant (HX in the presence of broadly neutralizing sera – IIIB-V3-13 neutralizati [Watkins et al.(1993)] • IIIB-13 V3: UK Medical Research Council AIDS reagent: ARP3046 • IIIB-13 V3: NIH AIDS Research and Reference Reagent Program: 1	gp120(311-319) (NYU Med. Center) 992), Laman et al.(19) as 1044-13 and as III IIIB but not MN [Lar a panel of antibodia dization of strains oth -V3-13 – a neutralizate ly neutralizing sera – l Research Council A	gp120(V3 308-316 IIIB) gp120(311-319) IQRGPGRAF L Peptide Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Laman et al.(1992), Laman et al.(1993), D'Souza et al.(1994), Watkins et al.(1993)] NOTES: IIIB-13 V3: Also known as 1044-13 and as IIIB-V3-13 (J. P. Moore, per. comm.) IIIB-13 V3: Neutralizes IIIB but not MN [Laman et al.(1992)] IIIB-13 V3: Included in a panel of antibodies used in a multi-lab study for antibody characterization and assay comparison, some neutralization of strains other than IIIB [D'Souza et al.(1994)] IIIB-13 V3: Called IIIB-V3-13 – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – IIIB-V3-13 neutralization was only slightly reduced by this mutation [Watkins et al.(1993)] IIIB-13 V3: NIH AIDS Research Council AIDS reagent: ARP3046 IIIB-13 V3: NIH AIDS Research and Reference Reagent Program: 1727	L I tkins et al.(1993)] nmm.) or antibody chara 994)] 81V) was selecte s only slightly red		$murine(IgG_1)$
412 M77	Donor: Advanced BioScience Laboratories, Rockville, MD, commercial References: [Pal et al.(1992), di Marzo Veronese et al.(1992), di Marzo Cook et al.(1994), Devico et al.(1995), Denisova et al.(1995)] • M77: MAbs against the glycosphingolipid GalCer block HIV infect from the brain and colon – this MAb can inhibit gp120 binding to Galler from the brain and colon – this MAb can inhibit gp120 binding to Galler from the practed with both reduced and non-reduced covalently cross-line and martin and colon – this MAb – a neutralization escape mutant (Honisova et al.(1995)] • M77: Stated to be a murine MAb – a neutralization escape mutant (Honisova et al.(1993)] • M77: Used M77 bound to gp120 as an immunogen – analysis of generated) response suggests the M77-gp120 immunogen generated or gp120 bound to CD4 [Denisova et al.(1996)] • M77: Native M77 is highly strain specific, and V3 binding is primar switched Fab version of M77 could recognize HIV-1 strains that had in GPGR is likely to be critical for binding [Watkins et al.(1996)]	gp120(309-322) E Laboratories, Rock C), di Marzo Veronese al.(1995), Denisova e al.(1995), Denisova e control isolates from tralization and native g glycosphingolipid Ga chis MAb can inhib reduced and non-redu earrangements upon l earrangements upon l ine MAb – a neutralizi y neutralizing sera – N y neutralizing sera – N to gp120 as an im gests the M77-gp120 i [Denisova et al.(1996 hly strain specific, an M77 could recognize ritical for binding [W	pp120(V3 IIIB) gp120(309-322) IRIQRGPGRAFVTI L HIV-1 infection Donor: Advanced BioScience Laboratories, Rockville, MD, commercial References: [Pal et al.(1992), di Marzo Veronese et al.(1992), di Marzo Veronese et al.(1993), Watkins et al.(1993), Cook et al.(1994), Devico et al.(1995), Denisova et al.(1995), Denisova et al.(1995), Watkins et al.(1996)] NOTES: NOTES	L 95), Watkins et z vo Veronese et al. (1993) wed through times binding [di Marziormally suscept ormally suscept ormally suscept erates novel epicerates nov		human(IgG)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
413 268-D	gp120(V3 MN) gp120(312-317) Donor: Susan Zolla-Pazner (NYU Med. Center)	gp120(312-317) r (NYU Med. Center)) HIGPGR	T	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\lambda})$
	References: [Gorny et al.(1991), D'Souza et al. VanCott et al.(1994), Zolla-Pazner et al.(1995) Stamatatos et al.(1997), LaCasse et al.(1998)] NOTES:	1991), D'Souza et al.(1991), Pazner et al.(1995), Casse et al.(1998)]	References: [Gorny et al.(1991), D'Souza et al.(1991), Karwowska et al.(1992b), Gorny et al.(1993), Spear et al.(1993), VanCott et al.(1994), Zolla-Pazner et al.(1995), Fontenot et al.(1995), McKeating et al.(1996), Wisnewski et al.(1996), Stamatatos et al.(1997), LaCasse et al.(1998)] NOTES:)), Gorny et al.(1 .ng et al.(1996),	1993), Spear et al.(1993), Wisnewski et al.(1996),	
	 268-D: Also called 268-11-D-IV and 268D 268-D: Called 268-11-D-IV – strain specifi 268-D: Reacts with MN, NY5, CDC4, R 	3-11-D-IV and 268D D-IV – strain specific IN, NY5, CDC4, RI	268-D: Also called 268-11-D-IV and 268D 268-D: Called 268-11-D-IV – strain specific weakly neutralizing [D'Souza et al.(1991)] 268-D: Rates with MN, NY5, CDC4, RF and SF2, does not cross-react with WM52 or HXB2 [Karwowska	et al.(1991)] ct with WM52	or HXB2 [Karwowska	
	et al.(1992b)] • 268-D: Neutralizes MI • 268-D: Mediated depo	N – binds SF2: YIGPosition of complement	et al.(1992b)] 268-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny et al.(1993)] 268-D: Mediated deposition of complement component C3 on HIV infected cells, but not in the presence of sCD4	75, RF, CDC4 [0 nd cells, but not	Forny et al.(1993)] in the presence of sCD4	
	[Spear et al.(1993)] • 268-D: Moderate disso	ciation rate and homo	[Spear et al.(1993)] 268-D: Moderate dissociation rate and homologous neutralization titer [VanCott et al.(1994)]	nCott et al.(1994	5]	
	 268-D: Serotyping students Pazner et al. (1995) 	ly using flow-cytomet	268-D: Serotyping study using flow-cytometry, if H of HIGPGR was substituted in virus, 268-D did not bind [Zolla-Pazner et al.(1995)]	tuted in virus, 26	58-D did not bind [Zolla-	
	 268-D: Failed to neutraling [McKeating et al.(1996)] 	alize HXB2 and chin	268-D: Failed to neutralize HXB2 and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]	nmary isolates i	in an HXB2 background	
	• 268-D: 268-D is V _H 4	- V-region heavy ch	268-D: 268-D is $V_H4 - V_T$ egion heavy chain usage was examined and a bias of enhanced V_H1 and V_H4 , and reduced V_T3 was noted among HIV infected individuals (Wisnewski et al. (1906))	a bias of enhan	ced $V_H 1$ and $V_H 4$, and	
	 268-D: Poor reactivity against HIV-1 and 257-D [Stamatatos et al (1997)] 	against HIV-1 isolates	268-D: Poor reactivity against HIV-1 isolates SF162 and SF128A and no neutralization, in contrast to MAbs 391/95-D and 257-D [Stamatatos et al. (1997)]	tralization, in co	ntrast to MAbs 391/95-D	
	• 268-D: A T-cell line-ad	dapted (TCLA) deriva	268-D: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by	acquired the ab	ility to be neutralized by	
	 anti-V3 MAbs. The primary isolate could use either CCR5 or C; directed via either pathway, however the TCLA derivative uses C2 268-D: UK Medical Research Council AIDS reagent: ARP3024 268-D: NIH AIDS Research and Reference Reagent Program: 1: 	imary isolate could us way, however the TCI esearch Council AIDS search and Reference	anti-V3 MAbs. The primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse et al.(1998)] 268-D: UK Medical Research Council AIDS reagent: ARP3024 268-D: NIH AIDS Research and Reference Reagent Program: 1511	l was not neutral and is neutraliza	lized when infection was ed [LaCasse et al.(1998)]	
414 polyclonal	gp120(V3 MN)	gp120(313-320)) IGPGRAFY	Т	gp120- <i>B. abortus</i> complex (SF2 or MN)	murine(IgG2a)
	Donor: Susan Zolla-Pazner (NYU Med. Center) References: [Golding et al.(1995)] NOTES:	r (NYU Med. Center) .(1995)]				

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)	
415 0.5β	gp120(V3 316-330 HXB2) gp120(313-326) RGPGRAFVTIGKIC	gp120(313-326)	RGPGRAFVTIGKIG	L (IIIB)	IIIB Env	$\mathrm{murine}(\mathrm{IgG}_{1\kappa})$	
	References: [Matsushita et al. (1988), Skinner et al. (1988b), Skinner et al. (1988a), Reitz Jr. et al. (1988), Nara et al. (1990	.(1988), Skinner et a	1.(1988b), Skinner et al.(1988.	a), Reitz Jr. et al.((1988), Nara et al.(199	90),	
	D'Souza et al.(1991), Matsushita et al.(1992), Emini et al.(1992), Maeda et al.(1992), McKeating et al.(1992a), Sperlagh	hita et al.(1992), Em	uini et al.(1992), Maeda et al.(1992), McKeatin	g et al.(1992a), Sperl	agh	

NOI HO:

• 0.5β : Also called 0.5 beta and 0.5beta

Zvi et al.(1997), Wyatt et al.(1997), Faiman & Horovitz(1997)]

• 0.5β : Type-specific neutralization of IIIB – does not neutralize MN or RF [Matsushita et al.(1988), Skinner et al.(1988b)]

et al.(1995a), Jagodzinski et al.(1996), Warrier et al.(1996), McDougal et al.(1996), Jeffs et al.(1996), Huang et al.(1997) et al.(1994), Thali et al.(1994), Okada et al.(1994), Boudet et al.(1994), Broder et al.(1994), Zvi et al.(1995b), Zvi

et al.(1993), di Marzo Veronese et al.(1993), Moore et al.(1993b), Klasse et al.(1993a), Watkins et al.(1993), Cook

- 0.5 β : Emergence of virus resistant to MAb 0.5 β and autologous sera neutralization in IIIB infected chimps [Nara et al.(1990)
- 0.5β : Potent neutralizing activity [D'Souza et al.(1991)]
- 0.5 β : Chimeric mouse-human MAb C β 1 was constructed by combining the human C γ 1 and C κ constant regions with the 0.5β murine MAb – ADCC and neutralizing activity[Matsushita et al.(1992)]
- 0.5β : sCD4 causes loss of IIIB type-specificity, allowing binding and neutralization of MN, in contrast to MAb μ 5.5 [Maeda et al.(1992)]
- 0.5β : Monoclonal anti-idiotype antibodies that mimic the 0.5β epitope were generated [Sperlagh et al.(1993)]
- 0.5β : Neutralization of virus carrying an A to T substitution (contrast with MAb M77) [di Marzo Veronese et al. (1993)]
- 0.5β : Binding to native gp120 100-300 fold greater than to denatured [Moore et al.(1993b)]
- 0.5β : The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization is not affected [Reitz Jr. et al.(1988), Klasse et al.(1993a)] resistance to some antiserum and conformationally sensitive neutralizing MAbs – neutralization efficiency of 0.5β
- 0.5β : A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – of the MAbs tested , 0.5β neutralization was the most profoundly affected by this mutation [Watkins et al.(1993)]
- 0.5β : MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer in vitro [Cook et al.(1994)]
- 0.5β : gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali et al.(1994)]

MAb ID
D Location
WEAU Sequence
ce Neutralizin
ng Immunogen
Species(Isotype)

415 cont.

NOTES:

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

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
416 924	gp120(V3 309-318 IIIB) gp120(308-316) RKSIRIQRGPG Donor: ? References: [Chesebro & Wehrly(1988), Pincus et al.(1991), Pincus & McClure(1993) et al.(1994), Pincus et al.(1996), Pincus et al.(1998)] NOTES:	gp120(308-316) Wehrly(1988), Pincu 96), Pincus et al.(199	RKSIRIQRGPG s et al.(1991), Pincus & N	1cClure(1993), Pi	vaccinia-gp160 IIIB 3), Pincus et al.(1993), Cook	$\operatorname{murine}(\operatorname{Ig} \operatorname{G}_{1_K})$
	 924: HIV IIIB strain specific [Chesebro & Wehrly(1988)] 924: Epitope sequence is based on database count of a speci specific [Pincus et al.(1991)] 924: MAb was coupled to ricin A chain (RAC) – immunotox although the efficacy of gp41 MAb immunotoxins in vitro inc 924: Ab response in IIIB lab workers was compared to gp10 control – infected lab workers and a vaccinia gp160 vaccine gp160 did not generate anti-V3 response [Pincus et al.(1993)] 924: MAbs against the glycosphingolipid GalCer block HIV from the brain and colon – this MAb can inhibit gp120 bindin 924: A panel of immunotoxins were generated by linking Env1 	is based on database [Chesebro & Wis based on database 91)] to ricin A chain (R.4 gp41 MAb immunote B lab workers was corkers and a vaccinia unti-V3 response [Pinglycosphingolipid Gillowins MAb can inhicotxins were generated.	924: HIV IIIB strain specific [Chesebro & Wehrly(1988)] 924: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIB strain-specific [Pincus et al.(1991)] 924: MAb was coupled to ricin A chain (RAC) – immunotoxin efficacy was not significantly decreased by sCD4, although the efficacy of gp41 MAb immunotoxins in vitro increased 30-fold by sCD4 [Pincus & McClure(1993)] 924: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – MAb 924 was used as a control – infected lab workers and a vaccinia gp160 vaccine had strong V3 MAb response, but alum absorbed rec gp160 did not generate anti-V3 response [Pincus et al.(1993)] 924: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer <i>in vitro</i> [Cook et al.(1994)] 924: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing,	m – 924-RAC imn was not significate old by sCD4 [Pinc cine recipients –] V3 MAb response V3 MAb response r in vitro [Cook et et in A – immunotox	C immunotoxin is IIIB strain- nificantly decreased by sCD4, [Pincus & McClure(1993)] ats – MAb 924 was used as a sponse, but alum absorbed rec usceptible CD4 negative cells ook et al.(1994)] notoxins mediated cell killing,	
417 907	gp120(V3 309-318)	gp120(308-316)	gp120(308-316) RKSIRIQRGPG	L	vaccinia-gp160 IIIB	$\operatorname{murine}(\operatorname{IgG}_{1_K})$
	Donor: ? References: [Chesebro & Wehrly(1988), Pincus et al.(1989), Pincus et al.(1991), Pincus et al.(1996)]	ehrly(1988), Pincus	et al.(1989), Pincus et al.(19	91), Pincus et al.(1	[996)]	
	907: Strain specific bind907: Coupled to ricin A[Pincus et al.(1989)]	ling, and neutralization, chain (RAC), MAb	907: Strain specific binding, and neutralization of only the LAV strain [Chesebro & Wehrly(1988)] 907: Coupled to ricin A chain (RAC), MAb 907 inhibited protein synthesis and cell growth in HIV-infected cells [Pincus et al.(1989)]	hesebro & Wehrly lesis and cell grow	(1988)] rth in HIV-infected cells	-
	 907: Epitope sequence is ba specific [Pincus et al.(1991)] 	is based on database (91)]	907: Epitope sequence is based on database count of a specified location – 924-RA0 specific [Pincus et al.(1991)]	n – 924-RAC imn	C immunotoxin is IIIB strain-	·
	 907: A panel of immunotoxins were generated by linking Env MAbs to r but killing was not directly proportional to binding [Pincus et al.(1996)] 	toxins were generated tlv proportional to bi	907: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al. (1996)]	in A – immunotox	ins mediated cell killing,	-

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
418 Cβ1	gp120(V3 316-330 HXB2)	gp120(313-326)	RGPGRAFVTIGKIG	Т	IIIB Env	human ($\lg G_1$) 0.5β
	Donor: ? References: [Emini et al.(1992)] NOTES: • $C\beta1$: passive transfer to chimpanzees confers p 0.5β human IgG_1 chimera [Emini et al.(1992)]	2)] himpanzees confers ı [Emini et al.(1992	nor: ? ferences: [Emini et al.(1992)] TES: Cβ1: passive transfer to chimpanzees confers protection against challenge with homologous cell-free virus – mouse 0.5β human IgG ₁ chimera [Emini et al.(1992)]	vith homologous	cell-free virus – mouse	·
419 386-D	gp120(V3 MN) gp120(312-317) HIGPGR L Donor: ? References: [Karwowska et al.(1992b), Gorny et al.(1993), VanCott et al.(1994), Fontenot	gp120(312-317) HIGPGR	HIGPGR al.(1993), VanCott et al.(1994)	L), Fontenot et al.	HIV-1 infection et al.(1995)]	$\operatorname{human}(\operatorname{Ig} G_{1\lambda})$
	NOTES: • 386-D: Neutralizes MN – • 386-D: Slow dissociation	binds SF2: YIGPG rate, potent homolo	TES: 386-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny et al.(1993)] 386-D: Slow dissociation rate, potent homologous neutralization [VanCott et al.(1994)]	5, RF, CDC4 [G t al.(1994)]	orny et al.(1993)]	
420 5021	gp120(V3)	gp120(312-318)	QrGPGRa	L	15 mer BH10 V3 peptide	murine(IgG)
	Donor: ? References: [Durda et al.(1988), Durda et al.(1990), Langedijk et al.(1991), Moore et al.(1 NOTES:	8), Durda et al.(199	0), Langedijk et al.(1991), Mo	ore et al.(1993b)])]	
	 5021: Generation and fine mapping of murine MAbs [Langedijl 5021: Binding to native gp120 100-300 fold greater than to dechanges outside the loop have little effect [Moore et al.(1993b)] 	mapping of muring gp120 100-300 fold have little effect [Mo	5021: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)] 5021: Binding to native gp120 100-300 fold greater than to denatured – 314G/W suchanges outside the loop have little effect [Moore et al.(1993b)]	4G/W su	bstitution abolishes binding,	
421 5042	gp120(V3) Donor: ?	gp120(312-318)	QRGPGRA	L	peptide	murine
	References: [Durda et al.(1988), Durda et al.(1990), Moore et al.(1993b)] NOTES:	8), Durda et al.(199	0), Moore et al.(1993b)]			
	• 5042: Binding to native gp120 100-300 fold greater than to d changes outside the loop have little effect [Moore et al.(1993b)]	gp120 100-300 fold nave little effect [Mo	5042: Binding to native gp120 100-300 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]	314G/W substitu	ıtion abolishes binding,	
422 F58/D1	gp120(V3) Donor: ?	gp120(311-318)	IXXGPGRA	L	virus derived gp120	human
	References: [Akerblom et al.(1990), Broliden et al.(1991), Moore et al.(1993b)] NOTES:	1990), Broliden et a	ul.(1991), Moore et al.(1993b)			
	• F58/D1: Binding to native gp120 1-3 fold greater than to outside the loop have little effect [Moore et al.(1993b)]	gp120 1-3 fold grea effect [Moore et al	F58/D1: Binding to native gp120 1-3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]	substitution abo	olishes binding, changes	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
423 P1/D12	gp120(V3)	gp120(311-318)	IXXGPGRA	T	virus derived IIIB gp120	murine(IgG)
	Donor: ? References: [Ake NOTES: • P1/D12: Bind	Donor:? References: [Akerblom et al.(1990), Moore et al.(1993b)] NOTES: P1/D12: Binding to native gp120 1-3 fold greater than to denatured – 314G/W substitution abolishes binding, changes	.(1993b)] ater than to denatured	∃−314G/W substitution abo	olishes binding, changes	~
424 P4/D10	gp120(V3)	gp120(311-318) IXXGPGRA	IXXGPGRA	Т	virus derived IIIB gp120	$\text{murine}(\text{IgG}_{1\kappa})$
	References: [Ake: Arendrup et al.(19 NOTES: P4/D10: Neut P4/D10: Bind outside the loo P4/D10: Prim P4/D10 [Aren P4/D10: Used decrease in an P4/D10: Revi	 Donor: ? References: [Akerblom et al.(1990), Broliden et al.(1990), Broliden et al.(1991), Marks et al.(1992), Moore et al.(1993b), Arendrup et al.(1993), Hinkula et al.(1994), Jacobson(1998)] NOTES: P4/D10: Neutralizing and ADCC activity [Broliden et al.(1990)] P4/D10: Variable domain sequenced and is identical to F58/H3 [Marks et al.(1992)] P4/D10: Binding to native gp120 3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)] P4/D10: Primary isolates from different time points from one individual were not susceptible to neutralization by P4/D10 [Arendrup et al.(1993)] P4/D10: Used for passive immunotherapy in four late-stage HIV-infected patients – the serum level of p24 did not decrease in any of these four – see also MAb F58/H3 [Hinkula et al.(1994)] P4/D10: Review of passive immunotherapy, summarizing [Hinkula et al.(1994)] in relation to other studies [Jacobson(1998)] 	I.(1990), Broliden et son(1998)] roliden et al.(1990)] lentical to F58/H3 [Jentical to F58/H3 [Jer than to denatured I.(1993b)] e points from one in four late-stage HIV F58/H3 [Hinkula et summarizing [Hinku	tal.(1991), Marks et al.(199 Marks et al.(1992)] – 314G/W substitution abordividual were not susception of the seron al.(1994)] ula et al.(1994)] in relation	al.(1992), Moore et al.(1993b), ion abolishes binding, changes usceptible to neutralization by the serum level of p24 did not elation to other studies [Jacob-	
425 419-D	gp120(V3) Donor: ?	gp120(311-317) IHIGPGR	IHIGPGR	L	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\lambda})$
	References: [Karv NOTES: • 419-D: MN,] et al.(1992b)]	 References: [Karwowska et al.(1992b), Gorny et al.(1993), Spear et al.(1993), Fontenot et al.(1995)] NOTES: 419-D: MN, NY5 and SF2 strain specific, does not cross-react with RF, CDC4, WM52 or HXB2 [Karwowska et al.(1992b)] 	al.(1993), Spear et a does not cross-react	al.(1993), Fontenot et al.(1) t with RF, CDC4, WM52	995)] or HXB2 [Karwowska	2
	419-D: Neutra419-D: Mediarabbit anti-hu	419-D: Neutralizes MN – binds SF2: IYIGPGR [Gorny et al.(1993)] 419-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear et al.(1993)]	GR [Gorny et al.(199 component C3 on H	93)] IV infected cells, enhancec	1 by second Ab binding	•

				q	O	
426 537-D	gp120(V3) Donor: ?	gp120(313-317) IGPGR	IGPGR	Т	HIV-1 infection	$human(IgG_{1\lambda})$
	References: [Karwowska et al. (1992b), Gorny et al. (1992), Gorny et al. (1993), VanCott et al. (1994), Fontenot et al. (1995)] NOTES:	tal.(1992b), Gorny et	al.(1992), Gorny et al.(1993	3), VanCott et al.(199	4), Fontenot et al. (1995)	Ĺ
	• 537-D: Reacts with N et al.(1992b)]	ſN, NY5, CDC4, RF	537-D: Reacts with MN, NY5, CDC4, RF, WM52 and SF2, but does not cross-react al.(1992b)]	es not cross-react v	act with HXB2 [Karwowska	а
	et al.(1992b)] • 537-D: MN type specii • 537-D: Moderate homo	fic neutralization obse ologous neutralization	et al.(1992b)] 537-D: MN type specific neutralization observed – binds SF2, also IGPGR [Gorny et al.(1992), Gorny et al.(1993)] 537-D: Moderate homologous neutralization, relatively rapid dissociation constant [VanCott et al.(1994)]	PGR [Gorny et al.(19) on constant [VanCo	992), Gorny et al.(1993) tt et al.(1994)]	
427 NM-01	gp120(V3 MN) Donor: M. Terada	gp120(314-317) GPGR	GPGR	L	IIIB MN	murine(IgG)
	References: [Ohno et al.(1991), Yoshida et al.(1997), Smith et al.(1998)] NOTES:	991), Yoshida et al.(1	997), Smith et al.(1998)]			
	 NM-01: Resistance mu et al.(1997)] 	ntation selected by pro	NM-01: Resistance mutation selected by propagation of molecular cloned isolate in the et al.(1997)]	ed isolate in the pres	presence of NM-01 [Yoshida	a
	 NM-01: The tip of the 	MN V3 loop was ins	NM-01: The tip of the MN V3 loop was inserted into cold causing human rhinovirus		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	s o

MAb ID	MAb ID Location	WEAU	Sequence	Neutralizing Immunogen	Species(Isotype)
428 447-52D	gp120(V3 MN) Donor: Dr. Susan Zolla-Paz References: [Gorny et al.(19 Cavacini et al.(1993a), Spear Moore et al.(1994a), Satten Pazner & Sharpe(1995), Mo et al.(1996a), Sattentau(199 et al.(1997), Parren et al.(19	gp120(314-317) GPXR rner, NYU Med Center NY, 192), Buchbinder et al. (1992) et al. (1993), Conley et al. (1941), Fontenot et al. (1995), Fontenot et al. (1995), Fontenot et al. (1997), Bouza et al. (1997), Bouza et al. (1997), Inou	gp120(V3 MN) gp120(314-317) GPXR L HIV-1 inft Donor: Dr. Susan Zolla-Pazner, NYU Med Center NY, NY, or Cellular Products Inc, Buffalo, NY, USA References: [Gorny et al.(1992), Buchbinder et al.(1992), Karwowska et al.(1992b), Gorny et al.(1993), Ke Cavacini et al.(1993a), Spear et al.(1993), Conley et al.(1994a), Laal et al.(1994), VanCott et al.(1994), Gor Moore et al.(1994a), Sattentau(1995), Fontenot et al.(1995), Saarloos et al.(1995), Zolla-Pazner et al. Pazner & Sharpe(1995), Moore et al.(1995a), Moore & Ho(1995), Forthal et al.(1995), Jagodzinski et al. et al.(1996a), Sattentau(1996), D'Souza et al.(1997), Binley et al.(1997), Fouts et al.(1997), Hioe et al.(1997), Parren et al.(1997b), Hill et al.(1997), Inouye et al.(1998), Mondor et al.(1998), Smith et al.	gp120(V3 MN) gp120(314-317) GPXR L HIV-1 infection hur Donor: Dr. Susan Zolla-Pazner, NYU Med Center NY, NY, or Cellular Products Inc, Buffalo, NY, USA References: [Gorny et al.(1992), Buchbinder et al.(1992), Karwowska et al.(1992b), Gorny et al.(1993), Keller et al.(1993) Cavacini et al.(1993a), Spear et al.(1993), Conley et al.(1994a), Laal et al.(1994), VanCott et al.(1994), Gorny et al.(1994) Moore et al.(1994a), Sattentau(1995), Fontenot et al.(1995), Saarloos et al.(1995), Zolla-Pazner et al.(1995), Moore et al.(1995a), Moore & Ho(1995), Forthal et al.(1995), Jagodzinski et al.(1996), Trkola et al.(1996a), Sattentau(1996), D'Souza et al.(1997), Binley et al.(1997), Fouts et al.(1997), Hioe et al.(1997), Boots et al.(1997b), Hill et al.(1997b), Hill et al.(1997), Inouye et al.(1998), Mondor et al.(1998), Smith et al.(1998), Parren	human(IgG _{3λ}) 1993), 1994), Zolla- Trkola Boots Parren

et al.(1998), Connor et al.(1998), Gorny et al.(1998)]

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

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

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- 447-52D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding - 447-52D bound monomer, oligomer, and neutralized JRFL [Fouts et al.(1997)]
- 447-52D: Tested using a resting cell neutralization assay [Hioe et al.(1997)]
- 447-52D: Viral binding inhibition by 447-D was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 447-52D: Neutralizes TCLA strains but not primary isolates [Parren et al.(1997b)]
- 447-52D: Called 447 gp120 can inhibit MIP-1 α from binding to CCR5, but this inhibitory effect is blocked by effect [Hill et al.(1997)] pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no
- 447-52D: Called 447-D 447-D resistance took longer to acquire in virus with the M184V substituted RT, and had wildtype RT [Inouye et al.(1998)] the form (AAC N to TAC Y) at position 5 of the V3 loop, rather than the GPGR to GPGR resistance found with
- 447-52D: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library protocols [Boots et al.(1997)] the sequence QRGPGR, showing type specific mimotyopes can be enriched by strain specific ligand competition et al.(1993)] - in Keller et al., with no competition, LxGPxR was the most common six-mer, 38% of the peptides - after competition with a gp120 IIIB ligand (QRGPGR)i, RGPxR was the most common and one peptide had 447-52D has an epitope involving the tip of the V3 loop, that was previously studied with this method [Keller
- 447-52D: Inhibits binding of Hx 10 to both CD4 positive and negative HeLa cells [Mondor et al.(1998)]
- 447-52D: Called 447-52-D The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) was among the Abs used - chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 447-52D
- 447-52D: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated epitope [Parren et al.(1998)] authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the
- 447-52D: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could 2G12, IgG1b12, 2F5 and 447-52D [Connor et al.(1998)] were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses
- 447-52D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 1324E was comparable to 447-52D [Gorny et al.(1998)]

MAU	Location	WEAU	Sequence	Neutranzing	ımmunogen	Species(isotype)
429 59.1	gp120(308-313 MN)	gp120(314-319) GPGRAF	GPGRAF	Т	cyclic V3 MN peptide	$murine(IgG_1)$
	Donor: Mary White-Scharf and A. Profy, Repligen Corporation References: [D'Souza et al.(1991), White-Scharf et al.(1993), Potts et al.(1993), Ghiara et al.(1993), Bou-Habib et al.(1994), D'Souza et al.(1994), Seligman et al.(1996), Ghiara et al.(1997), Smith et al.(1998)] NOTES:	and A. Profy, Replig ul.(1991), White-Sch 994), Seligman et al	en Corporation arf et al.(1993), Potts et al.(1996), Ghiara et al.(1997)	al.(1993), Ghiara e , Smith et al.(1998)	t al.(1993), Bou-Habib	-
	 59.1: Called R/V3-59.1 59.1: Epitope defined b Scharf et al.(1993)] 	 potent neutralizing peptide reactivity a 	59.1: Called R/V3-59.1 – potent neutralizing MAb [D'Souza et al.(1991)] 59.1: Epitope defined by peptide reactivity and binding affinity with amino acid substitutions – GPGRAF [White-Scharf et al.(1993)])] nino acid substitutio	ons – GPGRAF [White-	
	 59.1: Synergistic neutra 59.1: Crystal structure of 	lization of MN when of a 24 amino acid pe	59.1: Synergistic neutralization of MN when combined with sCD4 or the CD4BS MAb F105 [Potts et al.(1993)] 59.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 Fab fragment – contact residues	e CD4BS MAb F10 and to 59.1 Fab frag	nent – contact residues	
	IGPGRAF [Ghiara et al.(1993)]59.1: Greater affinity for T-cell t	.(1993)] r T-cell tropic strain 7	IGPGRAF [Ghiara et al.(1993)] 59.1: Greater affinity for T-cell tropic strain T-CSF than the primary isolate JR-CSF, from which T-CSF was derived	ate JR-CSF, from w	hich T-CSF was derived	
	[Bou-Habib et al.(1994)]59.1: Multi-lab study fo] or antibody characte	[Bou-Habib et al.(1994)] 59.1: Multi-lab study for antibody characterization and assay comparison – neutralizes MN and IIIB [D'Souza	ison – neutralizes]	MN and IIIB [D'Souza	
	et al.(1994)] • 59.1: Competition ELIS	As with serial deletion	et al.(1994)] 59.1: Competition ELISAs with serial deletions produced longer estimate of epitope length than x-ray crystallography	of epitope length th	an x-ray crystallography	•
	 59.1: A conformational crystal stucture shows in 	ly restricted analog	59.1: A conformationally restricted analog of the tip of the V3 loop was constructed and bound with Fab 59.1 – crystal stricture shows interactions between 59.1 and an MN pentide and 59.1 and the modified pentide are similar.	as constructed and	and bound with Fab 59.1 –	
	but NMR studies reveal	that the modified per	but NMR studies reveal that the modified peptide is more ordered in solution, retaining	ıtion, retaining the l	the Fab bound form [Ghiara	Γ `
	et al.(1997)] • 59.1: The tip of the MY	N V3 loop was inser	et al.(1997)] 59.1: The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were	n rhinovirus 14 (H	RV14) – chimeras were	-
	immunoselected, and chused – chimeric viruses	imeric viruses were elicited potent NAbs	immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 59.1 was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith et al.(1998)]	p antibodies, and 59	nd 59.1 was among the Abs mith et al.(1998)]	-

MAb ID Location	430 50.1 gp120(V3 MN) Donor: Mary W References: [D' Bou-Habib et al. Fontenot et al.(1' NOTES:	NOTES:	• 50.1: No	• 50.1: Cry KRIHIGI	● 50 1· Crv	to the left	to the left • 50.1: No T-CSF, de	to the left • 50.1: No T-CSF, de • 50.1: Pot	to the left 50.1: No T-CSF, de 50.1: Pot 50.1: Ch ization [F	to the left 50.1: No T-CSF, de 50.1: Pot 50.1: Chi ization [F 50.1: She 50.1: Use	to the left 50.1: No T-CSF, de 50.1: Pot 50.1: Chi ization [F 50.1: She 50.1: Con and alani	to the left 50.1: No T-CSF, de 50.1: Pot 50.1: Chi ization [F 50.1: She 50.1: Cou and alani 50.1: Bir 50.1: A '
WEAU	gp120(V3 MN) gp120(310-314) RIHIG L V3 MN peptide Donor: Mary White-Scharf, Repligen Corporation, Cambridge, MA References: [D'Souza et al.(1991), White-Scharf et al.(1993), Potts et al.(1993), Ghiara et al.(1993), Rini et al.(1993), Bou-Habib et al.(1994), VanCott et al.(1994), Robert-Guroff et al.(1994), Moore et al.(1994b), VanCott et al.(1995), Fontenot et al.(1995), Seligman et al.(1996), Berman et al.(1997), LaCasse et al.(1998)] NOTES:	TES: 50.1: Called R/V3-50.1 – potent neutralizing MAb [D'Souza et al.(1991)] 50.1: Epitope defined by peptide reactivity and changes affinity with amino acid substitutions – epitope RIHIGP [White-Scharf et al.(1993)]	50.1: No synergistic neutralization of MN when combined with CD4BS MAb F105 [Potts et al.(1993)]	50.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 and KRIHIGP [Ghiara et al.(1993)]	50.1: Crystal structure of V3 loop bound to $50.1 - light chain binds just to the left of GPG, heavy chain binds further to the left [Rini et al.(1993)]$, [1 cm or cm:(1775)]	50.1: No neutralization of primary isolate JR-CSF – greater affinity for and neutralization of T cell tropic strain T-CSF, derived from JR-CSF [Bou-Habib et al.(1994)]	50.1: No neutralization of primary isolate JR-CSF – greater affinity for an T-CSF, derived from JR-CSF [Bou-Habib et al.(1994)] 50.1: Potent MN neutralization, slow dissociation rate [VanCott et al.(1994)] 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FAC.	50.1: No neutralization of primary isolate JR-CSF – greater affinity for and neutral T-CSF, derived from JR-CSF [Bou-Habib et al.(1994)] 50.1: Potent MN neutralization, slow dissociation rate [VanCott et al.(1994)] 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FACS signal, ization [Robert-Guroff et al.(1994)]	50.1: No neutralization of primary isolate JR-CSF – greater affinity for and neutralization of T cell t T-CSF, derived from JR-CSF [Bou-Habib et al.(1994)] 50.1: Potent MN neutralization, slow dissociation rate [VanCott et al.(1994)] 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FACS signal, Ab affinity, and vization [Robert-Guroff et al.(1994)] 50.1: Shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore et al.(1994b)] 50.1: Used to monitor HIV-1 Env expression in infected H9 cells [VanCott et al.(1995)]	50.1: No neutralization of primary isolate JR-CSF – greater affinity for and neutralization of T cell tropic strain T-CSF, derived from JR-CSF [Bou-Habib et al.(1994)] 50.1: Potent MN neutralization, slow dissociation rate [VanCott et al.(1994)] 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FACS signal, Ab affinity, and viral neutralization [Robert-Guroff et al.(1994)] 50.1: Shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore et al.(1994b)] 50.1: Used to monitor HIV-1 Env expression in infected H9 cells [VanCott et al.(1995)] 50.1: Competition ELISAs with serial deletions produced comparable estimate of epitope length to crystal structure and alanine substitution – KRIHIGP [Seligman et al.(1996)]	50.1: No neutralization of primary isolate JR-CSF – greater affinity for and neutralization of T cell tropic strain T-CSF, derived from JR-CSF [Bou-Habib et al.(1994)] 50.1: Potent MN neutralization, slow dissociation rate [VanCott et al.(1994)] 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FACS signal, Ab affinity, and viral neutralization [Robert-Guroff et al.(1994)] 50.1: Shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore et al.(1994b)] 50.1: Used to monitor HIV-1 Env expression in infected H9 cells [VanCott et al.(1995)] 50.1: Competition ELISAs with serial deletions produced comparable estimate of epitope length to crystal structure and alanine substitution – KRIHIGP [Seligman et al.(1996)] 50.1: Binds to 6/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)] 50.1: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by
Sequence) RIHIG ion, Cambridge, MA arf et al.(1993), Potts et al.(1993) Robert-Guroff et al.(1994), Mo rman et al.(1997), LaCasse et al	g MAb [D'Souza et al.(1991)]	when combined with CD4BS I	eptide from the V3 loop bound 1	50.1 – light chain binds just to tl	IR-CSF – oreater affinity for a	t al.(1994)]	t al.(1994)] t al.(1994)] ciation rate [VanCott et al.(1994)]	t al.(1994)] ciation rate [VanCott et al.(1994) ackground allows increased FA	t al.(1994)] ciation rate [VanCott et al.(1994) ackground allows increased FA B clade gp120s, little outside F in in infected H9 cells [VanCott	t al.(1994)] ciation rate [VanCott et al.(1994) ackground allows increased FA ackground allows increased FA B clade gp120s, little outside Fa in in infected H9 cells [VanCott ions produced comparable estin man et al.(1996)]	t al.(1994)] ciation rate [VanCott et al.(1994)] vackground allows increased FA vackground allows increased FA B clade gp120s, little outside FA in in infected H9 cells [VanCott ions produced comparable estin man et al.(1996)] gh cases from a MN gp120 vacc tive of SI primary isolate 168P
Neutralizing Ir	L V), Ghiara et al.(199 ore et al.(1994b), \(\text{V}\).(1998)]	no acid substitutior		o 59.1 and 50.1 Fab	ne left of GPG, heav		nd neutralization o	nd neutralization o)] CS signal Ab affini	nd neutralization o)] CS signal, Ab affini	nd neutralization o [] CS signal, Ab affini clade [Moore et al et al.(1995)]	nd neutralization o)] CS signal, Ab affini c clade [Moore et al et al.(1995)] nate of epitope lengt	nd neutralization o [S] [CS] [S] [CS] [S] [CS] [CS] [Ab affinity [CS] [CS]
Immunogen	V3 MN peptide et al.(1993), Rini et al.(1993), 1994b), VanCott et al.(1995),	ıs – epitope RIHIGP	$-$ isotype stated to be IgG_{2a}	50.1 Fab fragments – epitope	y chain binds further	f T cell tropic strain		in amount, and that nearth	.(1994b)]		th to crystal structure	:h to crystal structure t al.(1997)] to be neutralized by
Species(Isotype)		·	ν-		7	_				•		

MAb ID Location	431 58.2 gp120(V3 MN) Donor: Mary V References: [W NOTES:	• 58.2: isolat • 58.2: when • 58.2: et al.1 • 58.2: Alani	432 Nea 9301 gp120(V3 IIIB) Donor: Dupont References: [W NOTES: • 694/98-D:	433 694/98-D gp120(V3 IIIB) Donor: Drs. S. References: [G et al.(1994), Van Li et al.(1997), Van NOTES:
WEAU Sequence	gp120(V3 MN) gp120(312-319) HIGPGRAF Donor: Mary White-Scharf, Repligen Corporation, Cambridge, MA References: [White-Scharf et al.(1993), Potts et al.(1993), Moore et al.(1994b), Seligman et al.(1996)] NOTES: Self- Enitone defined by pentide reactivity and changes in affinity with amino acid substitutions.	58.2: Epitope defined by peptide reactivity and changes in affinity with amino acid substitutions – 4/7 primarily isolates were neutralized [White-Scharf et al.(1993)] 58.2: Did not synergistically neutralize MN in combination with MAb F105 – there was synergistic neutralization when combined with sCD4 [Potts et al.(1993)] 58.2: Modest cross-reactivity among B clade gp120s, little outside B clade – core epitope as I-IHIG [Moore et al.(1994b)] 58.2: Competition ELISAs with serial deletions produced longer estimates of epitope length, RIHIGPGRAFY, than Alanine substitution, suggesting significance of non-contact residues [Seligman et al.(1996)]	gp120(V3 IIIB) gp120(312-327) RIQRGPGRAFVTIGKI Donor: Dupont, commercial References: [Wagner et al.(1996)] NOTES: • 694/98-D:	gp120(V3 IIIB) gp120(316-319) GRAF Donor: Drs. S. Zolla-Pazner and M. Gorny, NYU Med Center NY, NY References: [Gorny et al.(1992), Gorny et al.(1993), Cavacini et al.(1993a), Spear et al.(1993), Gorny et al.(1994), Laal et al.(1994), VanCott et al.(1994), VanCott et al.(1995), Zolla-Pazner et al.(1995), Forthal et al.(1995), Li et al.(1997), Zolla-Pazner et al.(1997), Smith et al.(1998), Li et al.(1998), Andrus et al.(1998)] NOTES:
Neutralizing Immunogen	L MN V3 peptide , MA ore et al.(1994b), Seligman et al.(1996)]	in affinity with amino acid substitutions – 4/7 primarily on with MAb F105 – there was synergistic neutralization ittle outside B clade – core epitope as I-IHIG [Moore longer estimates of epitope length, RIHIGPGRAFY, than it residues [Seligman et al.(1996)]	\FVTIGKI	L HIV-1 infection NY, NY et al.(1993), Gomy et al.(1994), Spear et al.(1993), Gomy et al.(1994), Zolla-Pazner et al.(1995), Forthal et al.(1998), Andrus et al.(1998)]
Species(Isotype)	$\operatorname{murine}(\operatorname{IgG}_1)$	rimarily lization [Moore ⁷ Y, than	murine	human(IgG _{1λ}) 4), Laal .(1995),

• 694/98-D: Called 694-D - complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear et al.(1993)]

694/98-D: Type-specific lab isolate neutralization was observed – binds with 1-3 fold greater affinity to gp120 than to peptides [Gorny et al.(1992)]
694/98-D: Neutralizes MN and IIIB (GRAF) – binds SF2 (GRAF) – binding reactivity: MN, IIIB, SF2, NY5, RF, CDC4, WM52 [Gorny et al.(1993)]

433 cont.

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

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
434 9205	gp120(V3 IIIB) gp120(Donor: NEN, Boston MA, commercial References: [Durda et al.(1990), Trujill NOTES:	gp120(317-319) commercial 1990), Trujillo et al.(19	gp120(V3 IIIB) gp120(317-319) RAF (core reactivity) L IIIB V3 Peptide Donor: NEN, Boston MA, commercial References: [Durda et al.(1990), Trujillo et al.(1993), Allaway et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)] NOTES:	L anCott et al.(1994	IIIB V3 Peptide), Fontenot et al.(1995)]	$\operatorname{murine}(\operatorname{Ig} \operatorname{G}_1)$
	 9205: Called NEA-9205, epitope RIQRGPGRAFVTIGK – r kDa molecular weight – similar to 9284 [Trujillo et al.(1993)] 9205: Synergy with combinations of CD4-based molecules in et al.(1993)] 	05, epitope RIQRGPC – similar to 9284 [Tru mbinations of CD4-ba	9205: Called NEA-9205, epitope RIQRGPGRAFVTIGK – reacts with three human brain proteins of 35, 55, 110 kDa molecular weight – similar to 9284 [Trujillo et al.(1993)] 9205: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)]	hree human brain HIV-1 Env media	the brain proteins of 35, 55, 110 mediated cell fusion [Allaway	. •
	• 9205: Neutralizes IIIB	but not MN – significa	9205: Neutralizes IIIB but not MN - significantly slower dissociation constant for IIIB		than MN [VanCott et al.(1994)]	_
435 N11-20	gp120(V3 317-325) Donor: J. C. Mazie, Hybridolab, Institut Pasteur References: [Valenzuela et al.(1998)] NOTES: NOTES: N11-20: Also called 110-H N11-20: Neutralization of LAI in CEM cells binding to the cell [Valenzuela et al.(1998)]	gp120(314-322) dolab, Institut Pasteur t al.(1998)] 10-H n of LAI in CEM cel enzuela et al.(1998)]	20(V3 317-325) gp120(314-322) GPGRAFVTI L (LA nor: J. C. Mazie, Hybridolab, Institut Pasteur erences: [Valenzuela et al.(1998)] TES: N11-20: Also called 110-H N11-20: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11-20 binding to the cell [Valenzuela et al.(1998)]		ιΙ) unk is through inhibition of virus	$\operatorname{murine}(\operatorname{Ig} \operatorname{G}_{1\kappa})$
436 902	gp120(V3 IIIB) gp120(315-326) PGRAFVTIGKIG Donor: Bruce Chesebro, Rocky Mountain National Laboratory, Montana References: [Chesebro & Wehrly(1988), Laman et al.(1993), Broder et al.	gp120(315-326) ocky Mountain Nation Wehrly(1988), Laman	gp120(V3 IIIB) gp120(315-326) PGRAFVTIGKIG L vaccinia-gp160 IIIB Donor: Bruce Chesebro, Rocky Mountain National Laboratory, Montana References: [Chesebro & Wehrly(1988), Laman et al.(1993), Broder et al.(1994), Earl et al.(1994), Sakaida et al.(1997)]	L 4), Earl et al.(199	vaccinia-gp160 IIIB 4), Sakaida et al.(1997)]	$\operatorname{murine}(\operatorname{IgG}_{1\kappa})$
	 902: Strain specific neutralizati 902: Epitope may be partially 1 902: Used as a control in a student of the strain of the strain	utralization of HIV [C artially masked or alte in a study of the influ .(1994)]	902: Strain specific neutralization of HIV [Chesebro & Wehrly(1988)] 902: Epitope may be partially masked or altered in the oligomeric molecule [Broder et al.(1994)] 902: Used as a control in a study of the influence of oligomeric structure of Env in determining the repertoire of the Ab response [Earl et al.(1994)]	le [Broder et al.(1994)] of Env in determining th	994)] ing the repertoire of the	ÿ
	 902: V3-BH10 peptide with loop-structure inhibits IL-2 induce intracellular signaling, and MAb 908 can block the peptide inhib 902: NIH AIDS Research and Reference Reagent Program: 522 	e with loop-structure i and MAb 908 can blo rch and Reference Rea	902: V3-BH10 peptide with loop-structure inhibits IL-2 induced T-cell proliferation, thoug intracellular signaling, and MAb 908 can block the peptide inhibition [Sakaida et al.(1997)] 902: NIH AIDS Research and Reference Reagent Program: 522	roliferation, thoug caida et al.(1997)]	, thought to be due to altering 1997)]	V-1

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
437 IIIB-V3-01	gp120(V3 IIIB)	gp120(322-330)	IGKIGNMRQ	Z	IIIB carboxy- terminus V3-loop peptide	$\operatorname{murine}(\operatorname{\lg} \operatorname{G}_1)$
	References: [Laman et al.(1993)] NOTES: • IIIB-V3-01: Specific for carboxy-terminal flank of the IIIB V3 loop denaturation [Laman et al.(1993)] • IIIB-V3-01: UK Medical Research Council AIDS reagent: ARP3046 • IIIB-V3-01: NIH AIDS Research and Reference Reagent Program: 1	993)] r carboxy-terminal fla al.(1993)] al Research Council A Research and Referer	erences: [Laman et al.(1993)] TES: IIIB-V3-01: Specific for carboxy-terminal flank of the IIIB V3 loop – epitope is hidden native gp120, exposed on denaturation [Laman et al.(1993)] IIIB-V3-01: UK Medical Research Council AIDS reagent: ARP3046 IIIB-V3-01: NIH AIDS Research and Reference Reagent Program: 1726	ppe is hidden na	ıtive gp120, exposed oı	1
438 9305	gp120(V3) gp120 Donor: Du Pont, Wilmington DE References: [McDougal et al.(1996)]	gp120 տ DE մl.(1996)]		L		murine
439 D/6D1	gp120(V4 351-382 LAI)	gp120(350-431)	ASKLREQFGNNKTIIFK- QSSGGDPEIVTHSFN	Z	Baculovirus- expressed rgp120	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: Du Pont, Wilmington DE References: [Bristow et al.(1994)] NOTES: • D/6D1: V4 MAb generated in	n DE 1994)] ıted in a study of the h	or: Du Pont, Wilmington DE erences: [Bristow et al.(1994)] TES: D/6D1: V4 MAb generated in a study of the humoral immune response to rgp120 and		LAI rgp160 [Bristow et al.(1994)]	.
440 4D7/4	gp120(V4 361-380 LAI)	gp120(364-384)	IFKQSSGGDPEIVTHSF- NCGG		Env glycopro	murine(IgG)
	 Donor: S. Ranjbar, NIBSC, UK References: [Moore et al.(1994c)] NOTES: 4D7/4: C3 region – the relative affinity for denatured/native gp1: 4D7/4: UK Medical Research Council AIDS reagent: ARP3051 	UK 994c)] relative affinity for de: search Council AIDS	 nor: S. Ranjbar, NIBSC, UK lerences: [Moore et al.(1994c)] YIES: 4D7/4: C3 region – the relative affinity for denatured/native gp120 is >10 [Moore et al.(1994c)] 4D7/4: UK Medical Research Council AIDS reagent: ARP3051 	Moore et al.(199	94c)]	
441 36.1(ARP 329)	gp120(V4 362-381 LAI)	gp120(365-385)	FKQSSGGDPEIVTHSFN-CGGE		Env glycopro	murine(IgG)
	 Donor: S. Ranjbar, NIBSC, UK References: [Thiriart et al.(1989), Moore et al.(1994c)] NOTES: 36.1: The relative affinity for denatured/native gp1 	UK 1989), Moore et al.(19 ty for denatured/nativ	 sor: S. Ranjbar, NIBSC, UK erences: [Thiriart et al.(1989), Moore et al.(1994c)] TES: 36.1: The relative affinity for denatured/native gp120 is >30 - mutations 380 G/F, 381 E/P impair binding [Moore 	80 G/F, 381 E/P	impair binding [Moor	(v
	et al.(1994c)] • 36.1: UK Medical Research Council AIDS reagent: ARP329	arch Council AIDS re	agent: ARP329			

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
442 C12	gp120(V4 362-381 LAI)	gp120(365-385)	FKQSSGGDPEIVTHSFN-CGGE		mis-folded LAI rgp160	$murine(IgG_1)$
	Donor: George Lewis References: [Moore & Ho(199 NOTES:	93), Moore et al.(19	Donor: George Lewis References: [Moore & Ho(1993), Moore et al.(1994c), Abacioglu et al.(1994), Moore et al NOTES:	[oore et al.(1994d)]	4d)]	
	 C12: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] C12: The relative affinity for denatured/native gp120 is >30 – mutatio binding – also binds GEFFYCNSTQLFNS, gp120(380-393 LAI) [Moore C12: C3 region – epitope boundaries mapped by peptide scanning, core leading to the content of the content	to denatured IIIB g for denatured/nativ TYCNSTQLFNS, g boundaries mapped	C12: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] C12: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P, and 384 Y/E impair binding – also binds GEFFYCNSTQLFNS, gp120(380-393 LAI) [Moore et al.(1994c)] C12: C3 region – epitope boundaries mapped by peptide scanning, core FNCGG [Abacioglu et al.(1994)]	80 G/F, 381 E. l.(1994c)] GG [Abaciogh	/P, and 384 Y/E impair u et al.(1994)]	
443 110.D	gp120(C3 380-393 LAI) gp120(384-397) GEFFY Donor: F. Traincard, Pasteur Institute, France References: [Moore et al.(1994c), Valenzuela et al.(1998)] NOTES:	gp 120(384-397) nstitute, France 4c), Valenzuela et a	GEFFYCNSTQLFNS L.(1998)]	Z	Env glycopro	murine(IgG)
444 B32	on120(380-393 I AI)	om120(384-397)	0/380_303 I AI)	ì	mis-folded I AI	murine(IoG.)
					rgp160	0 - 1
	Donor: F. Traincard, Pasteur Institute, France References: [Moore et al.(1994c), Abacioglu et al.(1994)] NOTES:	nstitute, France 4c), Abacioglu et al	.(1994)]			
	• B32: The relative affinity for denatured/native 386 N/R impair binding [Moore et al.(1994c)]	for denatured/native Moore et al.(1994c)]	B32: The relative affinity for denatured/native gp120 is >100 – mutations 380 G/F, 38 386 N/R impair binding [Moore et al.(1994c)]	· —	G/P, 382 F/L, 384 Y/E, and	
	• B3Z: C3 region – epitope	Doutidaties iliapped	52. C3 region – ephope boundaries mapped by peptide scanning – rr i (core) [Abaciogin et al.(1994)]	e) [Abactogiu	et al.(1994)]	
445 B2C	gp120(C3 HIV2ROD) gp120 Donor: F. Traincard, Pasteur Institute, France References: [Matsushita et al.(1995)] NOTES:	gp120 nstitute, France (1995)]	HYQ(core)	L	Peptide	murine
	B2C: Viral neutralization v	was type-specific fo	B2C: Viral neutralization was type-specific for HIV-2 ROD [Matsushita et al.(1995)]	(1995)]		
446 2H1B	gp120(C3 370-376 gp120(361-36 HIV2ROD) Donor: F. Traincard, Pasteur Institute, France References: [Matsushita et al.(1995)] NOTES: • 2H1B: Binds in WB, but binds poorly to 1	gp120(361-367) nstitute, France (1995)] inds poorly to Env	20(C3 370-376 gp120(361-367) RNISFKA N '2ROD) nor: F. Traincard, Pasteur Institute, France erences: [Matsushita et al.(1995)] TES: 2H1B: Binds in WB, but binds poorly to Env on the cell surface [Matsushita et al.(1995)]	N et al.(1995)]	Peptide	murine

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
447 2F19C	gp120(C3 HIV2ROD) gp120 Donor: F. Traincard, Pasteur Institute, France References: [Matsushita et al.(1995)] NOTES: • 2F19C: Binds in WB, but binds poorly to	gp120 Institute, France (1995)] t binds poorly to Env	onor: F. Traincard, Pasteur Institute, France eferences: [Matsushita et al.(1995)] OTES: 2F19C: Binds in WB, but binds poorly to Env on the cell surface [Matsushita et al.(1995)]	N et al.(1995)]	Peptide	murine
448 B15	gp120(V4 395-400 BH10)	gp120(394-399)	WFNSTW		mis-folded LAI rgp160	$\mathrm{murine}(\mathrm{IgG}_{2b})$
	 Donor: George Lewis References: [Moore & Ho(1993), Moore et al.(1993b), Abacioglu et al.(1994)] NOTES: B15: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] B15: Binds native BH10 gp120 with 5 fold less affinity than denatured – doe [Moore et al.(1993b)] B15: V4 region – epitope boundaries mapped by peptide scanning [Abacioglus et al. (1993b)] 	993), Moore et al.(19) y to denatured IIIB g gp120 with 5 fold less boundaries mapped	 nor: George Lewis erences: [Moore & Ho(1993), Moore et al.(1993b), Abacioglu et al.(1994)] TES: 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 [Moore et al.(1993b)] B15: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)] 		native or denatured MN gp120	
449 B34	gp120(V4 395-400 BH10)	gp120(394-399)	WFNSTW		mis-folded LAI rgp160	$\operatorname{murine}(\operatorname{IgG}_{2b})$
	Donor: George LewisReferences: [Abacioglu et al.(1994)]NOTES:B34: V4 region – epitope bounda	.(1994)] boundaries mapped	or: George Lewis erences: [Abacioglu et al.(1994)] TES: B34: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1	u et al.(1994)]		
450 7F11	gp120(397-439 IIIB) gp120(396-440)? Donor: George Lewis References: [Lasky et al.(1987), Nilsen et al.(1996)] NOTES: • 7F11: There is another MAb with this name that	gp120(396-440) 37), Nilsen et al.(1990) IAb with this name the	20(397-439 IIIB) gp120(396-440)? nor: George Lewis erences: [Lasky et al.(1987), Nilsen et al.(1996)] TES: 7F11: There is another MAb with this name that binds to integrase [Nilsen et al.(1996)]	al.(1996)]	purified gp120	murine
451 5C2E5	gp120(C4 406-415 IIIB) gp120(423-432) QFINM Donor: T. Gregory and R. Ward, Genentech, San Francisco References: [Lasky et al.(1987), Cordell et al.(1991)] NOTES:	gp120(423-432) ard, Genentech, San I 87), Cordell et al.(199	QFINMWQEVK Francisco 91)]		purified gp120	murine
	 5C2E5: Blocks the gp120-CD4 interaction [Lasky et al.(1987)] 5C2E5: Cross-competition with MAbs 5C2E5, ICR38.8f and IC 	D-CD4 interaction [Language on with MAbs 5C2E5]	5C2E5: Blocks the gp120-CD4 interaction [Lasky et al.(1987)] 5C2E5: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell et al.(1991)]	dell et al.(1991)]	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
452 G3-211	gp120(C4 423-437 IIIB)	gp120(424-438)	gp120(424-438) IINMWQKVGKAMYAP	Т	virus derived IIIB gp120	$murine(IgG_1)$
	 Donor: T. Gregory and R. Ward, Genentech, San Francisco References: [Sun et al.(1989)] NOTES: G3-211, 42, 299, 508, 519, 536, 537: Cross-react with diverse strains to CD4+ cells – different neutralization efficiencies [Sun et al.(1989)] 	urd, Genentech, San] , 536, 537: Cross-reneutralization efficie	nor: T. Gregory and R. Ward, Genentech, San Francisco erences: [Sun et al.(1989)] TES: G3-211, 42, 299, 508, 519, 536, 537: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun et al.(1989)]	nunofluoresceno	ce – blocks HIV binding	
453 G3-537	gp120(C4 423-437 IIIB)	gp120(424-438)	gp120(424-438) IINMWQKVGKAMYAP	T	virus derived IIIB gp120	$murine(IgG_1) \\$
	Donor: T. Gregory and R. Ward, Genentech, San Francisco References: [Sun et al.(1989), Ho et al.(1991b), McKeating et al.(1992b)] NOTES:	urd, Genentech, San , Ho et al.(1991b), N	Francisco AcKeating et al.(1992b)]			
	 G3-537, 211, 299, 508, 519, 536, 42: Cross-react with diverse strains to CD4+ cells – different neutralization efficiencies [Sun et al.(1989)] G3-537: Weakly neutralizing – binds to a linear binding domain of gpl et al.(1992b)] 	19, 536, 42: Cross-re neutralization efficie zing – binds to a linea	G3-537, 211, 299, 508, 519, 536, 42: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun et al.(1989)] G3-537: Weakly neutralizing – binds to a linear binding domain of gp120, NMWQEVGKAMYAPPISG [McKeating et al.(1992b)]	nunofluorescena MWQEVGKAN	escence – blocks HIV binding GKAMYAPPISG [McKeating	
454 polyclonal	gp120(CD4BS)	gp120(426-437)	NMWQEVGKAMYA	L	oral immunization – peptide plus cholera toxin adjuvant	murine(IgA)
	Donor: T. Gregory and R. Ward, Genentech, San Francisco References: [Bukawa et al.(1995)] NOTES:	ırd, Genentech, San 995)]	Francisco			
	 Polyclonal secretory IgA neutralization may be due et al.(1995)] 	antibody raised by n to the V3, CD4 or H	Polyclonal secretory IgA antibody raised by mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to the V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa et al.(1995)]	o neutralize IIIB omponent pepti	s, SF2, and MN – HIV-1 de immunogen [Bukawa	
455 MO86/C3	gp120(C4 429-443) gp120(430-444) EVGKA Donor: T. Gregory and R. Ward, Genentech, San Francisco References: [Ohlin et al.(1992)] NOTES:	gp120(430-444) urd, Genentech, San 2)]	EVGKAMYAPPISGQI Francisco		rIIIB Env 286-467	human(IgM)
	MO86: Generated throught	h <i>in vitro</i> "immuniza	MO86: Generated through <i>m vitro</i> "immunization" of uninfected-donor lymphocytes		[Onlin et al.(1992)]	

MAb ID	Location	WEAU	Sequence	Neutralizing Immunogen	Immunogen	Species(Isotype)
456 G3-42	gp120(C4 429-438 BRU)	gp120(430-439)	gp120(430-439) EVGKAMYAPP	T	virus derived IIIB gp120	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Sun et al.(1989), Moore et al.(1993b), Thali et al.(1993), Sattentau & Moore(1995), Jagodzinski et al.(1996) Moore & Sodroski(1996), Poignard et al.(1996a), Trkola et al.(1996a), Binley et al.(1997)] NOTES:	ic and David Ho, AD), Moore et al. (1993b) ignard et al. (1996a),	ARC, NY , Thali et al. (1993), Sattentau Trkola et al. (1996a), Binley o	& Moore(1995), et al.(1997)]	Jagodzinski et al.(1996)	•
	• G3-42: Neutralization of IIIB but not RF [Sun et al.(1989)]	IIIB but not RF [Su				,

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

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
457 G3-299	gp120(C4 429-438 BRU)	gp120(430-439) EVGKAMYAPP	EVGKAMYAPP	L	virus derived IIIB gp120	$murine(IgG_1)$
	Donor: M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY References: [Sun et al.(1989), Moore et al.(1993b), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Ditzel et al.(1997), Wyatt et al.(1997), Parren et al.(1998)] NOTES:	iosystems Inc and D), Moore et al.(199: 7), Ditzel et al.(199	avid Ho, ADARC, NY 3b), Sattentau & Moore(1997), Wyatt et al.(1997), Parrer	95), Moore & So et al.(1998)]	droski(1996), Poignard	
	 G3-299: Best neutralizati G3-299: C4 region – bind 299 lower affinity than G3 epitope spans V3-C4 region 	on of IIIB in panel o s HXB2 20mer KQI 3-508, G3-519, and ons – 433A/L, 435Y	G3-299: Best neutralization of IIIB in panel of 7 MAbs that bind overlapping epitope [Sun et al.(1989)] G3-299: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – G3-42, G3-299 lower affinity than G3-508, G3-519, and G3-536 – bound native gp120, not denatured – poor peptide binding, epitope spans V3-C4 regions – 433A/L, 435Y/H and 430V/S substitutions impaired binding, V3 loop cleavage or	ing epitope [Sun, and SF-2 and M.O., not denatured s impaired bindings of the state	[Sun et al.(1989)] and MN gp120s – G3-42, G3- atured – poor peptide binding, binding, V3 loop cleavage or	
	 insertion abolished binding [Moore et al.(1993b)] G3-299: Binds with higher affinity to monomer t C4 MAbs tested, with more potent neutralization 	g [Moore et al.(1993) er affinity to monom re potent neutralizati	insertion abolished binding [Moore et al.(1993b)] G3-299: Binds with higher affinity to monomer than to oligomer, slow association rate, although faster than other C4 MAbs tested, with more potent neutralization of lab strain [Sattentau & Moore(1995)]	sociation rate, al	hough faster than other	
	binding reciprocally inhib Sodroski(1996)]	ited by anti-V3 MA	binding reciprocally inhibited by anti-V3 MAbs – G3-229 enhances the binding of some anti-V2 MAbs [Moore & Sodroski(1996)]	inding of some a	ng she, and vz maos— nti-V2 MAbs [Moore &	
	 G3-299: Epitope described as KQIINMWQKVGKAMYAP virus and exposure of the gp41 epitope for MAb 50-69 [Poi G3-299: A low avidity antibody as assessed by urea elution 	d as KQIINMWQK gp41 epitope for M <i>t</i> tibody as assessed b	G3-299: Epitope described as KQIINMWQKVGKAMYAPPIS – binding resulted in virus and exposure of the gp41 epitope for MAb 50-69 [Poignard et al.(1996a)] G3-299: A low avidity antibody as assessed by urea elution		slight gp120 dissociation from	
	 G3-299: Binds both gp120 and solub by gp41 binding [Wyatt et al.(1997)] G3-299: The MAb and Fab binding 	and soluble gp120- al.(1997)] ab binding to the ol	G3-299: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt et al.(1997)] G3-299: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated —	ıggesting its gp12 1 neutralization v	0 epitope is not blocked/ ere highly correlated –	
	epitope [Parren et al.(1998)]	3)]	epitope [Parren et al. (1998)]	o coordinate	on a smion micopocasio oi mo	

					(
458 G3-508	gp120(C4 429-438 BRU)	gp120(430-439)	gp120(430-439) EVGKAMYAPP	Т	virus derived IIIB gp120	$murine(IgG_1)$
	Donor: M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY References: [Sun et al.(1989), Thali et al.(1993), Moore et al.(1993b), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Trkola et al.(1996a), Binley et al.(1997), Parren et al.(1998), Binley et al.(1998)] NOTES:	siosystems Inc and I 9), Thali et al.(199 1996a), Trkola et al.	David Ho, ADARC, NY 33), Moore et al.(1993b), S (1996a), Binley et al.(1997),	Sattentau & Moor , Parren et al.(1998	e(1995), Moore & So- s), Binley et al.(1998)]	
	 G3-508: Neutralization of IIIB and RF [Sun et al.(1989)] G3-508: Inhibits binding of CD4 inducible MAb 48d [Th 	f IIIB and RF [Sun of CD4 inducible N	G3-508: Neutralization of IIIB and RF [Sun et al.(1989)] G3-508: Inhibits binding of CD4 inducible MAb 48d [Thali et al.(1993)]			
	• G3-508: C4 region – bit denatured with 10 fold g	nds HXB2 20mer I reater affinity than	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 IMCora at 21 (1003b)1	PIS, and SF-2 and 430V/S substit	F-2 and MN gp120s – bound substitutions impaired binding	
	G3-508: Binds with highG3-508: Inhibits binding	er affinity to monon of some V3, C4 and	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	sociation rate [Sat nances binding of v	tentau & Moore(1995)] V2 region MAbs [Moore	
	& Sodroski(1996)] • G3-508: Rinding resulted	l in cliaht an120 dis	& Sodroski(1996)] 63.508: Rinding resulted in slight an 120 dissociation from virus and exposure of the and 1 enitone for MAA 50-60	∞ of the ∞	enitone for MAh 50-69	
	[Poignard et al.(1996a)]			,		
	• G3-508: Also called G3 5 et al.(1996a)]	08 – inhibits gp120	G3-508: Also called G3 508 – inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [11Kola et al.(1996a)]	MIP-1β-CCK-5 cc	impetition study [Trkola	_
	 G3-508: A low avidity antibody as assessed by urea elution G3-508: The MAb and Fab binding to the oligomeric form 	tibody as assessed labeled	G3-508: A low avidity antibody as assessed by urea elution G3-508: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated -	nd neutralization v	vere highly correlated –	
	authors suggest that neutr	alization is determi	authors suggest that neutralization is determined by the fraction of Ab sites occupied	es occupied on a	on a virion irrespective of the	
	 epitope [Fairen et al.(1998)] G3-508: A panel of MAbs v 	8)] os were shown to bi	epitope [Parren et al. (1998)] G3-508: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a	finity and similar	ompetition profiles to a	
	deglycosylated or variable a structure closely approx	e loop deleted core imating full length	deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)]	ınd V3), thus such ป.(1998)]	a core protein produces	-

MAb ID	MAb ID Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
459 G3-519	gp120(C4 429-438 BRU)	gp120(430-439)	gp120(430-439) EVGKAMYAPP	L	virus derived IIIB gp120	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY Before 15 (1990) Moor & Ho(1992) Moore at al. (1992) D'Source at al. (1994) Settoutes & Moore (1995)	c and David Ho, AD	DARC, NY	20 of al (1004) &	attenton & Moore (1005	

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

NOTES:

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

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
460 G3-536	gp120(C4 429-438 BRU)	gp120(430-439)	gp120(430-439) EVGKAMYAPP	Т	virus derived IIIB gp120	$murine(IgG_1)$
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Sun et al.(1989), Ho et al.(1991b), Cordell et al.(1991), McKeating et al.(1992b), Moore & Ho(1993), Moore et al.(1993b), Gorny et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Parren et al.(1998)] NOTES:	c and David Ho, AD), Ho et al.(1991b), t al.(1994), Sattenta	ARC, NY Cordell et al.(1991), McKo u & Moore(1995), Moore &	eating et al.(1992) 2 Sodroski(1996),	o), Moore & Ho(1993), Poignard et al.(1996a),	
	• G3-536: Weak neutraliza binding to CD4+ cells – 6	tion of IIIB and RF -	G3-536: Weak neutralization of IIIB and RF – cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – epitope:IINMWQKVGKAMYAP [Sun et al.(1989)]	uns by immunoflu 9)]	orescence – blocks HIV	
	G3-536: Cross-competitiG3-536: Weakly neutral	on with MAbs 5C2E izing – binds to a li	G3-536: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell et al.(1991)] G3-536: Weakly neutralizing – binds to a linear determinant in the CD4 binding domain of gp120 [McKeating	Cordell et al.(199) 14 binding domain	 f gp120 [McKeating 	
	et al.(1992b)] • G3-536: Neutralizes IIIR	is reactive with SF-	et al.(1992b)] G3-536: Neutralizes IIIB is reactive with SF-2 on 120 mild inhibition of HIV-1+ sera		hinding to IIIB on120 [Moore	
	& Ho(1993)]		,		;	
	 G3-536: C4 region – bi denatured with 15 fold g 	nds HXB2 20mer K reater affinity than n	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	PIS, and SF-2 an 8P/R, and 430V/	d MN gp120s – bound substitutions impaired	
	 binding [Moore et al.(1993b)] G3-536: Enhances binding of anti-V2 MAb 697-D [Gorny et al.(1994)] 	93b)] no of anti-V2 MAh 6	97-D [Gorny et al (1994)]			
	• G3-536: Binds with high	er affinity to monom	G3-536: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]	sociation rate [Sat	tentau & Moore(1995)]	
	 G3-536: Inhibits binding & Sodroski(1996)] 	of some V3, C4 and	G3-536: Inhibits binding of some V3, C4 and CD4 binding site MAbs, enhances binding of V2 region MAbs [Moore & Sodroski (1996)]	ances binding of \	V2 region MAbs [Moore	
	 G3-536: Epitope describe 	ed as KVGKAMYAI	G3-536: Epitope described as KVGKAMYAPP [Poignard et al.(1996a)]			
	• G3-536: The MAb and I	ralization is determin	G3-536: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated –	d neutralization v	vere highly correlated –	
	epitope [Parren et al.(1998)]	8)]	•	٠	-	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen
461 ICR38.1a	pp120(C4 429-438 BRU) gp120(430-439) EVGKAMYAPP Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Cordell et al.(1991), McKeating et al.(1992b), McKeating et al.(1992a), McKeating et al.(1992b), McKeating et al.(1991)] ICR38.1a: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with MAbs G3-536, SC2E5, and ICR38.8f [McKeating et al.(1992b), Cordell et al.(1991)] ICR38.1a: Unable to exert a synergistic effect in combination with V3 directed MAbs, in contrast to MAb 39.13g, that binds to a conformational epitope involved in CD4 binding [McKeating et al.(1992a)] ICR38.1a: Studied in the context of a neutralization escape mutant [McKeating et al.(1993a)] ICR38.1a: Unreactive with solid-phase decapeptide, competed in solution phase assay – ICR 38.1a and ICR 38.8f were initially reported to be independent MAbs, but are actually subclones of the same MAb [Moore et al.(1993b)] ICR38.1a: Called 38.1a – 10 to 20 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)] ICR38.1a: UK Medical Research Council AIDS reagent: ARP388/ARP389	gp120(430-439) nc and David Ho, AI 991), McKeating et a ul.(1993a), Moore et 8.1a 8.1a 8.1a fralizing – binds lina and ICR38.8f [McKeat asynergistic effettional epitope involve context of a neutra ith solid-phase decap be independent MA – 10 to 20 fold incre Research Council A	20(C4 429-438 BRU) gp120(430-439) EVGKAMYAPP L rBH10 gp120 nor: Tanox Biosystems Inc and David Ho, ADARC, NY erences: [Cordell et al.(1991), McKeating et al.(1992b), McKeating et al.(1992a), McKeating et al.(1992), McKeating et al.(1993b), McKeating et al.(1995)] TES: ICR38.1a: Also called 38.1a ICR38.1a: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with MAbs G3-536, 5C2E5, and ICR38.8f [McKeating et al.(1992b), Cordell et al.(1991)] ICR38.1a: Unable to exert a synergistic effect in combination with V3 directed MAbs, in contrast to MAb 39.13g, that binds to a conformational epitope involved in CD4 binding [McKeating et al.(1992a)] ICR38.1a: Studied in the context of a neutralization escape mutant [McKeating et al.(1993a)] ICR38.1a: Unreactive with solid-phase decapeptide, competed in solution phase assay – ICR 38.1a and ICR 38.8f were initially reported to be independent MAbs, but are actually subclones of the same MAb [Moore et al.(1993b)] ICR38.1a: Called 38.1a – 10 to 20 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)] ICR38.1a: UK Medical Research Council AIDS reagent: ARP388/ARP389	L (1992a), McKeating (5)] binding domain – let al.(1991)] directed MAbs, in outing et al.(1992a)] Keating et al.(1992a) Keating et al.(1993a) ion phase assay – Iones of the same MA or V1/V2 and V3 v	rBH10 gp120 et al.(1992), McKeating cross-competition with contrast to MAb 39.13g
462 ICR38.8f	gp120(C4 429-438 BRU) gp120(430-439) EVGKA Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Cordell et al.(1991)] NOTES: • ICR38.8f: Weakly neutralizing – binds linear determic ICR38.1a, 5C2E5, and G3-536 [Cordell et al.(1991)] • ICR38.8f:ICR 38.1a and ICR 38.8f were initially reported same MAb [Moore et al.(1993b)]	gp120(430-439) nc and David Ho, AI 991)] ralizing – binds line ralizing fordell et al ICR 38.8f were initi	20(C4 429-438 BRU) gp120(430-439) EVGKAMYAPP L rBH10 gp120 100: Tanox Biosystems Inc and David Ho, ADARC, NY 100: Tanox Biosystems Inc and David Ho, ADARC, NY 100: Perences: [Cordell et al.(1991)] 101: Perences: [Cordell et al.(1991)] 101: Perences: [Cordell et al.(1991)] 102: Perences: [Cordell et al.(1991)] 103: Perences: [Cordell et al.(1991)] 103: Perences: [Cordell et al.(1991)] 104: Perences: [Cordell et al.(1991)] 105: Perences: [Cordell et al.(1991)] 106: Perences: [Cordell et al.(1991)] 107: Perences: [Cordell et al.(1991)] 108: Perences: [Cordell et al.(1991)] 109: Perences: [Cordell et al.(1991)] 100: Perence	L binding domain – dent MAbs, but are :	rBH10 gp120 cross-competition with actually subclones of the

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
463 G45-60	gp120(C4 429-438 BRU) gp120(432-441) GKAMY Donor: Tanox Biosystems Inc and David Ho, ADARC, NY	gp120(432-441) nc and David Ho, AD.	GKAMYAPPIS ARC, NY	T	virus derived IIIB gp120	$\operatorname{murine}(\operatorname{Ig} \operatorname{G}_1)$
	References: [Sun et al.(1989), Moore et NOTES: • G45-60: C4 region – binds HXB2 2 – bound equivalently to native and binding [Moore et al.(1993b)] • G45-60: Enhances binding of anti- • G45-60: Non-reciprocal enhancem of some V2 region MAbs – reciproregions [Moore & Sodroski(1996)] • G45-60: The sulfated polysaccharic virus CRDS inhibits G45-60 bindin	nc and David Ho, AD, Moore et al. (1993b), Moore et al. (1993b) ids HXB2 20mer KQ native and denature (93b)] ing of anti-V2 MAb (enhancement of G45 bs – reciprocal inhibitski (1996)] lysaccharide curdlan (5-60 binding [Jagodz.	 Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Sun et al.(1989), Moore et al.(1993b), Gorny et al.(1994), Moore & Sodroski(1996), Jagodzinski et al.(1996)] NOTES: G45-60: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPI, decapeptide flanking peptides also bound – bound equivalently to native and denatured gp120 – 433A/L and 435Y/H (not 430V/S) substitutions impaired binding [Moore et al.(1993b)] G45-60: Enhances binding of anti-V2 MAb 697-D [Gorny et al.(1994)] G45-60: Non-reciprocal enhancement of G45-60 binding by some C1 and C5 antibodies – reciprocal enhancement of some V2 region MAbs – reciprocal inhibition with many MAbs that bind to the V3, C4 and CD4 binding site regions [Moore & Sodroski(1996)] G45-60: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus CRDS inhibits G45-60 binding [Jagodzinski et al.(1996)] 	odroski(1996), scapeptide flank H (not 430V/S H (not 430V/S cantibodies – d to the V3, C	1996), Jagodzinski et al.(1996)] le flanking peptides also bound 30V/S) substitutions impaired dies – reciprocal enhancement V3, C4 and CD4 binding site F-tropic viruses and neutralizes	
	virus CRDS inhibits G45-60 binding [Jagodzinski et al.(1996)]	5-60 binding [Jagodz	inski et al.(1996)]		c viruses and neutralizes	
464 1662	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	Z	poliovirus-antigen chimera	
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [McKeating et al.(1992b)] NOTES:	nc and David Ho, AD al.(1992b)]	ARC, NY			
	• 1662: Did not bind to na	ntive gp120, epitope r	1662: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]	.992b)]		
465 1663	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	Z	poliovirus-antigen chimera	
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [McKeating et al.(1992b)] NOTES:	nc and David Ho, AD al.(1992b)]	ARC, NY			
	• 1663: Did not bind to na	ntive gp120, epitope r	1663: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]	992b)]		
466 1664	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	Z	poliovirus-antigen chimera	
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [McKeating et al.(1992b)] NOTES:	nc and David Ho, AD al.(1992b)]	ARC, NY			
	• 1664: Did not bind to na	ative gp120, epitope r	1664: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]	992b)]		

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
467 1697	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	Z	poliovirus-antigen chimera	
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [McKeating et al.(1992b)] NOTES: 1607: Did not hind to native con120 enitone not expose	Inc and David Ho, AD, t al.(1992b)]	onor: Tanox Biosystems Inc and David Ho, ADARC, NY eferences: [McKeating et al.(1992b)] OTES: • 1607: Did not hind to native on 120 entrope not exposed (McKeating et al.(1992b))	(1902)		
468 1794	gp120(C4 IIIB)	gp120(434-443) AMYAPPISGQ	AMYAPPISGQ	Z	poliovirus env	
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [McKeating et al.(1992b)] NOTES:	Inc and David Ho, AD, t al.(1992b)]	ARC, NY		VILLION	
	• 1794: Did not bind to	native gp120, epitope n	1794: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]	(1992b)]		
469 1804	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	Z	poliovirus env chimera	
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [McKeating et al.(1992b)]	Inc and David Ho, AD, t al.(1992b)]	ARC, NY			
	• 1804: Did not bind to	native gp120, epitope n	1804: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]	(1992b)]		
470 1807	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	Z	poliovirus env chimera	
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [McKeating et al.(1992b)]	Inc and David Ho, ADatal.(1992b)]	ARC, NY			
	• 1807: Did not bind to	native gp120, epitope no	1807: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]	.(1992b)]		
471 1808	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	Z	poliovirus env	
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [McKeating et al.(1992b)]	Inc and David Ho, ADat al.(1992b)]	ARC, NY		CHIHETA	
	• 1808: Did not bind to	native gp120, epitope no	1808: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]	(1992b)]		

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
472 1795	gp120(CD4BS 425-441 IIIB)	gp120(426-442)	OPISG	L	poliovirus env chimera	
	 Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [McKeating et al.(1992b)] NOTES: 1795: CD4 binding site – weakly neutralizing – bindir [McKeating et al.(1992b)] 	and David Ho, AD, (1992b)] weakly neutralizing	nor: Tanox Biosystems Inc and David Ho, ADARC, NY ferences: [McKeating et al.(1992b)] YTES: 1795: CD4 binding site – weakly neutralizing – binding inhibited by WQEVGKAM [McKeating et al.(1992b)]		YA, GKAM may be involved	
473 13H8	gp120(C4 412-453) gp120(432-441) GKAMYAPPIS Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Nakamura et al.(1992), Nakamura et al.(1993), Jeffs et al.(1996)] NOTES:	gp120(432-441) GKAMYAPPIS and David Ho, ADARC, NY 1992), Nakamura et al.(1993), Jeffs o	GKAMYAPPIS ARC, NY t al.(1993), Jeffs et al.(1996)]	L	rgp120 MN	murine(IgG)
	 13H8: Cross blocks 5C2 in IIIB-rsgp160 ELISA – reac et al.(1992)] 13H8: Bound diverse strains, neutralizing activity against N 13H8: Binds V3 and C4 peptides (J. P. Moore, per. comm.) 13H8: 3 and 4.5 fold increased binding when V1/V2 or V1, et al.(1996)] 	in IIIB-rsgp160 E ns, neutralizing acti eptides (J. P. Moore ased binding when	13H8: Cross blocks 5C2 in IIIB-rsgp160 ELISA – reactive with diverse strains in rgp120 ELISA [Nakamura et al.(1992)] 13H8: Bound diverse strains, neutralizing activity against MN [Nakamura et al.(1993)] 13H8: Binds V3 and C4 peptides (J. P. Moore, per. comm.) 13H8: 3 and 4.5 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120, respectively [Jeffs et al.(1996)]	$+$ ω	in rgp120 ELISA [Nakamura	
474 1024	gp120(C4) Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Berman et al.(1997)] NOTES: • 1024: Binds to 1/7 isolates from breakthrough cases from	gp120 and David Ho, AD. 97)] s from breakthrough	gp120 or: Tanox Biosystems Inc and David Ho, ADARC, NY rences: [Berman et al.(1997)] IES: 1024: Binds to 1/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman et al.(1997)]	ne trial [Berma	n et al.(1997)]	
475 polyclonal	gp120(V5 LAI)	gp120(462-469) NNNNGSEI	NNNNGSEI		HIV-1 infection augmented by gp160 vaccine	human
	 Donor: Tanox Biosystems Inc and David Ho, ADARC, NY References: [Loomis-Price et al.(1997)] NOTES: HIV-1+ positive individuals were given a gp160 vaccine epitope as measured by a modified Pepscan technique 	and David Ho, AD, al.(1997)] s were given a gp16 modified Pepscan t	nor: Tanox Biosystems Inc and David Ho, ADARC, NY erences: [Loomis-Price et al.(1997)] TES: HIV-1+ positive individuals were given a gp160 vaccine as immunotherapy, and this region was the most reactive new entione as measured by a modified Penscan technique which improved sensitivity – 4/14 showed vaccine-induced entione as measured.	d this region w	gion was the most reactive new 4/14 showed vaccine-induced	

MAb ID	Location	WEAU	Sequence	Neutralizing Immunogen		Species(Isotype)
476 M91	gp120(V5 C5 451-470 LAI) gg Donor: Fulvia di Marzo Veronese	gp120(463-472) SNNESEIFRL nese	SNNESEIFRL	Z	451 Env	$\operatorname{rat}(\operatorname{IgG}_{2a})$
	Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996), Ditzel et al.(1997), Binley et al.(1998)] NOTES:	ese se et al.(1992), Moc)]	ore et al.(1994c), Moore et al.(1994d), Moore &	: Sodroski(1996), Ditzel	
	 M91: Immunoblot reactive, RIP negative, but pr RF, and RUTZ [di Marzo Veronese et al.(1992)] 	e, RIP negative, but Veronese et al.(199)	M91: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – react RF, and RUTZ [di Marzo Veronese et al.(1992)]	p120 – reacts wit	ts with strains IIIB, 451, MN,	
	• M91: The relative affinity	for denatured/nativ	M91: The relative affinity for denatured/native gp120 is 24 – mutation in position 470	osition 470 P/L	P/L impairs binding [Moore	
	• M91: 470 P/L impairs bir	nding, but not 475	M91: 470 P/L impairs binding, but not 475 D/V, in contrast to CRA1 – some C2 mutations can enhance binding	ome C2 mutatio	ns can enhance binding	
	[Moore et al.(1994d)]	sitono bindo wook	[Moore et al.(1994d)] Mol: C5 region linear enitement hinds weakly to nearly and meanwrise en 120	: cs120 Mo1	MOI hinding was anhanced	
	by 1C1, but 1C1 binding v	was inhibited by M	by 1C1, but 1C1 binding was inhibited by M91 – non-reciprocal binding enhancement of C1 and V2 antibodies –	enhancement of	C1 and V2 antibodies –	
	non-reciprocal binding inh	ibition of CD4 bin	non-reciprocal binding inhibition of CD4 binding site antibodies [Moore & Sodroski(1996)]	: Sodroski(1996)		
	 M91: A panel of MAbs v 	vere shown to binc	M91: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a	ity and similar c	ompetition profiles to a	
	deglycosylated or variable a structure closely approxi	loop deleted core a mating full length i	deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus a structure closely approximating full length folded monomer [Binley et al.(1998)]		such a core protein produces	

477 CRA1(ARP gp120(V5-C5 451-470 LAI) gp120(463-472) SNNESEIFRL

Z

Env glycopro

/copro

murine(IgG)

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

• CRA1: Also called CRA-1

CRA1: Bound preferentially to denatured IIIB and SF2 gp120 [Moore & Ho(1993)]

CRA1: Some C5 mutations abrogate binding 470 P/L or G, 475 M/S, some C2 mutations enhance binding [Moore et al.(1994d)]

CRA1: The relative affinity for denatured/native gp120 is 24 – C5 mutations 470 P/L or G, 475 M/S impairs binding to the native gp120 - only mutation 470 P/L impairs binding to denatured [Moore et al.(1994c)]

• CRA1: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – reciprocal binding inhibition with binding inhibition of some CD4 binding site antibodies [Moore & Sodroski(1996)] anti-C5 antibodies 1C1 and M91 - non-reciprocal binding enhancement some C1 and V2 antibodies - non-reciprocal

• CRA1: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]

CRA1: UK Medical Research Council AIDS reagent: ARP323

483 IC1		481 H11 482 M38	478 9301	MAb ID
gp120(C5 471-490 LAI) gp120(473-492) Donor: Repligen Inc, Cambridge, MA, commercial References: [Moore et al.(1994c), Moore et al.(199 NOTES: • 1C1: The relative affinity for denatured/native g • 1C1: C2 and V3 regions substitutions can influe • 1C1: Linear epitope not exposed on conformati • 1C1: C5 region linear epitope, binds weakly to 1C1, but 1C1 binding was inhibited by M91 – n non-reciprocal binding inhibition of some CD4	References: [Beretta et al.(1987), Ggleish(1994)] NOTES: • M38: Binds to gp120 and to a 80 nodes [Beretta et al.(1987)] • M38: Binds to the carboxy terminu homology) [Lopalco et al.(1993)] • M38: Infected individuals have H	gp120(C5 472-477 HXB2) gp120(474-479?) GGDMI References: [Pincus & McClure(1993), Pincus et al.(1996)] NOTES: • H11: Binds to gp120 but not to infected cells – when lin – sCD4 has no effect [Pincus & McClure(1993), Pincus and 120(C5 490-508) graph of the property of th	gp120(C5 471-490 LAI) Donor: Dupont, commercial References: [Skinner et al.(1 NOTES: 9301: Bound preferentia 9301: The relative affini 9301: Wagner et al. clai	Location
gp120(473-492) ridge, MA, commerce 994c), Moore et al.(1) y for denatured/native substitutions can in exposed on conform pitope, binds weakles inhibited by M91 nhibition of some Cl	(1987), Grassi et al d to a 80 kd human p d to a 80 kd human p (7)] (2) terminus of gp120 d.(1993)] ls have HLA class I.	gp120(474-479?) lure(1993), Pincus et. t not to infected cells - ncus & McClure(1993)	gp120(473-492) 988b), Moore & Ho lly to denatured IIII ty for denatured/nati m that Nea 9301 is a	WEAU
GGGDMRDNWRSELYKY- KVVK 4d), VanCott et al.(1995), Moore & S gp120 is 15 [Moore et al.(1994c)] ence binding [Moore et al.(1994d)] onally intact gp120 [VanCott et al.(1900) onondenatured monomeric gp120 — Non-reciprocal binding enhancement of binding site antibodies [Moore & Society 1994]	KRR .(1991), Lopalco et al.(1993), DeSantis protein expressed on a small fraction of m , in a gp41 binding region, and also to dena -gp120 cross-reactive antibodies [DeSanti	20(C5 472-477 HXB2) gp120(474-479?) GGDMRD? erences: [Pincus & McClure(1993), Pincus et al.(1996)] TES: H11: Binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)] 20(C5 400-508)	gp120(C5 471-490 LAI) gp120(473-492) GGGDMRDNWRSELYKY- Env glycopro KVVK Donor: Dupont, commercial KVVK References: [Skinner et al.(1988b), Moore & Ho(1993), Moore et al.(1994c), Moore et al.(1994d), Wagner et al.(1990) • 9301: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • 9301: The relative affinity for denatured/native gp120 is 19 [Moore et al.(1994d)] • 9301: Wagner et al. claim that Nea 9301 is anti-V3 – might they have meant MAb 9305? [Wagner et al.(1996)]	Sequence Neutrali
Env glycopro odroski(1996)] 95)] 95)] 195) Some C1 and V2 antibodies – lroski(1996)]	et al.(1994), Beretta & Dalononuclear cells in the lymph tured human HLAs (antigenic s et al.(1994)]	in did not mediate cell killing	Env glycopro .(1994d), Wagner et al.(1996)]	lizing Immunogen
murine (IgG)		murine	murine(IgG)	Species(Isotype)

MAb ID	Location	WEAU	Sequence Neutr	Neutralizing	Immunogen	Species(Isotype)
484 B221	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY- KVVK		Baculovirus- expressed mis- folded rgp160 IIIB:NL43, MicroGenSys	$\operatorname{murine}(\operatorname{Ig} \operatorname{G}_{1\kappa})$
	Donor: Rod Daniels References: [Moore & Ho(1993), Bristow et al.(1994), Moore et al.(1994c)] NOTES:	93), Bristow et al.(1	994), Moore et al.(1994c)]			
	B221: Called 221 – boundB221: MAbs generated in	d preferentially to do the context of a student	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 rgp	93)] rgp120 aı	120 and rgp160 – boundaries	
	 described as 443-462 of LAI [Bristow et al.(1994)] B221: The relative affinity for denatured/native gp120 is 12 – m B221: Called 221 – C2 and V3 substitutions influence binding B221: UK Medical Research Council AIDS reagent: ARP301 	Al [Bristow et al.(1) for denatured/native id V3 substitutions if rch Council AIDS r	described as 443-462 of LAI [Bristow et al.(1994)] B221: The relative affinity for denatured/native gp120 is 12 – mutation 477 D/V impairs binding [Moore et al.(1994c)] B221: Called 221 – C2 and V3 substitutions influence binding [Moore et al.(1994d)] B221: UK Medical Research Council AIDS reagent: ARP301	irs bindin)]	g [Moore et al.(1994c)]	
485 660-178	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY- KVVK		Env glycopro	murine(IgG)
	Donor: G. Robey, Abbott Labs References: [Moore et al.(1994c), Moore et al.(1994d)] NOTES:	s 94c), Moore et al.(19	994d)]			
	 660-178: The relative affi 660-178: ΔV1/V2 and Δ 	nity for denatured/n V1/V2/V3 reduce b	660-178: The relative affinity for denatured/native gp120 is >100 [Moore et al.(1994c)] 660-178: $\Delta V1/V2$ and $\Delta V1/V2/V3$ reduce binding – C2 and C5 mutations enhance binding [Moore et al.(1994d)]	94c)] e binding	[Moore et al.(1994d)]	
486 8C6/1	gp120(V5-C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY- KVVK		Env glycopro	murine(IgG)
	Donor: S. Ranjbar, NIBSC, UK References: [Moore et al.(1994c)] NOTES:	K)4c)]				
	 8C6/1: V5-C5 region – preferentially binds SDS-DTT denatur binding [Moore et al.(1994c)] 8C6/1: UK Medical Research Council AIDS reagent: ARP3052 	referentially binds 4c)] arch Council AIDS	8C6/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>30 fold) binding [Moore et al.(1994c)] 8C6/1: UK Medical Research Council AIDS reagent: ARP3052	I	mutation 485 K/V impairs	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
487 5F4/1	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY- KVVK		Peptide	murine
	Donor: S. Ranjbar, NIBSC, UK References: [Moore et al.(1994c)] NOTES: • 5F4/1: V5-C5 region – prefer binding [Moore et al.(1994c)]	UK 94c)] preferentially binds 94c)]	nor: S. Ranjbar, NIBSC, UK ferences: [Moore et al.(1994c)] TES: 5F4/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>10 fold) binding [Moore et al.(1994c)]	I	mutation 485 K/V impairs	
488 3F5	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRSELYKY- KVVK		Env	murine(IgG)
	Donor: S. Nigida, NCI, USA References: [Moore et al.(1994c)] NOTES: • 3F5: The relative affinity for d	94c)] for denatured/native	 onor: S. Nigida, NCI, USA eferences: [Moore et al.(1994c)] OTES: Figure 3F5: The relative affinity for denatured/native gp120 is 100 [Moore et al.(1994c)] 	94c)]		
489 MO101/ V3,C4	gp120(V3 314-323 + C5 gp120 494-503) Donor: S. Nigida, NCI, USA References: [Ohlin et al.(1992)] NOTES: • MO101: generated through <i>in vitro</i> "in V3 and C4 regions [Ohlin et al.(1992)]	gp120 (2)] gh <i>in vitro</i> "immuniz n et al.(1992)]	20(V3 314-323 + C5 gp120 GRAFVTIGKI + LGVA-503) PTKAKR nor: S. Nigida, NCI, USA erences: [Ohlin et al.(1992)] TES: MO101: generated through <i>in vitro</i> "immunization" of uninfected-donor lymphocytes: V3 and C4 regions [Ohlin et al.(1992)]		pB1 (IIIB Env 286-467)	human(IgM)
490 9201	gp120(C5 475-486 LAI) gp120(473-484) GGGDM Donor: Du Pont References: [McDougal et al.(1996)] NOTES: • 9201: Does not neutralize LAI [McDougal et al.(1996)]	gp120(473-484) L.(1996)] e LAI [McDougal et	gp120(473-484) GGGDMRDNWRSE? 996)] AI [McDougal et al.(1996)]	Z		murine

MAb ID	Location	WEAU	Sequence Neutralizing	ing Immunogen	Species(Isotype)
491 W2	gp120(C5 472-491 LAI)	gp120(474-493)	GGDMRDNWRSELYKYK- VVKI	Env	murine(IgG)
	Donor: D. Weiner, U. Penn., USA References: [Moore et al.(1994c)] NOTES: • W2: The relative affinity for definity fo	JSA 4c)] or denatured/native;	or: D. Weiner, U. Penn., USA erences: [Moore et al.(1994c)] TES: W2: The relative affinity for denatured/native gp120 is 30 – mutation 485 K/V impairs binding [Moore et al.(1994c)]	inding [Moore et al.(1994c)]	
479 42F	gp120(C5 491-500 HXB2) gp120(483-492) IEPLGV/ References: [Alsmadi et al.(1997), Alsmadi & Tilley(1998)] NOTES:	gp120(483-492) IEPLGVAPTK 997), Alsmadi & Tilley(1998)]	IEPLGVAPTK N ley(1998)]	HIV-1 infection	human $(\operatorname{IgG}_1\lambda)$
	 42F: 42F and 43F were iso taken 14 months apart – but for ADCC if the cell was in 42F: A study of 6 anti-Env SF-2, and RF – bound and Tilley(1998)] 	blated from a long to oth MAbs stained di infected with HIV-1. MAbs and their abil directed lysis again	42F: 42F and 43F were isolated from a long term non-progressor by EBV transformation of PBMC – samples were taken 14 months apart – both MAbs stained diverse strains of infected cells and directed ADCC – were more potent for ADCC if the cell was infected with HIV-1, rather than just presenting absorbed gp120 [Alsmadi et al.(1997)] 42F: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against strains IIIB, MN, SF-2, and RF, but not a clone of MN [Alsmadi & Tilley(1998)]	on of PBMC – samples were d ADCC – were more potent 20 [Alsmadi et al.(1997)] cells infected with IIIB, MN, at a clone of MN [Alsmadi &	
480 43F	gp120(C5 491-500 HXB2) gp1 References: [Alsmadi et al.(1997)] NOTES: • 42F: 42F and 43F were isolated taken 14 months apart – both M for ADCC if the cell was infect	gp120(483-492) 97)] blated from a long to th MAbs stained di infected with HIV-1.	20(C5 491-500 HXB2) gp120(483-492) IEPLGVAPTK N HIV-1 infection erences: [Alsmadi et al.(1997)] TES: 42F: 42F and 43F were isolated from a long term non-progressor by EBV transformation of PBMC – samples were taken 14 months apart – both MAbs stained diverse strains of infected cells and directed ADCC – were more potent for ADCC if the cell was infected with HIV-1, rather than just presenting absorbed gp120 [Alsmadi et al.(1997)]	HIV-1 infection on of PBMC – samples were d ADCC – were more potent 20 [Alsmadi et al.(1997)]	human(Ig $\mathbf{G}_1\lambda$)
492 Chim 1	gp120(C5 492-498 HXB2)	120(489-495)	KVVKEIP?		humanized chimpanzee
	 Donor: D. Weiner, U. Penn., USA References: [Pincus & McClure(1993), Pincus et al.(1996)] NOTES: Chim 1: Also called C-1 Chim 1: binds to gp120 but not to infected cells – who killing – sCD4 has no effect [Pincus & McClure(1993), 	re(1993), Pincus et re(1993), Pincus et out not to infected cct [Pincus & McClu	 nor: D. Weiner, U. Penn., USA erences: [Pincus & McClure(1993), Pincus et al.(1996)] TES: Chim 1: Also called C-1 Chim 1: binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus et al.(1996)] 	notoxin did not mediate cell	chimpanzee
493 RV110026	gp120(C5 491-500 LAI) gp120(493-502) IEPI Donor: Commercial, Olympus Inc References: [Moore et al.(1994c), Moore et al.(1994d)]	gp120(493-502) s Inc 4c), Moore et al.(19	IEPLGVAPTK 94d)]	Peptide	human
	• RV110026: Preferentially et al.(1994c)]	binds SDS-DTT	RV110026: Preferentially binds SDS-DTT denatured gp120 (15 fold using R1/87 et al.(1994c)]	as capture reagent) [Moore	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
494 110.1	gp120(C5 491-500 LAI)	gp120(493-502)	IEPLGVAPTK	Z	BRU infected cell lysates	$\mathrm{murine}(\mathrm{IgG}_{1\kappa})$
	 Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas References: [Gosting et al.(1987), Linsley et al.(1988), Kinney Thomas et al.(1988), et al.(1994c), Cook et al.(1994), McDougal et al.(1996), Binley et al.(1997), Valenzuela et NOTES: 110.1: There is another antibody with this ID that binds to gp120, but at aa 200-217 [P 110.1: Referred to as 110-1 – does not inhibit CD4-gp120 binding or neutralize HIV-1 110.1: Difference in the epitope: mapped to aa 421-429 (KQIINMWQE), the T1 se immunotoxin when linked to RAC [Pincus et al.(1991)] 	p, Seattle WA, E. Kii (1987), Linsley et a 1), McDougal et al.(1) ntibody with this ID ntibody with this ID 1 – does not inhibit epitope: mapped to d to RAC [Pincus et	or: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas rences: [Gosting et al.(1987), Linsley et al.(1988), Kinney Thomas et al.(1988), Pincus et al.(1991), Moore (1994c), Cook et al.(1994), McDougal et al.(1996), Binley et al.(1997), Valenzuela et al.(1998)] [TES: 110.1: There is another antibody with this ID that binds to gp120, but at aa 200-217 [Pincus et al.(1996)] 110.1: Referred to as 110-1 – does not inhibit CD4-gp120 binding or neutralize HIV-1 strains [Linsley et al.(1988)] 110.1: Difference in the epitope: mapped to aa 421-429 (KQIINMWQE), the T1 sequence – poor efficacy as an immunotoxin when linked to RAC [Pincus et al.(1991)]	al.(1988), Pincus et lenzuela et al.(1998)] 200-217 [Pincus et al alize HIV-1 strains [L , the T1 sequence –	Pincus et al.(1991), Moore al.(1998)] incus et al.(1996)] strains [Linsley et al.(1988)] quence – poor efficacy as an	
	 Immunotoxin when linked to RAC [Pincus et al.(1991)] 110.1: The relative affinity for denatured/native gp120 is 0.7 [M 110.1: MAbs against the glycosphingolipid GalCer block HIV i from the brain and colon – MAbs against the carboxy-terminus potently as anti-V3 MAbs – binding of GalCer to gp120 does not potently as not neutralize HIV-1 LAI [McDougal et al.(1996)] 110.1: A high avidity antibody as assessed by urea elution 110.1: Does effect LAI viral binding or entry into CEM cells [V 	d to KAC [Pincus et dy for denatured/native y for denatured/native y for denatured/native y for denatured/native Glycosphingolipid Glycosp	Immunotoxin when linked to KAC [Pincus et al.(1991)] 110.1: The relative affinity for denatured/native gp120 is 0.7 [Moore et al.(1994c)] 110.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the carboxy-terminus of gp120 inhibit gp120 binding to GalCer but not as potently as anti-V3 MAbs – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)] 110.1: Does not neutralize HIV-1 LAI [McDougal et al.(1996)] 110.1: A high avidity antibody as assessed by urea elution 110.1: Does effect LAI viral binding or entry into CEM cells [Valenzuela et al.(1998)]	1994c)] normally suscep hibit gp120 bind Ab binding [Coc	ssceptible CD4 negative cells binding to GalCer but not as [Cook et al.(1994)]	
495 GV1G2	gp120(494-499 IIIB)	gp120(496-501) LGVAPT	LGVAPT		gp120 complexed with MAb M77	murine
	Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas References: [Denisova et al.(1996)] NOTES:	p, Seattle WA, E. Ki 1996)]	nney-Thomas			
	• GV1G2: When anti-V3 l linear epitopes – MAbs G [Denisova et al.(1996)]	MAb M77 was bour V12F6 and GV3H1	GV1G2: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV12F6 and GV3H1 are homologous to GV1G2 and were generated in the same experiment [Denisova et al.(1996)]	amunogen, it sti d were generated	it stimulated many MAbs to erated in the same experiment	
496 722-D	gp120(C term 503-509) gp120(505-511) RRVVQRE Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas References: [Laal et al.(1994), Forthal et al.(1995)] NOTES:	gp120(505-511) RRVVQRE p, Seattle WA, E. Kinney-Thomas), Forthal et al.(1995)]	RRVVQRE nney-Thomas))]	Z	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} \operatorname{G}_{1\kappa})$
	722-D: Not neutralizing a722-D: No neutralizing a	lone, could synergiz ctivity, no ADCC act	722-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal et al.(1994)] 722-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]	ody neutralizatio activity [Forthal	n [Laal et al.(1994)] et al.(1995)]	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
497 670-D	gp120(C term 503-509) gp120(500-506) PTKAKRR' Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas References: [Zolla-Pazner et al.(1995), Forthal et al.(1995), Hi	gp120(500-506) , Seattle WA, E. Kir ıl.(1995), Forthal et	gp120(C term 503-509) gp120(500-506) PTKAKRR? NDonor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas References: [Zolla-Pazner et al.(1995), Forthal et al.(1995), Hill et al.(1997), Gorny et al. NOTES:	N ny et al.(1998)]	HIV-1 infection	$ ext{human}(ext{IgG}_{1\lambda})$
	 670-D: Group specific cross-clade binding i 670-D: Not neutralizing, positive ADCC a epitope is RRVVQRE [Forthal et al.(1995)] 	ss-clade binding in s positive ADCC acti rthal et al.(1995)]	 670-D: Group specific cross-clade binding in serotyping study using flow-cytometry 670-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity, epitope is RRVVORE [Forthal et al.(1995)] 		[Zolla-Pazner et al.(1995)] numbering provided suggests	
	• 257-D: Called 257 – gp1 pre-incubation of gp120 w effect [Hill et al.(1997)]	20 can inhibit MP- ith three anti-V3 M	257-D: Called 257 – gp120 can inhibit MIP- 1α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill et al.(1997)]	t this inhibit	ory effect is blocked by in the C5 region had no	
498 450-D	gp120(C term 475-486 or 503-509 BH10)	gp120(500-506)	PTKAKRR (or RRVVQ- RE, or MRDNWRSELYKY depending on reference)	Z	HIV-1 infection?	$\operatorname{human}(\operatorname{IgG}_{1\lambda})$
	Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Durda et al.(1988), Karwowska et al.(1992a Gorny et al.(1994), Cook et al.(1994), Forthal et al.(1995) NOTES:	YU Med Center, N 3), Karwowska et al.((1994), Forthal et al	Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Durda et al.(1988), Karwowska et al.(1992a), Karwowska et al.(1992b), Spear et al.(1993), Laal et al.(1994), Gorny et al.(1994), Cook et al.(1994), Forthal et al.(1995), Manca et al.(1995), Li et al.(1997)] NOTES:), Spear et al. et al.(1997)]	(1993), Laal et al.(1994),	
	 450-D: Also called 450-D-3 and 450D 450-D: Bound to MN, SF-2 and IIIB, but was not neutralizing 450-D: Did not mediate deposition of complement componen 450-D: Not neutralizing alone, could synergize anti-CD4 bind 450-D: Epitope is defined as PTKAKRR [Gorny et al.(1994)] 	-3 and 450D 2 and IIIB, but was sposition of complet lone, could synergize as PTKAKRR [Gor	450-D: Also called 450-D-3 and 450D 450-D: Bound to MN, SF-2 and IIIB, but was not neutralizing [Karwowska et al.(1992a)] 450-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear et al.(1993)] 450-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal et al.(1994)] 450-D: Epitope is defined as PTKAKRR [Gorny et al.(1994)]	al.(1992a)] scted cells [Syneutralization	pear et al.(1993)] on [Laal et al.(1994)]	
	 450-D: MAbs against the prior the brain and colonstrom the brain and colonstrom the binding of GalCer to gp12 450-D: No neutralizing aces 450-D: Virions complexed 450-D: One of 14 human env – 50% neutralization 	glycosphingolipid G – MAbs against the 0 does not inhibit M tivity, no ADCC act I to gp120 Ab facilit MAbs tested for abi ould not be achieve	450-D: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the carboxy-terminus of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)] 450-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] 450-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca et al.(1995)] 450-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 6 μg/ml [Li et al.(1997)]	rmally susceptor inhibit gp ivity [Forthal ppes to Th cel pes to Thypu+, while the following the fol	susceptible CD4 negative cells bit gp120 binding to GalCer – 'orthal et al.(1995)] Th cells [Manca et al.(1995)] +, which expressed HIV-1 IIIB nl [Li et al.(1997)]	
499 750-D	gp120(C term 503-509) gp120(500-506) PTK Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Forthal et al.(1995)] NOTES: 750-D: Not neutralizing positive ADCC activity ar	gp120(500-506) PTKAKRR YU Med Center, NY, NY 95)]	20(C term 503-509) gp120(500-506) PTKAKRR N HIV-1 intor: Susan Zolla-Pazner, NYU Med Center, NY, NY erences: [Forthal et al.(1995)] TES: TES: TES:	N Forthal e	ection	$\operatorname{human}(\operatorname{IgG}_{3\lambda})$

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
500 120-1	gp120(C term 503-532) gp120 ? Donor: ? References: [Chanh et al.(1986), Dalgleish et al.(1988)]	gp120 86), Dalgleish et al.(? (1988)]	Z	Peptide	$\operatorname{murine}(\operatorname{IgM}_\kappa)$
501 polyclonal	gp120(C term 508-516) gp120(505-513) RRVVQ. Donor: ? References: [Palker et al.(1987), Loomis-Price et al.(1997)]	gp120(505-513) RRVVQREKR 87), Loomis-Price et al.(1997)]	RRVVQREKR al.(1997)]		HIV-1 infection	human
	NOTES: • Most HIV-1+ individuals used as a positive control	s have an antibody re l for HIV-1+ gp160 v	ITES: Most HIV-1+ individuals have an antibody response to this epitope – in this study, reactivity to RRVVQREKR was used as a positive control for HIV-1+ gp160 vaccine recipients [Loomis-Price et al.(1997)]	study, reactivity et al.(1997)]	y to RRVVQREKR wa	38
502 858-D	gp120(C term 510-516) Donor: ?	gp120(507-513) VVQREKR	VVQREKR	Z	HIV-1 infection	human(IgG)
	 References: [Zolla-Pazner et al.(1995), Forthal et al.(1995)] NOTES: • 858-D: Group specific cross-clade binding in serotyping • 858-D: No neutralizing activity, no ADCC activity, and 	al.(1995), Forthal et oss-clade binding in ictivity, no ADCC act	ferences: [Zolla-Pazner et al.(1995), Forthal et al.(1995)] NTES: 858-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner et al. 858-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]		Zolla-Pazner et al.(1995)] orthal et al.(1995)]	
504 989-D	gp120(C term) gp120(; Donor: ? References: [Zolla-Pazner et al.(1995)]	gp120(507-513) VVQREKR al.(1995)]	VVQREKR		HIV-1 infection	human(IgG)
	NOTES: • 989-D: In serotyping study usin virus [Zolla-Pazner et al.(1995)]	(1995)] al.(1995)]	iterences: [Zolla-Pazner et al.(1995)] YTES: 989-D: In serotyping study using flow-cytometry, showed B clade specificity, but only reacted with 7/11 B clade virus [Zolla-Pazner et al.(1995)]	ty, but only rea	cted with 7/11 B clad	le
503 1131-A	gp120(C term 510-516 LAI) gp Donor: ? References: [Bandres et al.(1998)] NOTES: • 1131-A: A very high affinity are of CD4-on 120 interactions and	gp120(507-513) VVQREKR [1998)] introduction in the control of that this binding can be call as and that this binding can be call	20(C term 510-516 LAI) gp120(507-513) VVQREKR N HIV-1 infection or: ? rences: [Bandres et al.(1998)] TES: 1131-A: A very high affinity antibody used in studies that demonstrate that CXCR4 can bind to gp120 in the absence of CD4 on 120 interactions, and that this binding can be enhanced by Env decly convolution [Bandres et al.(1998)]	N XCR4 can bind	HIV-1 infection to gp120 in the absence	human(${ m Ig}{ m G}_3\lambda)$

gp120(C term)	gp120	?		Peptide from the C-term	sheep
Donor: Aalto BioReagents Ltd References: [Moore(1990), Sa Trkola et al.(1996a), Ditzel et a NOTES:	, Dublin, Irelan uttentau & Moo l.(1997), Ugolir	d re(1991), Moore et al.(1993a), M ni et al.(1997), Mondor et al.(1998	Moore et al.(19 3), Binley et al	193b), Wyatt et al.(1995 .(1998)]	ŷ,
D7324: Binding unaltered Moore(1991)] D7324: Binds to the last 1: D7324: Epitope in C5 – D competition study [Trkola of the competition of the competition study [Trkola of the competition of the competit	by gp120 binc arino acids in boes not neutrall et al.(1996a)]	ling to sCD4, in contrast to 110. gp120 – used for antigen capture ize JR-FL nor block gp120 intera	.5, 9284, 50-6 ELISA [Wyanction with CC	39 and 98-6 [Sattentau of the following of the state of the following of the follo	У
ompetition study [Trkola of the competition study] of the competition study of the competition of the competition study of the competition of the	et al.(1996a)] p120 onto solic et al.(1998)]	I phase for epitope mapping [Mo	ore et al.(199	3a), Moore et al.(1993b), (
gp120(C term) Donor: J. Robinson, Tulane Un References: [Thali et al.(1992a) NOTES:	gp120 iiversity, LA ı), Thali et al.(1)	? 993), Wu et al.(1996), Trkola et al	N l.(1996a), Fou	? ts et al.(1997)]	
3A: Called 2.3A – Did not lomain [Wu et al.(1996)]	block ability of _{	gp120-sCD4 complexes to inhibit l	MIP-1 $lpha$ bindiı	1g – binds to gp41-bindir	g
23A: C5 binding MAb – (Trkola et al.(1996a)]	does not inhibit	gp120 interaction with CCR-5 i	in a MIP-1 eta -(CCR-5 competition stud	jy
3A: Study shows neutralize ligomeric env binding – 2	zation is not pred 3A bound mone	mer, did not bind oligomer or neu	nonomeric gpl utralize JRFL	[Fouts et al.(1997)]	th
gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	Z	sCD4-(rHXB2 gp120)-complex	murine(IgG)
r: J. Robinson, Tulane Ur	niversity, LA 5)]				
	or: Aalto BioReagents Ltd erences: [Moore(1990), Sa bla et al.(1996a), Ditzel et a FES: D7324: Binding unaltered Moore(1991)] D7324: Epitope in C5 – D competition study [Trkola of D7324: Used to capture g Ditzel et al.(1997), Binley D7324: Tess: 20(C term) Or: J. Robinson, Tulane Unerences: [Thali et al.(1992a) FES: 23A: Called 2.3A – Did not domain [Wu et al.(1996)] 23A: Study shows neutraliate oligomeric env binding — 2 20(dis gp120-CD4) Or: J. Robinson, Tulane Unerences: [Devico et al.(1996)]	ponor: Aalto BioReagents Ltd, Dublin, Irelam References: [Moore(1990), Sattentau & Moor Trkola et al.(1996a), Ditzel et al.(1997), Ugolir NOTES: • D7324: Binding unaltered by gp120 bind Moore(1991)] • D7324: Binds to the last 15 amino acids in D7324: Epitope in C5 – Does not neutralic competition study [Trkola et al.(1996a)] • D7324: Used to capture gp120 onto solic Ditzel et al.(1997), Binley et al.(1998)] pp120(C term) pp120(C term) pp120(C term) pp120(C term) pp120 and not block ability of godomain [Wu et al.(1992a), Thali et al.(1998)] • 23A: Called 2.3A – Did not block ability of godomain [Wu et al.(1996a)] • 23A: Study shows neutralization is not prevoligomeric env binding — 23A bound monogp120(dis gp120-CD4) ponor: J. Robinson, Tulane University, LA References: [Devico et al.(1995)] NOTES:	gp120(C term) gp120 Ponor: Aalto BioReagents Ltd, Dublin, Ireland References: [Moore(1990), Sattentau & Moore(1991), Moore et al.(1993a), N Trkola et al.(1996a), Ditzel et al.(1997), Ugolini et al.(1997), Monore et al.(1998) NOTES: • D7324: Binding unaltered by gp120 binding to sCD4, in contrast to 110 Moore(1991)] • D7324: Binds to the last 15 amino acids in gp120 – used for antigen capture • D7324: Epitope in C5 – Does not neutralize JR-FL nor block gp120 interacompetition study [Trkola et al.(1996a)] • D7324: Used to capture gp120 onto solid phase for epitope mapping [Mo Ditzel et al.(1997), Binley et al.(1998)] gp120(C term) gp120(C term) gp120(Term) gp12	pgl120(C term) gpl20 Ponor: Aalto BioReagents Ltd, Dublin, Ireland References: [Moore(1990), Sattentau & Moore(1991), Moore et al.(1993a), Moore et al.(1997) Prkola et al.(1996a), Ditzel et al.(1997), Ugolini et al.(1997), Mondor et al.(1998), Binley et al. NOTES: • D7324: Binding unaltered by gpl20 binding to sCD4, in contrast to 110.5, 9284, 50-6 Moore(1991)] • D7324: Binds to the last 15 amino acids in gpl20 – used for antigen capture ELISA [Wya • D7324: Epitope in C5 – Does not neutralize JR-FL nor block gpl20 interaction with CC competition study [Trkola et al.(1996a)] • D7324: Used to capture gpl20 onto solid phase for epitope mapping [Moore et al.(1992) Ditzel et al.(1997), Binley et al.(1998)] pgl20(C term) References: [Thali et al.(1996a), Thali et al.(1993), Wu et al.(1996), Trkola et al.(1996a), Four NOTES: • 23A: Called 2.3A – Did not block ability of gpl20-sCD4 complexes to inhibit MIP-1α bindir domain [Wu et al.(1996a)] • 23A: Cs binding MAb – does not inhibit gpl20 interaction with CCR-5 in a MIP-1β-C [Trkola et al.(1996a)] • 23A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gpl oligomeric env binding – 23A bound monomer, did not bind oligomer or neutralize JRFL gpl20(dis gpl20-CD4) gpl20(dis) DISCONTINUOUS N Donor: J. Robinson, Tulane University, LA References: [Device et al.(1995)]	term) gp120 ? kalto BioReagents Ltd, Dublin, Ireland es: [Moore(1990), Sattentau & Moore(1991), Moore et al.(1993a), Moore et al.(1996a). Ditzel et al.(1997), Ugolini et al.(1997), Mondor et al.(1998), Binley et al. 44: Binding unaltered by gp120 binding to sCD4, in contrast to 110.5, 9284, 50-69 re(1991)] 44: Binds to the last 15 amino acids in gp120 – used for antigen capture ELISA [Wyat 24: Epitope in C5 – Does not neutralize JR-FL nor block gp120 interaction with CCI betition study [Tikola et al.(1996a)] 44: Used to capture gp120 onto solid phase for epitope mapping [Moore et al.(1993)] 45: Used to capture gp120 onto solid phase for epitope mapping [Moore et al.(1993)] 46: It al.(1997), Binley et al.(1998)] 47: Provided to capture gp120 onto solid phase for epitope mapping [Moore et al.(1993)] 48: It al.(1997), Binley et al.(1998)] 49: Called 2.3A – Did not block ability of gp120-sCD4 complexes to inhibit MIP-1α bindin in [Wu et al.(1996a)] 49: Called 2.3A – Did not block ability of gp120-sCD4 complexes to inhibit MIP-1α bindin in [Wu et al.(1996a)] 40: Stinding MAb – does not inhibit gp120 interaction with CCR-5 in a MIP-1/β-C olla et al.(1996a)] 40: Stinding MAb – does not inhibit gp120 interaction with CCR-5 in a MIP-1/β-C olla et al.(1996a)] 41: Stindy shows neutralization is not predicted by MAb binding to JRFL monomeric gp1: meric env binding – 23A bound monomer, did not bind oligomer or neutralize JRFL [40: Sp120-CD4) 41: Sp120-CD4) 42: Sp120-CD4) 43: Sp120-CD4) 44: Binding Moore et al.(1996) 45: Called 2.3A – Did not bind oligomer or neutralize JRFL [40: Sp120-CD4) 40: Sp120-CD4) 41: Sp120-CD4) 41: Sp120-CD4 42: Binding Moore et al.(1996) 42: Called 2.3A – Did not bind oligomer or neutralize JRFL [40: Sp120-CD4) 41: Sp120-CD4 42: Sp120-CD4 43: Sp120-CD4 44: Binding to JRFL [40: Sp120-CD4 45: Sp120-CD4 46: Sp120-CD4 47: Sp120-CD4 48: Sp120-CD4 49: Sp120-CD4 49: Sp120-CD4 40: Sp120-CD

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
508 8F102	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	Z	sCD4-(rHXB2 gp120)-complex	murine(IgG)
	Donor: J. Robinson, Tulane University, LA References: [Devico et al.(1995)] NOTES:	University, LA 195)]				
	 8F102: MAbs specifical dependent – competition 	ly reactive to crustudies indicate i	8F102: MAbs specifically reactive to crosslinked gp120 and CD4 were derived (8F101, 8F102) – conformation dependent – competition studies indicate the epitope is immunogenic in infected humans [Devico et al.(1995)]	re derived (8F10) infected humans	l, 8F102) – conformati [Devico et al.(1995)]	ion
509 CG-10	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	T	CD4/gp120 IIIB complex	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: Jonathan Gershoni, Tel Aviv University, Isreal References: [Gershoni et al.(1993), Wu et al.(1996), I NOTES:	el Aviv Universi 1993), Wu et al.(Donor: Jonathan Gershoni, Tel Aviv University, Isreal References: [Gershoni et al.(1993), Wu et al.(1996), Lee et al.(1997), Rizzuto et al.(1998), Sullivan et al.(1998)] NOTES:	ıto et al.(1998), Sı	ıllivan et al.(1998)]	
	 CG-10: Also called CG10 CG-10: Reacts exclusively 	0 ly with sCD4-on	CG-10: Also called CG10 (CG-10: Also called CG10) (CG-10: Reacts exclusively with sCD4-on 120 complex not with sCD4 or on 120 alone [Gershoni et al. (1993)]	or on 120 alone [G	ershoni et al (1993)]	
	 CG-10: Called CG10 – MIP-1α binding to CCR does not block this inhibition (Wn et al. (1996)) 	$ ext{IIP-}1lpha$ binding to	CG-10: Called CG10 – MIP-1 α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4, and MAb CG10 does not block this inhibition (Wn et al. (1996))	e inhibited by gp1	20-sCD4, and MAb CG	10
	 CG-10: Called CG10 – F CD4+ lines 	romotes envelop	CG-10: Called CG10 – Promotes envelope mediated fusion between both T-cell and macrophage tropic viruses and CD4+ lines	th T-cell and macı	ophage tropic viruses	md
	• CG-10: Called CG10 – obridging sheet of gp120 –	hisrupts gp120-C mutations in pos	CG-10: Called CG10 – disrupts gp120-CCR5 interaction and competes with MAb 17b –binds near the conserved bridging sheet of gp120 – mutations in positions K/D 121, T/D 123, K/D 207, K/D 421, Q/L 422, Y/S 435, M/A 434,	s with MAb 17b - 207, K/D 421, Q/	-binds near the conserv L 422, Y/S 435, M/A 4:	/ed 34,
	• CG-10: Called CG10 - (CD4BS MAb 15	CG-10: Called CG10 – CD4BS MAb 15e competes with CG-10 binding, probably due to the disruption of CD4-	ng, probably due	to the disruption of CI)4-
	gp120 by 15e – CD4i MA 17b, 48d and CG10 – MA	bs 17b and 48d c abs C11, 2G12 and	gp120 by 15e – CD4i MAbs 17b and 48d compete and the binding sites may overlap – MAb A32 enhances binding of 17b, 48d and CG10 – MAbs C11, 2G12 and 212A do not affect CG10 binding – CG-10 can bind gp120 with V1/V2	nay overlap – MAI inding – CG-10 ca	n bind gp120 with V1/	of V2
	HXBc2 mutations \triangle 298 CG10 epitope maps to th	-327 (V3), 384 Ne CD4 CDR2-lik	HXBc2 mutations \triangle 298-327 (V3), 384 Y/E, 298 R/G, 435 Y/S enhance recognition – the CD4 contribution to the CG10 epitope maps to the CD4 CDR2-like loop – CG10 can neutralize HIV-1 in the presence of sCD4 even though	the recognition – the HIV-1 in the press	e CD4 contribution to ence of sCD4 even thou	the lgh
	it does not do so in the co	ntext of cell surf	it does not do so in the context of cell surface CD4 binding to gp120 [Sullivan et al.(1998)]	ıllivan et al.(1998)]	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
510 CG-4	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	Z	CD4/gp120 complex	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: Jonathan Gershoni, Tel Aviv University, Isreal References: [Gershoni et al.(1993)] NOTES:	l Aviv Universit 993)]	y, Isreal			
	• CG-4: Reacts with gp120: • CG-4: Called CG4	and sCD4-gp12	CG-4: Reacts with gp120 and sCD4-gp120 complex, not with sCD4 [Gershoni et al.(1993)] CG-4: Called CG4	rshoni et al.(199	(3)]	
511 CG-9	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	T	CD4/gp120 complex	$murine(IgG_1)$
	References: [Gershoni et al.(1993)] NOTES: • CG-9: Reacts preferentially wit	993)] y with sCD4-gp	ferences: [Gershoni et al.(1993)] VIES: CG-9: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 [Gershoni et al.(1993)]	gp120 [Gershor	i et al.(1993)]	
512 CG-25	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	T	CD4/gp120 complex	$\operatorname{murine}(\operatorname{IgG}_1)$
	References: [Gershoni et al.(1993)] NOTES: • CG-25: Reacts preferentially w	993)] lly with sCD4-g	ferences: [Gershoni et al.(1993)] TES: CG-25: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 [Gershoni et al.(1993)]	h gp120 [Gersho	ni et al.(1993)]	
513 CG-76	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	Т	CD4/gp120 complex	$murine(IgG_1)$
	References: [Gershoni et al.(1993)] NOTES: • CG-76: Reacts equally well wit	993)] II with sCD4-gp	 eferences: [Gershoni et al.(1993)] OTES: CG-76: Reacts equally well with sCD4-gp120 and sCD4, but not with purified gp120 		[Gershoni et al.(1993)]	
514 ID6	gp120(1-193 BH10)	gp120	UNDEFINED AMINO TERMINUS	T	?	$murine(IgG_1)$
	References: [Ugen et al.(1993), Cook et al.(1994)] NOTES:), Cook et al.(19	94)]			
	 ID6: MAbs against the glycosphingolipid GalCer block HIV inferom the brain and colon – MAbs against the N-terminal half of binding of GalCer to gp120 does not inhibit MAb binding [Cook of ID6: NIH AIDS Research and Reference Reagent Program: 2343 	ycosphingolipid - MAbs against 0 does not inhib and Reference I	ID6: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)] ID6: NIH AIDS Research and Reference Reagent Program: 2343	of normally susce do not inhibit g	eptible CD4 negative copp120 binding to GalCe	ells

Do Re	517 MAG 45 gp	516 522-149 gp Do Re N (515 AD3 gp	MAb ID Lo
 Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994), Moore & Sodroski(1996), Wyatt et al.(1997)] NOTES: MAG 45: Also called #45 MAG 45: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)] MAG 45: Reciprocal binding inhibition with anti-C1-C5 and anti-C1-C4 discontinuous MAbs – binding enhanced by anti-V3 5G11 – inhibits binding of anti-CD4 binding site MAbs [Moore & Sodroski(1996)] MAG 45: Called #45 – binds to efficiently sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 	gp120(C1 dis)	gp120(C1 dis) gp120(dis) Donor: G. Robey, Abbott Inc. References: [Moore & Sodroski(1996), Trkola et al.(1996) NOTES: • 522-149: Binding is enhanced by C5 antibodies M9: 133/290 – binding is destroyed by a W/L (position 6 enhance binding to gp120 [Moore & Sodroski(1996)] • 522-149: Does not neutralize JR-FL nor block gp120 [Trkola et al.(1996a)] • 522-149: A panel of MAbs were shown to bind with a structure closely approximating full length folded m	gp120(1-193 BH10) References: [Ugen et al.(1993), Cook et al.(1994)] NOTES: AD3: MAbs against the glycosphingolipid GalCer block HIV inferom the brain and colon – MAbs against the N-terminal half of gbinding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1993)]	Location
), Moore & Sodi	gp120(dis)	gp120(dis) ki(1996), Trkola nced by C5 anti coyed by a W/L [Moore & Sodro ize JR-FL nor bloop deleted coo nating full leng	gp120), Cook et al.(19 lycosphingolipic – MAbs against 0 does not inhib n and Reference	WEAU
 Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994), Moore & Sodroski(1996), Wyatt et al.(1997)] NOTES: MAG 45: Also called #45 MAG 45: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)] MAG 45: Reciprocal binding inhibition with anti-C1-C5 and anti-C1-C4 discontinuous MAbs – binding enhanced by anti-V3 5G11 – inhibits binding of anti-CD4 binding site MAbs [Moore & Sodroski(1996)] MAG 45: Called #45 – binds to efficiently sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is 	DISCONTINUOUS	 gp120(C1 dis) gp120(dis) DISCONTINUOUS N Env glycopro Donor: G. Robey, Abbott Inc. References: [Moore & Sodroski(1996), Trkola et al.(1996a), Binley et al.(1998)] NOTES: 522-149: Binding is enhanced by C5 antibodies M91 and 1C1 – mutual binding-inhibition with anti-C1 antibodies enhance binding to gp120 [Moore & Sodroski(1996)] 522-149: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)] 522-149: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al.(1998)] 	20(1-193 BH10) gp120 UNDEFINED AMINO L ? TERMINUS rences: [Ugen et al.(1993), Cook et al.(1994)] TES: AD3: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)] AD3: NIH AIDS Research and Reference Reagent Program: 2342	Sequence
)] 8 N/P – does no [Kang et al.(1994 discontinuous] re & Sodroski(1 120+gp41, sugger- term amino acid	Z	N 98)] I binding-inhibit ino acid substitutino acid substitutino acid substitutino acid substitutino acid substitutino acid substitutino acid substitutional.	L of normally susc do not inhibit g	Neutralizing
t bind to C1 region 20 n 94)] MAbs – binding enhanc 996)] sting its gp120 epitope	sCD4-(rHXB2 gp120)-complex	Env glycopro tion with anti-C1 antibot tion – other C1 antibod cCCR-5 competition stu ar competition profiles te ch a core protein produc	? susceptible CD4 negative cells bit gp120 binding to GalCer –	Immunogen
ner xed t is	murine	ies ies idy o a	murine(IgG ₁) ells	Species(Isotype)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
518 MAG 95	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	Z	sCD4-(rHXB2 gp120)-complex	murine
	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)] NOTES: • MAG 95: Only observed an	mino acid subs	nor: C. Y. Kang, IDEC Inc ferences: [Kang et al.(1994)] TES: MAG 95: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer	88 N/P – does no	at bind to C1 region 20 i	mer
519 MAG 97	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	Z	sCD4-(rHXB2 gp120)-complex	murine
	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)] NOTES:	<u> </u>				
	 MAG 97: Only observed a peptides, tentative classific 	mino acid subs ation conforma	MAG 97: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)]	88 N/P – does no b [Kang et al.(19	s not bind to C1 region 20 i .(1994)]	mer
520 MAG 104	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	Z	sCD4-(rHXB2 gp120)-complex	murine
	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)]	<u> </u>				

Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese et al.(1992), Devico et al.(1995), Moore & Sodroski(1996), I et al.(1997), Binley et al.(1998)] NOTES: • M90: Reactive only with native gp120, so binds to a discontinuous epitope – reacts with n Veronese et al.(1992)] • M90: Reactive only with both non-reduced (but not denatured) covalently cross-linked gp120 et al.(1995)] • M90: Reciprocal inhibition of binding of other anti-C1 MAbs – inhibits CD4 binding site N of V2 MAbs G3-4 and SC258 [Moore & Sodroski(1996)] • M90: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epi binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with deleted [Wyatt et al.(1997)] • M90: A panel of MAbs were shown to bind with similar or greater affinity and similar deglycosylated or variable loop deleted core gp120 protein (\(\Delta \) V1, V2, and V3), thus sucl a structure closely approximating full length folded monomer [Binley et al.(1998)] p120(C1 dis HXBc2) gp120(dis) DISCONTINUOUS Donor: Fulvia di Marzo Veronese References: [Ditzel et al.(1997), Parren et al.(1997b)] NOTES: • p7: gp120 immobilized on solid phase by capture with sCD4 was used for selection of Fi Fabs were obtained that bind to similar epitopes, p7, p20, and p35 – a C1 W/S substitution binding, a Y/D at position 45 reduced binding, and C5 region substitutions 475 M/S and 49 – compete with MAbs M85, M90 and 212A, but not M91 and G3-299 [Ditzel et al.(1997)] • p7: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)] pg120(C1 dis HXBc2) gp120(dis) DISCONTINUOUS Donor: Fulvia di Marzo Veronese References: [Ditzel et al.(1997)] NOTES:		Location en120/C1 dis	WEAU on 120 (dis)	Sequence	N V V V V V V V V V V V V V V V V V V V	451 Fnv
p7 gp12 Don Refe NO1 P012 P12 P13 P14 P15 P15 P16	521 M90	gp120(C1 dis) Donor: Fulvia di Marzo Veron References: [di Marzo Verone. et al.(1997), Binley et al.(1998) NOTES: • M90: Reactive only with n Veronese et al.(1992)]	gp120(dis) ese se et al.(1992)] lative gp120, s	DISCONTINUOUS , Devico et al.(1995), Moore of the continuous of t	% Sodroski(1996) tope – reacts with	451 Env Ditzel et al. multiple stra
p7 gp12 Don Reft NO1 L19 gp12 pp12 NO2		 M90: Reacted with both et al.(1995)] M90: Reciprocal inhibition of V2 MAbs G3-4 and SC3 M90: Binds efficiently to binding – does not bind to deleted [Wyatt et al.(1997)] 	non-reduced (nof binding of 258 [Moore & sgp120 but not with the sgp120]	but not denatured) covalently other anti-C1 MAbs – inhibits Sodroski(1996)] ot soluble gp120+gp41, sugge of the 19 C-term amino acids	cross-linked gpl CD4 binding site CD4 binding site esting its gp120 e in conjunction w	20-CD4 cor MAbs – enl sitope is blo th C1 positi
p7 gp12 Don Refe NO1 119 2p12 100 100 Refe NO1		 M90: A panel of MAbs w deglycosylated or variable a structure closely approxii 	vere shown to loop deleted c mating full len	bind with similar or greater a core gp120 protein (Δ V1, V2 gth folded monomer [Binley of	affinity and simila	
L19 gp12 Don Refe	122 p7	gp120(C1 dis HXBc2) Donor: Fulvia di Marzo Veron References: [Ditzel et al.(1997 NOTES:	gp120(dis) ese '), Parren et al.	DISCONTINUOUS	et al.(1998)]	ch a core prote
L19 gp120(C1 dis HXBc2) gp120(dis) DISCONTINUOUS Donor: Fulvia di Marzo Veronese References: [Ditzel et al.(1997)] NOTES:		 p7: gp120 immobilized on Fabs were obtained that bit binding, a Y/D at position – compete with MAbs M8: p7: Does not neutralize TC 	n solid phase b nd to similar e 45 reduced bir 5, M90 and 21 LA strains or	.(1997b)]	et al.(1998)]	ch a core protein HIV infection
 L19: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for the selection of Fabs 	23 L19	gp120(C1 dis HXBc2) Donor: Fulvia di Marzo Veron		.(1997b)] y capture with sCD4 was use pitopes, p7, p20, and p35 – a anding, and C5 region substitution 2A, but not M91 and G3-299 primary isolates [Parren et al.	ext al.(1998)] It al.(1998)] It al.(1998)] It d for selection of C1 W/S substitutions 475 M/S and [Ditzel et al.(1997b)]	HIV infection

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
524 L100	gp120(C1-C2 dis HXBc2) gp120(dis) DISCONTINUOUS Donor: Fulvia di Marzo Veronese References: [Ditzel et al.(1997), Parren et al.(1997b), Parren & Burton(1997)] NOTES:	gp120(dis) ese), Parren et al.(DISCONTINUOUS 1997b), Parren & Burton(199	7)]	HIV infection	human $\operatorname{Fab}(\operatorname{IgG}_1)$
	 L100: Does not neutralize L100: gp120 immobilized a new Fab, L100, with a nebinding, and C2 substituti inhibits binding of MAbs N 	TCLA strains o on solid phase ovel specificity f ons 252 R/W, 2: 190 and G3-299,	L100: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)] L100: gp120 immobilized on solid phase by capture with sCD4 and then masked with Fab p7 allowed selection of a new Fab, L100, with a novel specificity for C1 and C2 – gp120 C1 substitutions 69 W/L and 76 P/Y abolish L100 binding, and C2 substitutions 252 R/W, 256 S/Y, 262 N/T and 267 E/L abolish or strongly inhibit L100 binding – inhibits binding of MAbs M90 and G3-299, but not M85, 212A, and M91 [Ditzel et al.(1997), Parren & Burton(1997)]	d.(1997b)] En masked with F satitutions 69 W/I abolish or strong [Ditzel et al.(1997)]	ab p7 allowed selectic , and 76 P/Y abolish I gly inhibit L100 bindi 7), Parren & Burton(19	on of 100 ng – 97)]
525 L17	gp120(V2 dis) gp120(dis) DISC Donor: Fulvia di Marzo Veronese References: [Ditzel et al.(1997), Parren et al.(1998)] NOTES:	gp120(dis) ese), Parren et al.(DISCONTINUOUS			human Fab
	• L17: The rank order of F. b14 > b13 > DO142-10 > form (3B3 > b12 > DO12 oligomeric form and neutr determined by the fraction	Ab binding affin DA48 > L17) \(\) DA48 > L00p 2 2-10 > L00p 2 alization were c of Ab sites occu	L17: The rank order of FAb binding affinity to monomeric gp120 (Loop $2>3B3>b12=D08i>b11>b3>b14>b13>D0142-10>DA48>L17)$ was markedly different than FAb binding affinity to the mature oligomeric form (3B3>b12>D0142-10>Loop $2>b11>L17>b6>D08i>b14>DA48>b3>b13)$ and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs—authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	op 2 > 3B3 > b. Ab binding affinit binding aff	> b12 = D08i > b11 > b3 > ffinity to the mature oligomeric 48 > b3 > b13) and binding to rs suggest that neutralization is e [Parren et al.(1998)]	33 > neric neric yn is
526 684-238	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L	IIIB gp120 from infected cells	murine
	Donor: Gerry Robey, Abbott Laboratories References: [Moore et al.(1993a), Thali et al.(1993), Gorny et al.(1994), Ditzel et al.(1995), Moore & Sodroski(1996), Ditzel et al.(1997)] NOTES:	aboratories 3a), Thali et al.((1993), Gorny et al.(1994), Di	itzel et al.(1995),	Moore & Sodroski(19	96),
	 684-238: Also called 52-684-238 and 52-684 684-238: Specific for BH10 or HXB2, does not bind to N inhibited by deletion of the V2 loop, and the following ami 183/184PI/SG, and 192-194YSL/GSS [Moore et al.(1993a)] 	84-238 and 52-6 10 or HXB2, d e V2 loop, and 4YSL/GSS [Mo	684-238: Also called 52-684-238 and 52-684 684-238: Specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – neutralizes BH10 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177FY/AT, 179/180LD/DL, 183/184PI/SG, and 192-194YSL/GSS [Moore et al.(1993a)]	7	neutralizes BH10 – binding 5/177FY/AT, 179/180LD/DL,	ding /DL,
	 684-238: Does not compete with IgG1b12, reciprocal inhibition with 684-238: Limited reciprocal enhancement of binding with anti-V3 a with V2 region antibodies [Moore & Sodroski(1996)] 	e with IgG1b12 al enhancement [Moore & Sodre	684-238: Does not compete with IgG1b12, reciprocal inhibition with MAbs L39, L40, and L78 [Ditzel et al.(1995)] 684-238: Limited reciprocal enhancement of binding with anti-V3 and C4 region antibodies – reciprocal inhibition with V2 region antibodies [Moore & Sodroski(1996)]	Abs L39, L40, an C4 region antiboo	d L78 [Ditzel et al.(19 lies – reciprocal inhib	95)] ition

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
527 CRA-3	gp120(V2 dis) gp120(dis) DISCONTINUOUS N Donor: Mark Page, NIBSC AIDS reagent project, Potters Bar, Herts, UK References: [Moore & Ho(1993), Moore et al.(1993a), Thali et al.(1993), Shotton et al.(1 Ditzel et al.(1997)] NOTES:	gp120(dis) DS reagent proj 3), Moore et al.(DISCONTINUOUS ect, Potters Bar, Herts, UK 1993a), Thali et al.(1993), Sho	N tton et al.(1995),	rBH10 gp120 995), Moore & Sodroski(1996),	murine($\lg G_{2a}$) 996),
	 CRA-3: Conformational, does not bind well to denatured gp120 [Percentage of the CRA-3: specific for BH10 or HXB2, does not bind to MN, RF, or V2 loop, and the following amino acid substitutions: 176/177 FY// YSL/GSS – epitope probably involves stem of V1/V2 loop structute. CRA-3: Many MAbs enhance binding, including some anti-C5, Ca a small number of anti-V3 loop MAbs [Moore & Sodroski(1996)] CRA-3: Called CRA3 – Same competition group as CRA6 [Shotto CRA-3: UK Medical Research Council AIDS reagent: ARP324 	oes not bind we or HXB2, does amino acid subs ly involves stennee binding, incloop MAbs [Mc me competition me competition arch Council Al	CRA-3: Conformational, does not bind well to denatured gp120 [Moore & Ho(1993)] CRA-3: specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS – epitope probably involves stem of V1/V2 loop structure [Moore et al.(1993a)] CRA-3: Many MAbs enhance binding, including some anti-C5, C1, V4, and C4 MAbs – enhances binding of only a small number of anti-V3 loop MAbs [Moore & Sodroski(1996)] CRA-3: Called CRA3 – Same competition group as CRA6 [Shotton et al.(1995)] CRA-3: UK Medical Research Council AIDS reagent: ARP324	& Ho(1993)] p120 – binding ii p120 – binding ii)/180 LD/DL, 18 vore et al.(1993a) and C4 MAbs – l.(1995)]	nhibited by deletion c 3/184 PI/SG, and 192] enhances binding of	only
528 CRA-6	gp120(V1V2 dis) gp120(dis) DISCONTINUOUS Donor: Mark Page, NIBSC AIDS reagent project, Potters Bar, Herts, UK References: [Shotton et al.(1995)] NOTES: • CRA-6: Called CRA6 – same competition group as CRA-3 [Shotton	gp120(dis) DS reagent proj (5)]	20(V1V2 dis) gp120(dis) DISCONTINUOUS Nor: Mark Page, NIBSC AIDS reagent project, Potters Bar, Herts, UK erences: [Shotton et al.(1995)] TES: CRA-6: Called CRA6 – same competition group as CRA-3 [Shotton et al.(1995)]	N 1.(1995)]	?	murine
529 CRA-4	gp120(V2 dis) gp120(dis) dp120(dis) DISCONTINUOUS L (HXB2) rBH10 gp120 Donor: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325 References: [McKeating et al.(1993b), Moore & Ho(1993), Moore et al.(1993a), Thali et al.(1993), Shotton et al.(1995), Moore & Sodroski(1996)] NOTES: • CRA-4: Also called CRA4 • CRA-4: Changes at residues 191/192/193 (YSL/GSS) within V2, 435 (Y/H) in C4, abrogate binding – type-specific neutralization [McKeating et al.(1993b)] • CRA-4: Conformational, does not bind well to denatured gp120 [Moore & Ho(1993)] • CRA-4: Specific for BH10 and HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)] • CRA-4: Cross-competes with MAbs 11/68b, 62c, 66c, 66a – similar to 66c and 66a – non-reciprocal inhibition by MAbs 12b, 60b and CRA-6 [Shotton et al.(1995)] • CRA-4: The only MAbs (Moore & Sodroski(1996)] • CRA-4: UK Medical Research Council AIDS reagent: ARP325	gp120(dis) C AIDS reagen 1993b), Moore. 1993b), Moore. 1993b), Moore. 1993b)] oes not bind we and HXB2, doe amino acid subs 93a)] ith MAbs 11/68 ith MAbs 11/68 if [Shotton et al.) if [Shotton et al.) if [Moore & Sod arch Council Al	pp120(dis) gp120(dis) DISCONTINUOUS L (HXB2) rBH10 gp120 or: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325 rences: [McKeating et al.(1993b), Moore & Ho(1993), Moore et al.(1993a), Thali et al.(1993), Shotton et al.(1995), re & Sodroski(1996)] IES: CRA-4: Also called CRA4 CRA-4: Changes at residues 191/192/193 (YSL/GSS) within V2, 435 (Y/H) in C4, abrogate binding – type-specific neutralization [McKeating et al.(1993b)] CRA-4: Conformational, does not bind well to denatured gp120 [Moore & Ho(1993)] CRA-4: Specific for BH10 and HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)] CRA-4: Cross-competes with MAbs 11/68b, 62c, 66c, 66a – similar to 66c and 66a – non-reciprocal inhibition by MAbs 12b, 60b and CRA-6 [Shotton et al.(1995)] CRA-4: The only MAbs that enhanced binding were anti-V3 MAb 5G11 and anti-C1 MAb 135/9 binding – reciprocal inhibition of anti-V2 MAbs [Moore & Sodroski(1996)] CRA-4: UK Medical Research Council AIDS reagent: ARP325	L (HXB2) 3a), Thali et al.(19 4h) in C4, abrogg 4k Ho(1993)] 4gp120 – binding i 1/180 LD/DL, 18 56c and 66a – no 16c and 66a – no 17d anti-C1 MAb	rBH10 gp120 al.(1993), Shotton et al.(1995), al.(1993), Shotton et	murine(IgG ₁) 995), ecific ecific y-194 n by rocal

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
530 66a	gp120(V2 dis) Donor: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325 References: [Shotton et al.(1995)] NOTES:	gp120(dis) C AIDS reager)5)]	DISCONTINUOUS at repository, ARP 325	, ,,,	rBH10 gp120	$\operatorname{murine}(\operatorname{Ig} \operatorname{G}_1)$
	 66a: Substitutions 176-177 FY/AT, 179-180 LD/DL, 183-18 same competition group as CRA4 [Shotton et al.(1995)] 66a: UK Medical Research Council AIDS reagent: ARP3074 	7 FY/AT, 179-: CRA4 [Shotto Council AIDS	66a: Substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184 PI/SG, and 191-193 same competition group as CRA4 [Shotton et al.(1995)] 66a: UK Medical Research Council AIDS reagent: ARP3074	35	YSL/GSS abrogate binding –	ıding –
531 66c	gp120(V2 dis) Donor: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325 References: [Shotton et al.(1995)] NOTES: • 66c: Substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184	gp120(dis) C AIDS reager (5)]	20(V2 dis) gp120(dis) DISCONTINUOUS L (HXB2 ior: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325 erences: [Shotton et al.(1995)] TES: 66c: Substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184 PI/SG, and 191-193	(19	rBH10 gp120 YSL/GSS abrogate binding -	$\operatorname{murine}(\operatorname{IgG}_1)$ Iding –
	same competition group as CRA4 [Shotton et al.(1995)]	CRA4 [Shotto	n et al.(1995)]			
532 11/68b	gp120(V1V2 dis) gp120(dis) DISCONTIN Donor: Shotton and Dean References: [McKeating et al.(1993b), Shotton et al.(1995)] NOTES:	gp120(dis) 1993b), Shotto	DISCONTINUOUS n et al.(1995)]	L (HXB2)	rBH10 gp120	$\operatorname{rat}(\operatorname{IgG}_1)$
	 11/68b: Changes at residue 11/68b: 435 (Y/H) in C4 d 11/68b: Cross-competes w 	s 183/184 (PI/S oes not abrogat ith MAbs 62c,	11/68b: Changes at residues 183/184 (PI/SG) within V2, 435 (Y/H) in C4, abrogate binding [McKeating et al.(1993b)] 11/68b: 435 (Y/H) in C4 does not abrogate binding (John Moore, per comm, 1996) 11/68b: Cross-competes with MAbs 62c, 66c, 66a, and CRA-4 – similar to MAb 62c – HXB2 neutralization escape	, abrogate bindin omm, 1996) r to MAb 62c – I	nding [McKeating et al.(1993b)] c – HXB2 neutralization escape	993b)] escape
	et al.(1995)] • 11/68b: UK Medical Research Council AIDS reagent: ARP3041	ion at residue 1 arch Council A	mutant had a D/N substitution at residue 185 – non-reciprocal inhibition of binding et al.(1995)] 11/68b: UK Medical Research Council AIDS reagent: ARP3041	of binding of C	of CRA-3 and CRA-6 [Shotton	Shotton
533 62c	gp120(V1V2 dis) Donor: Shotton and Dean References: [Shotton et al.(1995)]	gp120(dis) 95)]	DISCONTINUOUS	Z	rBH10 on120	$\mathrm{rat}(\mathrm{IgG}_1)$
	• 62c: Cross-competes with MAbs 11/68b, 66c, 66a, and CRA-4 – same cross-competition group as MAb 11/68b – non-reciprocal inhibition of binding of CRA-3 and CRA-6 – substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184				191110 Borne	

		536 L15			535 110-B						534 SC258	MAb ID
• L15: Does not neutra	NOTES: • L15: gp120 immobili anti-V2 Fabs were obrodent anti-V2 MAbs	gp120(V2 dis) gp120(dis) DISC Donor: Hybridolabs, Institute Pasteur, Paris, France References: [Ditzel et al.(1997), Parren et al.(1997b)]	• 110-B: specific for BH10, does and the following amino acid sub YSL/GSS [Moore et al.(1993a)]	Donor: Hybridolabs, Institute Pasteur, Paris, France References: [Moore et al.(1993a)] NOTES:	gp120(V2 dis)	droski(1996)] • SC258: Does not inhibit gp120 neutralizing [Trkola et al.(1996a)]	 SC258: Very poor reactivity with gp120 molec SC258: Does not compete with IgG1b12 – reci SC258: Several MAbs binding to various gp12 binding of was anti-CD4 binding site MAb F9 	 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)] SC258: HIV-1 RF V2 substitutions 177 Y/H and 17 	 SC258: Also called : by deletion of the V2 	Donor: Gerry Robey, Abbott Laboratories References: [Moore et al.(1993a), Thali e Ditzel et al.(1995), Moore & Sodroski(199 NOTES:	gp120(V2 dis)	Location
lize I CLA strains b	zed on solid phase lained with very sim SC258, CRA3, G3-	gp120(dis) itute Pasteur, Paris, (1997), Parren et al.	H10, does not bind no acid substitution al.(1993a)]	itute Pasteur, Paris, .(1993a)]	gp120(dis)	hibit gp120 interac	netivity with gp120 apete with IgG1b12 s binding to various TD4 binding site M	ZSL/GSS [Moore et 2 substitutions 177	52-581-SC258 – bir loop, and the follow	oott Laboratories .(1993a), Thali et al & Sodroski(1996),	gp120(dis)	WEAU
L15: Does not neutralize ICLA strains but neutralizes some primary isolates weakly [Fairen et al.(199/b]]	IES: L15: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – 2 anti-V2 Fabs were obtained with very similar epitopes, L15 and L17 – deletions in V1 and V2 abolished binding, and rodent anti-V2 MAbs SC258, CRA3, G3-G4,G3-136, BAT-085, and 52-684 all compete with L15 [Ditzel et al. (1997)]	DISCONTINUOUS France (1997b)]	110-B: specific for BH10, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 168 K/L, 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)]	France	DISCONTINUOUS	droski(1996)] SC258: Does not inhibit gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study – listed as not neutralizing [Trkola et al.(1996a)]	SC258: Very poor reactivity with gp120 molecules outside of clade B [Moore et al.(1994b)] SC258: Does not compete with IgG1b12 – reciprocal inhibition with MAbs L39, L40, and L78 [Ditzel et al.(1995)] SC258: Several MAbs binding to various gp120 epitopes enhance binding, but the only MAb that SC258 enhanced binding of was anti-CD4 binding site MAb F91 – reciprocal inhibition with V2 region antibodies [Moore & So-	PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)] SC258: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity – 177 Y/H inhibits	SC258: Also called 52-581-SC258 – binds to BH10, MN, and RF gp120 – neutralizes BH10 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184	Donor: Gerry Robey, Abbott Laboratories References: [Moore et al.(1993a), Thali et al.(1993), Gorny et al.(1994), Yoshiyama et Ditzel et al.(1995), Moore & Sodroski(1996), Trkola et al.(1996a), Ditzel et al.(1997)] NOTES:	DISCONTINUOUS	Sequence
Solates weakly [Pa	MAb L72 was uso deletions in V1 and 684 all compete w	P (weak)	binding inhibited l		Z	1β -CCR-5 compet	[Moore et al.(1994) MAbs L39, L40, ar ding, but the only on with V2 region	oop of RF reduce	p120 – neutralizes s: 176/177 FY/AT	23	L	Neutralizing
uren et al.(199/b)]	s used for selection of Fabs – 2 and V2 abolished binding, and the with L15 [Ditzel et al.(1997)]	HIV infection	by deletion of the V2 83/184 PI/SG, and 192		BRU infected cell lysates	ition study – listed a	4b)] nd L78 [Ditzel et al.(1 MAb that SC258 enha natibodies [Moore δ	affinity – 177 Y/H in	BH10 – binding inhi , 179/180 LD/DL, 183	al.(1994), Moore et al.(1994b),	IIIB gp120 from infected cells	Immunogen
	os – 2 3, and 997)]	$\mathrm{human}(\mathrm{Ig} \mathrm{G}_1)$	loop, 2-194		murine	s not	995)] anced & So-	hibits	ibited 3/184	94b),	murine	Species(Isotype)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
537 L39	gp120(V2-CD4BS dis) gp120(dis) DIS Donor: Hybridolabs, Institute Pasteur, Paris, France References: [Ditzel et al.(1995)] NOTES: • L39: This Fab does not inhibit sCD4 binding, bu competed by anti-V2 MAbs, and sensitive to am for L39 and L78 gp120 amino acid substitution MAbs, but is sensitive to amino acid changes a reciprocal inhibition with V2 MAbs SC258 and (Ditzel et al.(1995)]	gp120(dis) asteur, Paris, F)] bit sCD4 bindir bit sCD4 bindir ino acid substiro acid substiro acid char 2 MAbs SC258	20(V2-CD4BS dis) gp120(dis) DISCONTINUOUS N HIV-1 infection nor: Hybridolabs, Institute Pasteur, Paris, France ferences: [Ditzel et al.(1995)] TES: L39: This Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L39 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684-238 – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]	N bably due to con the V3 loop (sim thinding) – does binding unaffaction that the control of the	HIV-1 infection to conformational changes – it is (similar patterns were observed does not compete with CD4BS maffected by deglycosylation – the region sequence is available	$\begin{array}{c} \text{human}(\text{Ig}G_{1\kappa}) \\ \text{is} \\ \text{ed} \\ \text{3S} \\ \text{ole} \end{array}$
538 L40	gp120(V2-CD4BS dis) gp120(dis) DIS Donor: Hybridolabs, Institute Pasteur, Paris, France References: [Ditzel et al.(1995)] NOTES: • L40: This Fab does not inhibit sCD4 binding, – it is competed by anti-V2 MAbs, and sensitiv observed for L40 and L78 gp120 amino acid sul CD4BS MAbs, but is sensitive to amino acid cheglycosylation – reciprocal inhibition with V2 sequence is available [Ditzel et al.(1995)]	gp120(dis) Pasteur, Paris, F hibit sCD4 bin MAbs, and se gp120 amino a tive to amino a di inhibition wi et al.(1995)]	20(V2-CD4BS dis) gp120(dis) DISCONTINUOUS N HIV-1 infection nor: Hybridolabs, Institute Pasteur, Paris, France erences: [Ditzel et al.(1995)] TES: L40: This Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L40 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding only partially affected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684-238 – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]	N probably due ons in the V3 leducing binding d 370 – binding 38 – heavy and	HIV-1 infection due to conformational changes V3 loop (similar patterns were nding) – does not compete with nding only partially affected by and light chain variable region	$\begin{array}{c} \text{human}(\operatorname{IgG}_{1\kappa}) \\ \\ \text{es} \\ \\ \text{es} \\ \\ \text{th} \\ \\ \text{by} \\ \\ \text{on} \end{array}$
539 L78	gp120(V2-CD4BS dis) gp120(dis) DIS Donor: Hybridolabs, Institute Pasteur, Paris, France References: [Ditzel et al.(1995)] NOTES: • L78: Substitutions at V2: (152/153 GE/SM, 1: (257 T/R, 368 D/R, 370 E/R) inhibit binding, a not inhibit sCD4 binding, but is inhibited by stanti-V2 MAbs, and sensitive to amino acid subs is sensitive to amino acid changes at positions 3 deglycosylation – reciprocal inhibition with V2 sequence is available [Ditzel et al.(1995)]	gp120(dis) Pasteur, Paris, F (152/153 GE/S R) inhibit bind but is inhibited but is inhibited te to amino acid hanges at posit d inhibition wi	20(V2-CD4BS dis) gp120(dis) gp120(dis) DISCONTINUOUS L HIV-1 infection 1 nor: Hybridolabs, Institute Pasteur, Paris, France erences: [Ditzel et al.(1995)] TES: L78: Substitutions at V2: (152/153 GE/SM, 183/184 PI/SG, 191/193 YL/GS), 262 N/T, V3 (314 G/W), CD4BS (257 T/R, 368 D/R, 370 E/R) inhibit binding, and some C4 and C5 substitutions enhance binding – this Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – Fab neutralizes MN and LAI – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684-238 – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]	L L/GS), 262 N/ titutions enhan formational ch does not compe izes MN and L. 38 – heavy and	HIV-1 infection 2 N/T, V3 (314 G/W), CD4BS ahance binding – this Fab does al changes – it is competed by impete with CD4BS MAbs, but ind LAI – binding unaffected by and light chain variable region	human($\operatorname{IgG}_{1\kappa}$) 8S es oy ut on

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
540 L25	gp120(V2-CD4BS dis) gp120(dis) DISCONTINUOUS Donor: Hybridolabs, Institute Pasteur, Paris, France References: [Ditzel et al.(1995), Ditzel et al.(1997), Parren et al.(1997b)] NOTES:	gp120(dis) Pasteur, Paris, F 5), Ditzel et al.(DISCONTINUOUS France 1997), Parren et al.(1997b)]	L (weak)	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_1)$
	 L25: gp120 immobilized a single anti-V2-CD4 BS rodent anti-V2 MAb SC25 L25: Neutralizes TCLA st 	on solid phase I Fab was obtaind 88 competes wit rains weakly, bu	L25: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – a single anti-V2-CD4 BS Fab was obtained with with sensitivity to substitutions in the V2 and CD4 BS regions – rodent anti-V2 MAb SC258 competes with L25 [Ditzel et al.(1997)] L25: Neutralizes TCLA strains weakly, but not primary isolates [Parren et al.(1997b)]	MAb L72 was us stitutions in the et al.(1997b)]	ed for selection of Fai	bs – ns –
541 C11	gp120(C1-C5 dis) gp120(dis) Donor: J. Robinson, Tulane University, LA	gp120(dis) niversity, LA	DISCONTINUOUS	Z	HIV-1 infection	human
	References: [Robinson et al.(1992), Moore et al.(1994d), Moore & Sodroski(1996), Trkola et al.(1996a), Wu et al.(1996), Binley et al.(1997), Fouts et al.(1997), Wyatt et al.(1997), Parren et al.(1997b), Sullivan et al.(1998)] NOTES:	992), Moore et a .(1997), Wyatt e	al.(1994d), Moore & Sodroski() t al.(1997), Parren et al.(1997b	1996), Trkola et a), Sullivan et al.	I.(1996a), Wu et al.(19 [1998)]	96),
	• C11: Mutations that inhibi	it binding: C1 (4	C11: Mutations that inhibit binding: C1 (45 W/S, 88 N/P) – V5 (463 N/D) – and C5	0) – and C5 (491	(491 I/F,493 P/K and 495 G/K)	}/K)
	and enhance binding: C1 (C11: Binding enhanced by	(36 V/L) - VI-V anti-V3 MAb 5	and enhance binding: C1 (36 V/L) – V1-V2 (152/153 GE/SM) – and \(\Delta\text{V}\text{I/V2/V3}\) [Moore et al.(1994d)] C11: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore & Sodroski(1996)]	1/V2/V3 [Moore h anti-C1 MAbs	et al.(1994d)] Moore & Sodroski(19	96)]
	 C11: Did not block ability [Wu et al.(1996)] 	y of gp120-sCD	C11: Did not block ability of gp120-sCD4 complexes to inhibit MIP-1 α binding – [Wu et al.(1996)]	χ binding – bind	binds to gp41-binding domain	nain
	 C11: Does not neutralize [Trkola et al.(1996a)] 	JR-FL nor bloc	C11: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]	ϵ -5 in a MIP-1 β -	CCR-5 competition st	tudy
	 C11: A low avidity antibody as assessed by urea elution C11: Study shows neutralization is not predicted by MA 	dy as assessed b	C11: A low avidity antibody as assessed by urea elution C11: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with	L monomeric gr	120, but is associated	with
	oligomeric Env binding – • C11: Binds efficiently to	C11 bound mon sgp120 but not	oligomeric Env binding – C11 bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)] C11: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41	neutralize JRFI ing its gp120 ep	[Fouts et al.(1997)] itope is blocked by g	;p41
	deleted [Wyatt et al.(1997)] C11: Does not neutralize To C11: Does not compete w)] TCLA strains or with binding o	deleted [Wyatt et al.(1997)] C11: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)] C11: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 [Sullivan	(1997b)] to gp120-CD4	CD4 complex, CG10 [Sullivan	ivan

1121200				0	9,000	Species (reserved)
542 212A	gp120(C1-C5 dis) gp120(dis) DISCONTINUOUS N HIV-1 infection Donor: J. Robinson, Tulane University, LA References: [Robinson et al. (1992), Moore et al. (1994d), Moore & Sodroski (1996), Binley et al. (1997), Fouts et al. (1997).	gp120(dis) iversity, LA 92), Moore et a	DISCONTINUOUS 1.(1994d), Moore & Sodroski	N 1996), Binley et a	HIV-1 infection ul.(1997), Fouts et al.(1997)	human 7),
543 L81	References: [Robinson et al. (1992), Moore et al. (1994d), Moore & Sodroski (1996), Binley et al. (1997). Ditzel et al. (1997), Wyatt et al. (1997), Parren et al. (1997b), Sullivan et al. (1998), Binley et al. (1998)] NOTES: • 212A: Mutations that inhibit binding: C1 (45 W/S) and V5 (463 N/D) – and enhance binding: V2 and C5 (495 G/K) [Moore et al. (1994d)] • 212A: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore et 212A: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore et 212A: A low avidity antibody as assessed by urea elution • 212A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, bur oligomeric Env binding – 212A bound monomer, did not bind oligomer or neutralize JRFL [Fout et 212A: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids are deleted [Wyatt et al.(1 et 212A: Does not neutralize TCLA strains or primary isolates [Parren et al. (1997b)] • 212A: Does not compete with binding of MAb generated in response to gp120-CD4 comples et al.(1998)] • 212A: A panel of MAbs were shown to bind with similar or greater affinity and similar compe deglycosylated or variable loop deleted core gp120 protein (\(\Delta \V \), V2, and V3), thus such a core a structure closely approximating full length folded monomer [Binley et al.(1998)] gp120(C1-C5 dis) Donor: J. Robinson, Tulane University, LA References: [Ditzel et al.(1997), Parren et al.(1997b)] • L81: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection binding is abolished by C1 substitution 45 W/S, C5 substitution 491 l/F, and C3 substitution L/A [1]	92), Moore eta 1997), Parren eta 1997), Parren eta 1997), Parren eta 1997), Parren et binding: C1 (et al.(1994d)] anti-V3 MAb 5 dy as assessed dy as assessed dy as assessed dy as assessed explication is not properly but no sign 120 but no sign 120 but no 1XBC2 gp120 in CLA strains confict the strains of the coop deleted coop deleted coop deleted coop atting full leng gp120 (dis) iversity, LA iversity, LA isolid phase by substitution 45	Ferences: Robinson et al. (1992), Moore et al. (1994d), Moore & Sodroski (1996), Binley et al. (1997), Fouts et al. (1997), Fouts et al. (1997), Parren et al. (1997b), Sullivan et al. (1998), Binley et al. (1998)] 7ES: 212A: Mutations that inhibit binding: C1 (45 W/S) and V5 (463 N/D) – and enhance binding: V2 (179/180 LD/DL) and C5 (495 G/K) [Moore et al. (1994d)] 212A: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore & Sodroski (1996)] 212A: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore & Sodroski (1996)] 212A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 212A bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al. (1997)] 212A: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids are deleted [Wyatt et al. (1997)] 212A: Does not neutralize TCLA strains or primary isolates [Parren et al. (1997b)] 212A: Does not neutralize TCLA strains or primary isolates [Parren et al. (1997b)] 212A: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 [Sullivan et al. (1998)] 212A: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (\Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley et al. (1998)] 20(C1-C5 dis) 30 MHIV infection 31 MHIV infection 32 MES: 13 L81: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – L81 binding is abolished by C1 substitution 45 W/S, C5 substitution 491 I/F, and C3 substitution L/A [Ditzel et al. (1997)]	and enhance bine ith anti-C1 MAbs if al. (1997b)] e to gp120-CD4 ffinity and similar, and V3), thus sused and C3 substitution and C3 substitution.	et al.(1997), Fouts et al.(1997), t al.(1998)] inding: V2 (179/180 LD/DL) Abs [Moore & Sodroski(1996)] c gp120, but is associated with JRFL [Fouts et al.(1997)] 0 epitope is blocked by gp41 yatt et al.(1997)] D4 complex, CG10 [Sullivan nilar competition profiles to a s such a core protein produces HIV infection HIV infection sed for selection of Fabs – L81 aution L/A [Ditzel et al.(1997)]	7), (5)] (ith 41 41 41 human(IgG ₁) 81

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	544 2G12	MAb ID
D	Ç	
onor: Herman Katinger, Ins	gp120(C2-C3-V4 dis)	Location
t. Appl. Mi	gp120(dis)	WEAU
Donor: Herman Katinger, Inst. Appl. Microbiol. or Polymun Scientific Inc., Vienn	gp120(dis) DISCONTINUOUS	Sequence
ja,	LΡ	Neutralizing
Austria, MRC AIDS reagent	HIV-1 infection	Immunogen
eagent	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$	Species(Isotype)

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

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

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

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

MAb ID	Location	WEAU	Sequence Neutralizing Immunogen Species(Isotype)
545 SUMMARY CD4BS	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS
	 Donor: Herman Katinger, Inst. Appl. Microbiol. or Polymun S project References: [Thali et al.(1993), Moore & Sodroski(1996)] NOTES: Shared components of MAb epitopes and the discontinuous C 370, Lys 421 through Trp 427 and Asp 457 [Thali et al.(1993)] Anti-CD4 binding site antibodies (CD4BS) competitively inhibit in precise dependence on gp120 residues, but generally require 	. Appl. Micr , Moore & Soc b epitopes and 27 and Asp 45 oodies (CD4BS p120 residues,	 Donor: Herman Katinger, Inst. Appl. Microbiol. or Polymun Scientific Inc., Vienna, Austria, MRC AIDS reagent project References: [Thali et al.(1993), Moore & Sodroski(1996)] NOTES: Shared components of MAb epitopes and the discontinuous CD4 binding regions included Thr 257, Asp 368, Glu 370, Lys 421 through Trp 427 and Asp 457 [Thali et al.(1993)] Anti-CD4 binding site antibodies (CD4BS) competitively inhibit CD4 binding to monomeric gp120, and they differ in precise dependence on gp120 residues, but generally require Asp-368 and Glu-370 [Moore & Sodroski(1996)]
546 2G6	gp120(CD4BS dis) Donor: Herman Katinger, Inst. / References: [Fouts et al.(1998)] NOTES:	gp120(dis) Appl. Microb	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS Donor: Herman Katinger, Inst. Appl. Microbiol. or Polymun Scientific Inc., Vienna, Austria References: [Fouts et al.(1998)] NOTES:
	 2G6: Binds to JRFL oligon of oligomer is not always pr authors propose a model when enhances CCR5 binding an 	ner with an affi redictive of neurere 205-46-9 de thus counter	2G6: Binds to JRFL oligomer with an affinity comparable to IgG1b12, but does not neutralize the virus, so binding of oligomer is not always predictive of neutralization – conclusions of this paper contrast with [Parren et al.(1998)] – authors propose a model where 205-46-9 and 2G6 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts the neutralizing effect [Fouts et al.(1998)]
547 588-D	gp120(CD4BS dis) gp120(dis) DISCON Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Karwowska et al.(1992a), Buchbinder et a NOTES:	gp120(dis) /U Med Cente (1992a), Buchl	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L HIV-1 infection human($\lg G_{1\kappa}$) Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Karwowska et al.(1992a), Buchbinder et al.(1992), Moore & Ho(1993), Jeffs et al.(1996)] NOTES:
	 588-D: Also called 588 588-D: Conformational – re 588-D: 4-fold increase in ne et al.(1992)] 	eactive with III utralization po	588-D: Also called 588 588-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska et al.(1992a)] 588-D: 4-fold increase in neutralization potency for 588-D when combined 1:1 with human MAb 447-D [Buchbinder et al.(1992)]
	 588-D: Weak neutralization 588-D: Called 588 – slight, [Jeffs et al.(1996)] 	of IIIB – strong not significant	588-D: Weak neutralization of IIIB – strong inhibition of HIV+human sera binding to IIIB gp120 [Moore & Ho(1993)] 588-D: Called 588 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
552 S1-1	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Lake et al.(1992), Moran et al.(1993), Wisnewski et al.(1996)] NOTES:	gp120(dis) U Med Center, Moran et al.(19	DISCONTINUOUS NY, NY 993), Wisnewski et al.(1996)]	T	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\lambda})$
	 S1-1: Neutralizes IIIB and MN without complement, and neutralizes RF and a clinical isolate with complement – binds to native but not denatured gp120 – inhibits sCD4-gp120 binding [Lake et al.(1992)] S1-1: Heavy (V_HI) and light (V_λIII) chain sequenced – no enhancing activity – similar germline sequence to MAb 86, but very different activity [Moran et al.(1993)] S1-1: S1-1 is V_H1 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisnewski et al.(1996)] 	MN without co ured gp120 – in t (V_{λ} III) chain y [Moran et al.(y n heavy chain	S1-1: Neutralizes IIIB and MN without complement, and neutralizes RF and a clinical isolate with complement – binds to native but not denatured gp120 – inhibits sCD4-gp120 binding [Lake et al.(1992)] S1-1: Heavy (V_H I) and light (V_λ III) chain sequenced – no enhancing activity – similar germline sequence to MAb 86, but very different activity [Moran et al.(1993)] S1-1: S1-1 is V_H 1 – V-region heavy chain usage was examined and a bias of enhanced V_H 1 and V_H 4, and reduced V_H 3, was noted among HIV infected individuals [Wisnewski et al.(1996)]	F and a clinical is Lake et al.(1992) stivity – similar g as of enhanced V)]	solate with complemen $]$ ermline sequence to M. $_{H}^{1}$ and V_{H}^{4} , and reduc	(t – Ab ;ed
553 559/64-D	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Karwowska et al.(1992a), McKeating et al.(1992), Spear et al.(1993), Forthal Hioe et al.(1997)] NOTES:	gp120(dis) U Med Center, 992a), McKeat	DISCONTINUOUS NY, NY ing et al.(1992), Spear et al.(1		HIV-1 infection et al.(1995), Jeffs et al.(1996),	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$ 6),
	 559/64-D: Also called 559 559/64-D: Conformational – 559/64-D: Did not mediate c 559/64-D: Neutralizing activ 559/64-D: Called 559 – slig gp120 [Jeffs et al.(1996)] 559/64-D: Used in the devel 	reactive with leposition of coality, no ADCC ht, not signification.	559/64-D: Also called 559 559/64-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska et al.(1992a)] 559/64-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear et al.(1993)] 559/64-D: Neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] 559/64-D: Called 559 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)] 559/64-D: Used in the development of resting cell neutralization assay [Hioe et al.(1997)]	3 assay [Karwow. HIV infected cel ng activity [Forth '1/V2 or V1/V2 &	ska et al.(1992a)] ls [Spear et al.(1993)] al et al.(1995)] ınd V3 were deleted fra	Эm
554 428	gp120(CD4BS dis) Ponor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Karwowska et al.(1992a), Jeffs et al.(1996)]	U Med Center, 1992a), Jeffs et	? NY, NY al.(1996)]		HIV-1 infection	human
	• 428: Slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]	increased bin	ding when V1/V2 or V1/V2	and V3 were d	eleted from gp120 [Je	:ffs
555 558-D	gp120(CD4BS dis) gp120(dis) DISCON Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [McKeating et al.(1992)] NOTES:	gp120(dis) U Med Center, 992)]	DISCONTINUOUS NY, NY	٢	HIV-1 infection	human
	• 558-D: Blocks gp120-CD4 binding – binds a panel of conformationally disruptive [McKeating et al.(1992)]	inding – binds i [McKeating et	558-D: Blocks gp120-CD4 binding – binds a panel of mutants all except for 256 S/Y and 262 N/T, which are probably conformationally disruptive [McKeating et al.(1992)]	or 256 S/Y and 26	2 N/T, which are probal	bly

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
556 448-D	gp120(CD4BS dis) gp120(dis) Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Karwowska et al.(1992a), McKeating et al. Manca et al.(1995), Li et al.(1997), Wyatt et al.(1998)] NOTES: 448-D: Also called 448D 448-D: Conformational – reactive with IIIB gp120 i 448-D: Called 448D – blocks gp120-CD4 binding – reduce binding – epitope similar to rat MAbs 39.13; 448-D: Did not mediate deposition of complement o 448-D: Dissociation constant gp120 IIIB 0.029 – na [Laal et al.(1994)] 448-D: Neutralizing activity, positive ADCC activity 448-D: Virions complexed to gp120 Ab facilitate pr	gp120(dis) NYU Med Cente L(1992a), McKe 997), Wyatt et a 997), Wyatt ot a reactive with II ocks gp120-CD4 similar to rat M. leposition of con stant gp120 IIIB d to gp120 Ab fi	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L HIV-1 infection Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Karwowska et al.(1992a), McKeating et al.(1992), Spear et al.(1993), Laal et al.(1994), Forthal et al.(1995), Manca et al.(1995), Li et al.(1997), Wyatt et al.(1998)] NOTES: 448-D: Also called 448D 448-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska et al.(1992a)] 448-D: Called 448D – blocks gp120-CD4 binding – substitutions at gp120 residues 88, 113, 117, 257, 368 and 370 reduce binding – epitope similar to rat MAbs 39.13g and 39.3b [McKeating et al.(1992)] 448-D: Dissociation constant gp120 IIIB 0.029 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D [Laal et al.(1994)] 448-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca et al.(1995)] 448-D: One of 14 burner, MAbs tested for ability to neutralize a chimeric SHIVA must, which expressed HIVA IIIIB	L 1993), Laal et al.(1993), Laal et al.(20 residues 88, 1 ting et al.(1992)] [V infected cells [V infected cells [synergistically wincing activity [For	HIV-1 infection et al.(1994), Forthal et al.(1995), swska et al.(1992a)] 88, 113, 117, 257, 368 and 370 992)] cells [Spear et al.(1993)] lly with anti-V3 MAb 447-52D y [Forthal et al.(1995)] o Th cells [Manca et al.(1995)] o Th cells [Manca et al.(1995)]	human($\lg G_{1\lambda}$) 995), 370 52D 593
557 729-D	is known about mutations that reduce NAb binding: interference with CD4 binding [Wyatt et al.(1998)] gp120(CD4BS dis) gp120(CD4BS dis) gp120(dis) Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Laal et al.(1994), D'Souza et al.(1997), Li NOTES: 729-D: Also called 729-30D 729-D: Dissociation constant gp120 IIIB 0.025 – ne [Laal et al.(1994)] 729-D: In a multilaboratory blinded study, failed to reported here to have a \(\lambda \) light chain, but originally re 729-D: Called 720-30D – one of 14 human MAbs test HIV-1 IIIB env [Li et al.(1997)] 729-D: Neutralizes TCLA strains, but not primary is	s that reduce NA nding [Wyatt et a gp120(dis) NYU Med Cente c), D'Souza et al. OD stant gp120 IIIB stant gp120 IIIB ory blinded stud light chain, but cone of 14 human 1997)]	is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)] gp120(CD4BS dis) gp120(cD4BS dis) gp120(dis) gp120(dis) gp120(dis) DISCONTINUOUS L HIV-1 infection probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)] propor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Laal et al.(1994), D'Souza et al.(1997), Li et al.(1997), Parren et al.(1997b)] • 729-D: Also called 729-30D • 729-D: Dissociation constant gp120 IIIB 0.025 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D [Laal et al.(1994)] • 729-D: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – reported here to have a \(\lambda \) light chain, but originally reported in [Laal et al.(1994)] to be IgG _{1/6} [D'Souza et al.(1997)] • 729-D: Called 720-30D – one of 14 human MAbs tested for ability to neutralize chimeric SHIV-vpu+, which expressed HIV-1 IIIB env [Li et al.(1997)] • 729-D: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]	et al.(1997b)] et al.(1997b)] et al.(1997b)] lize any of nine 1 l.(1994)] to be Ig(ralize chimeric SH	HIV-1 infection HIV-1 infection HIV-1 solution HIV-1 infection Output HIV-1 infection Output Ou	human(IgG _{1κ}) 52D 97)] ssed

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
558 HF1.7	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	Т	purified anti-Leu-3a MAb	murine(IgM)
	Donor: ? References: [Chanh et al.(1987)] NOTES: • HF1.7: An anti-Id antibody, stim mimics CD4 [Chanh et al.(1987)]	87)] dy, stimulated b	nor: ? ferences: [Chanh et al.(1987)] NTES: HF1.7: An anti-Id antibody, stimulated by anti-CD4 MAb Leu-3a, binds a recombinant gp160, suggesting HF1.7 mimics CD4 [Chanh et al.(1987)]	nds a recombinan	t gp160, suggesting HF	1.7
559 D20	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	T	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)
	Donor: ? References: [Broder et al.(1994), Richardson Jr et al.(1996), Otteken et al.(1996), Earl et al.(1997)] NOTES:	94), Richardson	Jr et al.(1996), Otteken et al.	(1996), Earl et al.([1997)]	
	 D20: Binding completely blocked t D20: Human sera blocked binding and T4 [Richardson Jr et al.(1996)] D20: Pulse label experiments of 4 these anti-CD4 MAbs bound with 	blocked by pood binding in oligal. (1996)] nents of 4 MAbsund with a dela	D20: Binding completely blocked by pooled human sera [Broder et al.(1994)] D20: Human sera blocked binding in oligomeric ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 [Richardson Jr et al.(1996)] D20: Pulse label experiments of 4 MAbs (D20, D27, T20, and T22) binding to noncleavable gp160 revealed that these anti-CD4 MAbs bound with a delay, and that the epitope formed with a t _{1/2} of about 10 minutes [Otteken	(1994)] milar extent for gp binding to noncles ad with a $t_{1/2}$ of i	r gp41 MAbs D20, D43, D61, ncleavable gp160 revealed that of about 10 minutes [Otteken	61, hat :en
	et al.(1996)]D20: Used for comparisEnv than any of 38 confo	on in a study of rmation depende	et al.(1996)] D20: Used for comparison in a study of gp41 antibodies – D20 binds to a greater Env than any of 38 conformation dependent anti-gp41 MAbs [Earl et al.(1997)]		extent to cell surface expressed	šed
560 D60	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	Т	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)
	Donor: ? References: [Richardson Jr et al.(1996)]	t al.(1996)]				
561 50-61A	gp120(CD4BS dis) Donor: ?	gp120(dis)		Т	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_\kappa)$
	NOTES: [FeVIIEI et al.(1993)]	95)]	DISCONTINUOUS			

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
562 48-16	gp120(CD4BS dis) Donor: ? References: [Fevrier et al.(1995)] NOTES: • 48-16: Broadly cross-reactive seropositive subjects – bindin;	gp120(dis) 995)] active, reacts out nding affinity 2 -	onor: ? eferences: [Fevrier et al.(1995)] OTES: • 48-16: Broadly cross-reactive, reacts outside the CD4 binding site and V3 region seropositive subjects – binding affinity 2 – 5 × 10 ⁻⁹ M [Fevrier et al.(1995)]		HIV-1 infection - competes with sera from 45	$\operatorname{human}(\operatorname{IgG}_{\kappa})$ $\operatorname{m} 45$
563 L41	gp120(CD4BS dis) Donor: ? References: [Ditzel et al.(1995)] NOTES: • L41: Substitutions at 133 D. binding – paradoxically, this I is sensitive to deglycosylatio	gp120(dis) 95)] 3 D/R, 256 S/Y, 2 his Fab was retrievation – heavy and	or: ? or: Pirences: [Ditzel et al.(1995)] IES: L41: Substitutions at 133 D/R, 256 S/Y, 257 T/R, 368 D/R or D/T, 370 E/Q or E/R, 384 Y/E, and 421 K/L reduce binding – paradoxically, this Fab was retrieved from the library after masking with known anti-CD4BS MAbs – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]	L E/Q or E/R, 38- ing with known ar equence is availal	HIV-1 infection 4 Y/E, and 421 K/L renti-CD4BS MAbs—bir ble [Ditzel et al.(1995)	$\begin{array}{c} human(IgG_{1\kappa}) \\ \\ \\ \\ \\ \\ \\ \\ \\ \\ \\ \\ \\ \\ \\ \\ \\ \\ \\$
564 L28	gp120(CD4BS dis) Donor: ? References: [Ditzel et al.(1995)] NOTES: • L28: Substitutions at 257 T, binding was enhanced by rer 435 Y/H or Y/R – binding is ; [Ditzel et al.(1995)]	gp120(dis) 95)] 7 T/R, 368 D/R, removal of the V g is sensitive to de	20(CD4BS dis) gp120(dis) DISCONTINUOUS L HIV-1 infection nor: ? erences: [Ditzel et al.(1995)] TES: L28: Substitutions at 257 T/R, 368 D/R, 370 E/R and 370 E/Q, 475 M/S 102 E/L and 463 N/D reduce binding — binding was enhanced by removal of the V3 loop and by substitutions 45 W/S, 298 R/G, 381 E/P, 382 F/L, 420 L/R, 435 Y/H or Y/R — binding is sensitive to deglycosylation — heavy and light chain variable region sequence is available [Ditzel et al.(1995)]	L A/S 102 E/L and 5 W/S, 298 R/G, nt chain variable r	HIV-1 infection and 463 N/D reduce binding – R/G, 381 E/P, 382 F/L, 420 I/R, able region sequence is available	$\text{human}(\text{Ig}G_{1\kappa})$ $\text{ing } -$ $\text{O I/R},$ ilable
565 L33	gp120(CD4BS dis) Donor: ? References: [Ditzel et al.(1995)] NOTES: • L33: binding is sensitive to et al.(1995)]	gp120(dis) 95)] to deglycosylat	20(CD4BS dis) gp120(dis) DISCONTINUOUS L HIV-1 infection nor:? erences: [Ditzel et al.(1995)] TES: L33: binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]	L ariable region se	HIV-1 infection quence is available [L	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$ Ditzel
566 L42	gp120(CD4BS dis) gp120(dis) Donor: ? References: [Ditzel et al.(1995)] NOTES: • L42: Substitutions at 257 T/R, 368 D/R, 3 enhanced by 381 E/P and 382 F/L – bindisequence is available [Ditzel et al.(1995)]	gp120(dis) 95)] 7 T/R, 368 D/R, 3 1 382 F/L – bindi tzel et al.(1995)]	20(CD4BS dis) gp120(dis) DISCONTINUOUS L HIV-1 infection or: ? erences: [Ditzel et al.(1995)] IES: L42: Substitutions at 257 T/R, 368 D/R, 370 E/R, 266 A/E and 477 D/V reduce binding – binding was significantly enhanced by 381 E/P and 382 F/L – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]	L / reduce binding tion – heavy and	HIV-1 infection ing – binding was significantly and light chain variable region	$ ext{human}(ext{Ig}G_{1\kappa})$ antly $ ext{egion}$

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
567 L52	gp120(CD4BS dis) Donor: ? References: [Ditzel et al.(1995)] NOTES: • L52: Binding is sensitive to et al.(1995)]	gp120(dis))] o deglycosylati	20(CD4BS dis) gp120(dis) DISCONTINUOUS L HIV-1 infection leave and light chain variable region sequence is available [Ditzel et al.(1995)]	L ariable region se	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$ itzel
568 GP13	gp120(CD4BS dis) gp120(dis) JISCONTINUOUS References: [Schutten et al.(1993), Back et al.(1993), Bagley et al.(1994), Schutten et al.(1995a), Schutten et al.(1995b), Bolmstedt et al.(1996), Wisnewski et al.(1996), Schutten et al.(1996), Schutten et al.(1997)] NOTES: CD13: Neutralized a broad range of HIV.1 strains from abulgametically different subfamilies — the following cm120	gp120(dis) 93), Back et al.(ski et al.(1996)	DISCONTINUOUS (1993), Bagley et al.(1994), S., Schutten et al.(1996), Schut	L chutten et al.(199) ten et al.(1997)]	HIV-1 infection 5a), Schutten et al.(199	$\begin{array}{c} \text{human(IgG}_1) \\ \text{5b),} \end{array}$
	 GP13: Neutralized a broad range of I amino acid substitutions strongly inh D), 384(Y/E) [Schutten et al.(1993)] GP13: Mutations in a neutralization in neutralization, but the escape was noted of GP13: Neutralizes IIIB – only slight viruses, that incorporated different et in GP13: Neutralizes T-cell adapted et al.(1995b)] GP13: Sera was obtained from guine N406, N448, and N463 – these sera c [Bolmstedt et al.(1996)] GP13: GP13 is V_H5 – V-region heav V_H3, was noted among HIV infected GP13: IIIB neutralizing MAbs <i>in vit.</i> GP13: Neutralized (50%) an SI-envet al.(1997)] 	range of HIV-1 rongly inhibit bul.(1993)] alization resistate was not as closely was not as closely slight inhibitifierent envs from guinea pigs rom guinea pigs rom guinea pigs rese sera could lese sera could ly infected individuals. Vinfected individuals an SI-env chiral	GP13: Neutralized a broad range of HIV-1 strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D), 384(Y/E) [Schutten et al.(1993)] GP13: Mutations in a neutralization resistant isolate obtained by passage of the IIIB isolate in chimpanzees reduced neutralization, but the escape was not as clear as seen with anti-V3 MAbs [Back et al.(1993)] GP13: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten et al.(1995a)] GP13: Neutralizes T-cell adapted viruses but not the SI strain 16.2, despite high binding affinity [Schutten et al.(1995b)] GP13: Sera was obtained from guinea pigs vaccinated either with gp160, or with gp160 lacking N-linked glycans at N406, N448, and N463 – these sera could block equally well both the CD4 BS MAb GP13 and the V3 MAb F58/H3 [Bolmstedt et al.(1996)] GP13: GP13 is V _H 5 – V-region heavy chain usage was examined and a bias of enhanced V _H 1 and V _H 4, and reduced V _H 3, was noted among HIV infected individuals [Wisnewski et al.(1996)] GP13: Neutralized (50%) an SI-env chimeric virus and enhanced (>5 fold) an NSI-env chimeric virus [Schutten et al.(1996)]	different subfam 262(N/T), 368(D 262(N/T), 368(D e of the IIIB isola bs [Back et al.(19 bng enhancement et al.(1995a)] 2, despite high 2, or with gp1601 D4 BS MAb GP1 D4 BS MAb GP1 bias of enhanced \(\) (6)] fel <i>in vivo</i> [Schutt fold) an NSI-em	bfamilies – the following gp120 68(D/R or K), 370(E/R or Q or isolate in chimpanzees reduced ul.(1993)] nent of NSI phenotype chimeric ligh binding affinity [Schutten 160 lacking N-linked glycans at GP13 and the V3 MAb F58/H3 ced V _H 1 and V _H 4, and reduced chutten et al.(1996)] SI-env chimeric virus [Schutten	120 Q or Iced Iceric Itten Is at I/H3 Iced Iced

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
569 GP44	gp120(CD4BS dis) References: [Schutten et al.(19 NOTES: GP44: Exhibited a more re acid substitutions strongly [Schutten et al.(1993)] GP44: GP44 is V _H 1 – V-re V _H 3, was noted among H	gp120(dis) 993), Bagley et a stricted pattern of inhibit binding gjion heavy chai	gp120(CD4BS dis) gp120(dis) dp120(dis) DISCONTINUOUS L HIV-1 infection References: [Schutten et al.(1993), Bagley et al.(1994), Wisnewski et al.(1996)] NOTES: • GP44: Exhibited a more restricted pattern of neutralizing activity than GP13 and GP68 – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D) [Schutten et al.(1993)] • GP44: GP44 is V _H 1 – V-region heavy chain usage was examined and a bias of enhanced V _H 1 and V _H 4, and reduced V _H 3, was noted among HIV infected individuals [Wisnewski et al.(1996)]	L [3] [3] [4] [5] [7] [7] [7] [8] [8] [7] [8] [7] [8] [7] [8] [9] [1] [9] [1] [1] [1] [1] [1] [1] [1] [1] [1] [1	HIV-1 infection ne following gp120 am K), $370(E/R \text{ or } Q \text{ or } H)$ H and $V_H A$, and redu	human(IgG1) iino D) ced
570 L72	gp120(CD4BS dis) gp120(dis) DISCONTINUOU Donor: Dr. Hariharam, IDEC Pharmaceuticals Corp La Jolla, CA References: [Ditzel et al.(1997)] NOTES: L72: Used to bind gp120 to solid phase to select MAbs from a	gp120(dis) Pharmaceuticals ')] o solid phase to	gp120(dis) DISCONTINUOUS or: Dr. Hariharam, IDEC Pharmaceuticals Corp La Jolla, CA erences: [Ditzel et al.(1997)] TES: L72: Used to bind gp120 to solid phase to select MAbs from a phage selection library [Ditzel et al.(1997)]	ction library [Di	tzel et al.(1997)]	murine
571 GP68	gp120(CD4BS dis) Donor: Dr. Hariharam, IDEC Pharmaceuticals Corp La Jolla, CA References: [Schutten et al.(1993), Klasse et al.(1993a), Bagley et z OTES: GP68: Neutralized a broad range of HIV-1 lab strains from phygp120 amino acid substitutions strongly inhibit binding: 117(K) 370(E/R or Q), 384(Y/E), 435(Y/H) [Schutten et al.(1993)] GP68: The gp41 mutation 582(Ala to Thr) results in conform resistance to a class of conformation sensitive neutralizing MAI to neutralize the mutant than wild type [Klasse et al.(1993a)] GP68: Neutralizes IIIB – only slight inhibition of SI phenotype, viruses, that incorporated different envs from the same donor [S GP68: GP68 is V _H 1 – V-region heavy chain usage was examined V _H 3, was noted among HIV infected individuals [Wisnewski et GP68: UK Medical Research Council AIDS reagent: ARP3055	gp120(dis) Pharmaceuticals Pharmaceuticals 993), Klasse et a d range of HIV- tions strongly in 435(Y/H) [Schu 435(Y/H) [Schu 6582(Ala to Th fformation sensi an wild type [Kl 1 nly slight inhibi fifferent envs fro gion heavy chai V infected indiv cch Council AIL	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L HIV-1 infection Donor: Dr. Hariharam, IDEC Pharmaceuticals Corp La Jolla, CA References: [Schutten et al.(1993), Klasse et al.(1993a), Bagley et al.(1994), Schutten et al.(1995a)] NOTES: • GP68: Neutralized a broad range of HIV-1 lab strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 117(K/W), 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q), 384(Y/E), 435(Y/H) [Schutten et al.(1993)] • GP68: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – GP68 required markedly higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] • GP68: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten et al.(1995a)] • GP68: GP68 is V _H 1 – V-region heavy chain usage was examined and a bias of enhanced V _H 1 and V _H 4, and reduced V _H 3, was noted among HIV infected individuals [Wisnewski et al.(1996)] • GP68: UK Medical Research Council AIDS reagent: ARP3055	L Schutten et al.(1 ally different su VY), 257(T/G), anges in gp120 required marke required marke (enhancement o al.(1995a)] s of enhanced V	HIV-1 infection t al.(1995a)] rent subfamilies – the following T/G), 262(N/T), 368(D/R or K), pp120 that confer neutralization markedly higher concentrations nent of NSI phenotype chimeric plant of V _H 1 and V _H 4, and reduced ced V _H 1 and V _H 4, and reduced	human(IgG1) ring K), rion ons eric ced

pp120(CD4BS dis) gp120(dis) dp120(CD4BS dis) pmor: Jackie Cordell and C. Dean References: [Cordell et al.(1991), McKeating et al.(1992a), McKeating et al.(1993b), Moore & Ho(1993), Thali et al.(1993b), Klasse et al.(1993a), McLain & Dimmock(1994), Beretta & Dalgleish(1994), McKeating et al.(1993b), Moore & Ho(1993), Thali et al.(1993b), Klasse et al.(1993a), McLain & Dimmock(1994), Beretta & Dalgleish(1994), McKeating et al.(1993b), Moore & Ho(1993), Thali et al.(1993b), Klasse et al.(1993a), McLain & Dimmock(1994), Beretta & Dalgleish(1994), McKeating et al.(1993b), Moore & Ho(1993b), Thali et al.(1993b), Klasse et al.(1993b), McLain & Dimmock(1994b), McKeating et al.(1993b), McLain & Dimmock(1994b), McKeating et al.(1993b), McCa 39.13g; Slass known as ICR39.13g and 39.13g and 15e [Cordell et al.(1991)] ICR 39.13g; Neutralization activity against HXB10, RF, SF-2 and MN strains of HIV-1 [McKeating et al.(1993b)] ICR 39.13g; Neutralization activity against HXB10, RF, SF-2 and MN strains of HIV-1 [McKeating et al.(1993b)] ICR 39.13g; Neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG McLain & Dimmock(1994)] ICR 39.13g; The gp41 mutation 582(A4 to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] ICR 39.13g; Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)] ICR 39.13g; Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)]	MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
References: [Cordell et al.(1991), McKeating et al.(1992a), McKeating et al.(1992), McKeating et al.(1993b), Moore & Ho(1993), Thali et al.(1993), Klasse et al.(1993a), McLain & Dimmock(1994), Beretta & Dalgleish(1994), McKeating et al.(1993), Thali et al.(1993), Klasse et al.(1993a), McLain & Dimmock(1994), Beretta & Dalgleish(1994), McKeating et al.(1993); Service of the properties of	572 ICR 39.13g	gp120(CD4BS dis) Donor: Jackie Cordell and C. D	gp120(dis) ean	DISCONTINUOUS	L	rgp120 BH10	$\operatorname{rat}(\operatorname{IgG}_{2b})$
NOTES: NOTES:		References: [Cordell et al.(1991) Ho(1993), Thali et al.(1993), Kl), McKeating asse et al.(199	et al.(1992a), McKeating et a 93a), McLain & Dimmock(19		ing et al.(1993b), Mod algleish(1994), McKe	ore & ating
 ICR 39.13g: also known as ICR39.13g and 39.13g ICR 39.13g: Cross-competes with MAbs ICR 39.3b and 15e [Cordell et al.(1991)] ICR 39.13g: Binds to a conformational epitope involved in CD4 binding – exerts a synergistic effect in combination with V3 directed MAbs [McKeating et al.(1992a)] ICR 39.13g: Neutralization activity against HXB10, RF, SF-2 and MN strains of HIV-1 [McKeating et al.(1993b)] ICR 39.13g: Conformational, does not bind denatured gp120 – weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)] ICR 39.13g: Strongly inhibits CD4 inducible MAb 48d [Thali et al.(1993)] ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)] ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] ICR 39.13g: Called 39.13g Neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)] ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Klasse & Sattentau(1996)] 		et al.(1996), Armstrong & Dimn NOTES:	nock(1996), K	lasse & Sattentau(1996)]			
 ICR 39.13g: Binds to a conformational epitope involved in CD4 binding – exerts a synergistic effect in combination with V3 directed MAbs [McKeating et al.(1992a)] ICR 39.13g: Neutralization activity against HXB10, RF, SF-2 and MN strains of HIV-1 [McKeating et al.(1993b)] ICR 39.13g: Conformational, does not bind denatured gp120 – weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Hot(1993)] ICR 39.13g: Strongly inhibits CD4 inducible MAb 48d [Thali et al.(1993)] ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)] ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] 		 ICR 39.13g: also known as ICR 39.13g: Cross-compete 	ICR39.13g ar s with MAbs	nd 39.13g ICR 39.3b and 15e [Cordell e	t al.(1991)]		
 ICR 39.13g: Neutralization activity against HXB10, RF, SF-2 and MN strains of HIV-1 [McKeating et al.(1993b)] ICR 39.13g: Conformational, does not bind denatured gp120 – weak neutralization of IIIB – strong inhibition of HIV+human sera binding to IIIB gp120 [Moore & Ho(1993)] ICR 39.13g: Strongly inhibits CD4 inducible MAb 48d [Thali et al.(1993)] ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)] ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralizes the mutant than wild type [Klasse et al.(1993a)] ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] 		• ICR 39.13g: Binds to a conf	ormational ep	pitope involved in CD4 bindin	g – exerts a syner	gistic effect in combin	ation
 ICR 39.13g: Conformational, does not bind denatured gp120 – weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)] ICR 39.13g: Strongly inhibits CD4 inducible MAb 48d [Thali et al.(1993)] ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)] ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] 		• ICR 39.13g: Neutralization	activity again	st HXB10, RF, SF-2 and MN	strains of HIV-1	[McKeating et al.(199	3b)]
 HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)] ICR 39.13g: Strongly inhibits CD4 inducible MAb 48d [Thali et al.(1993)] ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)] ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] 		• ICR 39.13g: Conformation	al, does not b	ind denatured gp120 - weak	neutralization of	IIIB – strong inhibiti	on of
 ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)] ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] 		HIV+ human sera binding to	o IIIB gp120 [Moore & Ho(1993)]	03)]		
and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)] • ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] • ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] • ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)] • ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)]		• ICR 39.13g: Kinetics of ne	utralization st	udied - no lag for 39.3b, whi	le ICR 39.13g an	d ICR 41.1i have lags	s of 5
 ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] 		and 15 min respectively – m	ediates neutra	lization with 2.3 molecules o	f IgG [McLain &	Dimmock(1994)]	
 Concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] 		 ICR 39.13g: The gp41 mut ization resistance to a class 	ation 582(Ala of conformati	a to Thr) results in conformation sensitive neutralizing MA	tional changes in hs – ICR 39 130	gp120 that confer ne	utral- ioher
 ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] 		concentrations to neutralize	the mutant th	an wild type [Klasse et al.(19	93a)]	,	(
 in an HXB2 background [McKeating et al.(1996)] ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)] ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] 		• ICR 39.13g: Called 39.13g1	Neutralizes H.	XB2, but fails to neutralize chi	meric virus with g	gp120 from primary iso	olates
• ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)]		• ICR 39.13g: Post-attachmen	nt neutralization	on mechanism, in contrast to	MAb 39.3b [Arm	strong & Dimmock(19	996)]
C 20 22 C Application December 1 C Toppen A C Toppen A C Toppen A C C Toppen A		• ICR 39.13g: Variants of LA	I have differin	ng neutralization susceptibility	y to 39.13g [Klass	se & Sattentau(1996)]	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
573 ICR 39.3b	gp120(CD4BS dis) Donor: J. Cordell and C. Dean		gp120(dis) DISCONTINUOUS	Т	rgp120 BH10	$\operatorname{rat}(\operatorname{IgG}_{2b})$
	References: [Cordell et al.(1991), McKeating et al.(1992), Moore et al.(1993b), McLain & Dimmock(1994), Armstrong & Dimmock(1996), Jeffs et al.(1996), Wyatt et al.(1998)] NOTES:	91), McKeatin (1996), Wyatt	ng et al.(1992), Moore et al.(et al.(1998)]	1993b), McLain &	z Dimmock(1994), <i>A</i>	Armstrong
	• ICR 39.3b: also known as 39.3, 39.3b and ICR39.3b	39.3, 39.3b a	nd ICR39.3b			
	• ICR 39.3b: Cross-compet	es with MAbs	ICR 39.3b: Cross-competes with MAbs ICR 39.13g and 15e [Cordell et al.(1991)]	ell et al.(1991)]		
	 ICR 39.3b: Conformation ICR 39.3b: Kinetics of ne 	al, does not bi utralization stı	ICR 39.3b: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and	e & Ho(1993)] ile ICR 39.13g and	d ICR 41.1i have lag	s of 5 and
	15 min respectively [McLain & Dimmock(1994)]	ain & Dimmo	ck(1994)]	((
	• ICR 39.3b: Neutralizes o	nly if the antil	ICR 39.3b: Neutralizes only if the antibody is added prior to the attachment of		the virus to the cell, in contrast to	contrast to
	39.13g [Armstrong & Dimmock(1996)]	nmock(1996)]				
	 ICR 39.3b: Called 39.3l et al.(1996)] 	- increased	ICR 39.3b: Called 39.3b – increased binding when V1/V2 or V1/V2 and V3 et al.(1996)]		were deleted from gp120 [Jeffs	120 [Jeffs
	• ICR 39.3b: Called 39.3 –	summary of t	ICR 39.3b: Called 39.3 – summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is brown about mutations that radices NAb binding a probable machanism of neutralization by	tal structure of the	core of gp120 bour	nd to CD4
	and 1/b with what is kno CD4BS Ab is direct interl	wn about muta erence with C	and 1/b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)]	ling – probable m 98)]	echanism of neutrali	ization by
571 156	• ICK 39.3b: UK Medical I	Research Coun	ICR 39.3b: UK Medical Kesearch Council AIDS reagent: ARP391	1	HIV 1 infection	human/InC.
	Donor: J. Robinson, Tulane University, LA, and David Ho, ADARC, NY, NY	Iniversity, LA,	and David Ho, ADARC, N	Y, NY		•

et al. (1993), Bagley et al. (1994), Thali et al. (1994), Cook et al. (1994), Moore et al. (1994b), Moore et al. (1994a), Sattentau et al.(1992), Wyatt et al.(1992), Thali et al.(1992a), Takeda et al.(1992), Moore & Ho(1993), Thali et al.(1993), Wyatt **References:** [Robinson et al.(1990), 1 nan et al.(1991), Corden et al.(1991), Ho et al.(1991), Koup et al.(1991), Ho

et al.(1996a), McDougal et al.(1996), Wisnewski et al.(1996), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997),

& Moore(1995), Lee et al.(1995), McKeating et al.(1996), Moore & Sodroski(1996), Poignard et al.(1996a), Trkola

Binley et al.(1998), Trkola et al.(1998), Fouts et al.(1998)] Wyatt et al. (1997), Berman et al. (1997), Parren et al. (1997b), Wyatt et al. (1998), Parren et al. (1998), Sullivan et al. (1998)

NOI HS

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

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

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

Spizo(CD+BB dis) Spizo(dis) Discontino CB
Donor: Shermaine Tilley. Public Health Research Institute. USA

et al.(1995), Warrier et al.(1996), Pincus et al.(1996), Wyatt et al.(1998), Alsmadi & Tilley(1998), Yang et al.(1998)]

OIES.

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

MAb ID	Location	WEAU	Sequence	Neutralizing Immunogen	Immunogen	Species(Isotype)
576 5145A	gp120(CD4BS dis) gp120(dis) DISCONTINUOU: Donor: Shermaine Tilley, Public Health Research Institute, USA	gp120(dis) ublic Health Re	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L HIV-1 in Donor: Shermaine Tilley, Public Health Research Institute, USA	L L	HIV-1 infection	human(IgG1)
	71 47 A D	=		7	3	
	 5145A: Syneroistic net 	tralization of HI	5145A: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G (Warrier et al. (1996))	i-V2 MAh C1080	i (Warrier et al.(1996	<i>3</i> 1
	• 5145A: A panel of imi	niinotovine were	5145A. A nanel of immunotoxing were generated by linking Fny Make to rigin	TAhe to ricin A -	A - immunotoving mediated cell	ated cell
	killing, but killing was	not directly prop	killing, but killing was not directly proportional to binding [Pincus et al.(1996)]		пиний осохииз пісси	מוכם ככוו
	 5145A: A study of 6 ar 	ti-Env MAbs an	5145A: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB,	t ADCC against t	arget cells infected w	ith IIIB,
	MN, SF-2, and RF $-$ bo	ound and directed	MN, SF-2, and RF – bound and directed lysis against all four strains [Alsmadi &	[Alsmadi & Tille	Tilley(1998)]	
577 21h	gp120(CD4BS dis)	gp120(dis)	gp120(dis) DISCONTINUOUS	L	HIV-1 infection	human(IoG,)

Donor: J. Robinson, Tulane University, LA

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

NOTES:

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

- 21h: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Poignard et al.(1996a)]
- 21h: 21h is $V_H3 V_T$ egion heavy chain usage was examined and a bias of enhanced V_H1 and V_H4 , and reduced V_H 3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- 21h: Called 2.1H Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]
- 21h: A low avidity antibody as assessed by urea elution
- 21h: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding - 21h bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- 21h: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env -50% neutralization could not be achieved at a maximal concentration of 67 μ g/ml [Li et al.(1997)]
- 21h: Viral binding inhibition by 21h strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 21h: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked [Wyatt et al.(1997)] by gp41 binding - major deletions in C1 and C5 and deletions of the V1V2 and V3 loops do not diminish binding
- 21h: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- 21h: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what interference with CD4 binding [Wyatt et al.(1998)] is known about mutations that reduce NAb binding - probable mechanism of neutralization by CD4BS Ab is direct
- 21h: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope
- 21h: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer - conclusions of this paper contrast with [Parren et al.(1998)] [Fouts et al.(1998)]
- 21h: UK Medical Research Council AIDS reagent: ARP3017

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		578 F105	MAb ID Location
References: [Posner et al.	Donor: Marshall Posner, Boston MA	gp120(CD4BS dis)	Location
(1991), Thali et	oston MA	gp120(dis)	WEAU Sequence
References: [Posner et al.(1991), Thali et al.(1991), Thali et al.(1992a), Marasco		gp120(dis) DISCONTINUOUS	Sequence
et		L	Neutralizing
al.(1992), Wyatt et al.(1992)		HIV-1 infection	Immunogen
.(1992),		$\operatorname{human}(\operatorname{IgG}_{1\kappa})$	Species(Isotype)

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

NOIES:

- F105: First description of F105, binds topographically near the CD4-binding site inhibits binding of free, infectious assays with HIV-1 and MN strains [Posner et al.(1991)] pre-bound to infected cells inhibits F105 binding – F105 inhibits infection of HT-H9 cells in standard neutralization virions to uninfected HT-H9 cells, but does not react with virus adsorbed to uninfected HT-H9 cells - soluble rCD4
- F105: Neutralization escape mutants result from changes in amino acids in four discontinuous regions: C2, 256-262 C3, 386,370; C4, 421
- F105: Amino acid substitutions that impair F105 neutralization inhibit gp120-CD4 interaction [Thali et al.(1992a)]
- F105: MAb cDNA sequence V_{H4} V71-4 rearranged with a D_{H} D-D fusion product of dlr4 and da4, and with J_{H5} $-V_{\kappa}$ is from the *Humvk325* germline gene joined with J κ 2 [Marasco et al.(1992)]
- F105: Precipitation of △ 297-329 env glycoprotein, with has a deleted V3 loop, is much more efficient than precipitation of wild type [Wyatt et al.(1992)]
- F105: F105 mediates ADCC against SF2 through the CD16+ population of PBMC does not mediate complementdependent cytotoxicity [Posner et al.(1992b)]
- F105: Significant enhancement of F105 binding to RF infected cells preincubated with V3-specific MAbs V3-2 and V3-1 [Posner et al.(1992a)]
- F105: Called F-105 neutralizes IIIB strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore &
- F105: F105 binds to and neutralizes selected lab strains and 3/9 HIV-1 primary isolates synergistic enhancement of neutralization by seropositive sera [Posner et al.(1993)]
- F105: No neutralization of primary isolates observed (John Moore, pers comm)
- F105: Additive MN or SF2 neutralization when combined with anti-V3 MAbs 447-52D and 257-D [Cavacini et al.(1993a)]
- F105: Serum from all asymptomatic HIV-1 positive people tested block F105 binding, but only from 27% of symptomatic individuals [Cavacini et al.(1993b)]
- F105: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is 2.4- and 13-fold greater, respectively, than binding to wildtype gp120 [Wyatt et al.(1993)]
- F105: Study of synergism between F105 and sera from vaccinated volunteers with V3-loop specific neutralization activity – 2/3 sera demonstrated neutralization synergy, and 3/3 binding/fusion-inhibition synergy [Montefiori

- F105: Study of synergism of neutralization and binding comparing F105 and sCD4 with the V3 MAbs: 50.1, 59.1 binding of the second (e. g. V3 loop MAbs) due to conformational changes [Potts et al.(1993)] 83.1, and 58.2 - synergy was observed, and the data suggest that binding of one ligand (F105) can increase the
- F105: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs required > 81 fold higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)]
- F105: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients F105 was used as a control at lower titers than the infected lab workers [Pincus et al.(1993)] infected lab workers and some of the gp160 vaccinees had a MAb response that could inhibit gp120-CD4 binding.
- neutralizing sera F105 neutralization was not affected by this mutation [Watkins et al.(1993)] F105: Comparison of MAb F105 sequences with those of MAbs 21h and 15e [Bagley et al.(1994)] F105: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly
- F105: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MABs 48d, 21h, 15e and 17b) [Thali et al.(1994)]
- F105: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells hindrance - binding of GalCer to gp120 inhibited but did not completely block F105 binding[Cook et al.(1994)] from the brain and colon - anti-CD4 MAbs moderately inhibit gp120 binding to GalCer, possibly through steric
- F105: Administered intravenously to four cynomologus monkeys, plasma pharmacokinetics and biological activity tested [Cavacini et al.(1994b)]
- F105: Fab fragments show reduced capacity to neutralize IIIB, MN, and RF compared to intact IgG1, suggesting bivalent interaction may be important in binding and neutralization [Cavacini et al. (1994a)]
- F105: Used as a positive control for CD4 BS antibodies in a study of the influence of oligomeric structure of Env in determining the repertoire of the Ab response [Earl et al.(1994)]
- F105: A human CD4+ T lymphocyte line was transduced to express Fab fragments of F105 heavy and light chains can protect surrounding lymphocytes by secreting neutralizing antibodies [Marasco et al. (1993), Chen et al. (1994a)] and extracellular binding activities of the expressed Fab make transduced cells resistant to HIV-1 infection and also envelope protein and inhibits HIV-1 production - secreted Fab fragments neutralize cell-free HIV-1 - combined intraare joined by an inter-chain linker – in the transduced cells infected with HIV-1, the Fab binds intracellularly to the
- F105: An immunoassay for titrating CD4BS serum antibody was developed using a gp120-coated solid phase and CD4BS Abs are not cross-reactive [Turbica et al.(1995)] detected soon after seroconversion and persisted - 0/21 HIV-2+ sera reacted, indicating that the HIV-1 and HIV-2 competition with MAb F105 - 109/110 French HIV-1+ sera and 51/56 HIV-1+ African sera had detectable CD4BS Abs using this assay, demonstrating CD4 binding site conservation among diverse subtypes - CD4BS Abs were
- F105: Eight patient phase Ia trial for use as an immunotherapeutic no clinical or biochemical side effects observed plasma levels \geq of 10 μ g/ml maintained for 21 days [Posner et al.(1995)]
- F105: Efficient neutralization of T-cell adapted lines HXBc2 and MN, no neutralization of primary isolates 89.6 ADA and YU2 - even some enhancement of infection of ADA and YU2 [Sullivan et al.(1995)]
- F105: Biotinylated F105 was used for competition studies with Ab derived from pregnant HIV-1+ women a correlation between maternal anti-CD4 BS Abs overlapping the F105 binding site and lack of HIV-1 transmission to infants was noted[Khouri et al.(1995)]

- F105: Changing heavy chain from IgG₁ to IgG₃ increased neutralization efficiency [Cavacini et al. (1995)]
- F105: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes described as 256-257 ST, 368-370 DPE, 421 K, and 470-484 PGGGDMRDNWRSELY [Jagodzinski et al.(1996)] virus - deletion of the V3 loop results in less potent inhibition of F105 binding by CRDS - binding site of F105
- F105: Phase I study MAb clearance in plasma has a 13 day half-life [Wolfe et al.(1996)]
- F105: Neutralizes HIV-1 LAI less potently than V3 specific MAbs [McDougal et al.(1996)]
- F105: F105 is $V_H4 V_T$ egion heavy chain usage was examined and a bias of enhanced V_H1 and V_H4 , and reduced V_H3 , was noted among HIV infected individuals [Wisnewski et al.(1996)]
- F105: A panel of immunotoxins were generated by linking Env MAbs to ricin A immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al. (1996)]
- F105: Binding of F105 to oligomeric gp120 occurs despite the fact it cannot neutralize primary isolates [Litwin et al.(1996)]
- F105: Intracellular co-expression of heavy and light chains of the Fab105 fragment MAb F105 was enhanced by effectively blocked [Chen et al.(1996)] and secrete the Fab105 fragments while maintaining normal growth - several primary HIV-1 patient isolates were an adeno-associated virus (AAV) shuttle vector, and transduced into human lymphocytes which were able to produce inclusion of an internal ribosome entry site (IRES) sequence – the Fab105 IRES expression cassette was cloned into
- F105: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- F105: In a multilaboratory blinded study, failed to neutralize any of nine B clade primary isolates [D'Souza et al.(1997)]
- F105: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env - F105 could only achieve 50% neutralization alone - all Ab combinations tested showed synergistic neutralization - F105 has synergistic response with MAbs 694/98-D (anti-V3), 48d, 2F5, and 2G12, and also with HIVIG [Li
- F105: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to a CD4BS MAb F105 or sCD4 [Cao et al.(1997)]
- F105: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked 31-93, are deleted [Wyatt et al.(1997)] by gp41 binding - does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions
- F105: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what interference with CD4 binding [Wyatt et al.(1998)] is known about mutations that reduce NAb binding - probable mechanism of neutralization by CD4BS Ab is direct
- F105: Phase I dose escalation study, single dose of 100 or 500 mg/m^2 was given to 4 HIV+ patients sustained anti-HIV-1 activity and virus was not diminished at day 1 or 7, by culture or plasma RNA [Cavacini et al.(1998)] levels, no immune response against F105, no toxicity, infused Ab retained function – there was no evidence of
- F105: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth
- F105: NIH AIDS Research and Reference Reagent Program: 857

MAb ID
Location
WEAU
Sequence
Neutralizing
Immunogen
Species(Isotype)

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

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

NOI ES:

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

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

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

- IgG1b12: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with interaction between CD4 and conserved amino acids on the V1-V2 stem [Wyatt et al.(1998)] a neutralizing antibody and it is susceptible to changes in the V1-V2 stem loop structure, and so it may disrupt an direct interference with CD4 binding - IgG1b12 is an unusual CD4BS antibody because it is particularly potent as what is known about mutations that reduce NAb binding - probable mechanism of neutralization by CD4BS Ab is
- IgG1b12: Enhances binding of Hx10 to CD4 positive or negative HeLa cells, inhibits binding to CD4+ T-cell line A3.01 - neutralizes HeLa and A3.01 cell Hx10 infection [Mondor et al.(1998)]
- monovalent Fab b12 [Parren et al.(1998)] sites occupied on a virion irrespective of the epitope - binding affinity of divalent IgG1b12 is 17-fold greater than IgG1b12: IgG1b12, FAb b12 and 3B3 derived from b12 were all included in this study - the rank order of FAh were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab Loop 2 > b11 > L17 > b6 > D08i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 = DO8i > b14 > b15 > DO142-10 > DA48 = DO8i > b15 > b15 > DO142-10 > DA48 = DO8i > b15 > b15 > DO142-10 > DA48 = DO8i > DO142-10 > DA48 = DO142-10 > DA48 = DO142-10 > DA48 = DO142-10 > DO142-10 > DA48 = DO142-10 > DO142-10 > DA48 = DO142-10 > DO142-10 > DO142-10 > DA48 = DO142-10 > DO142-10 > DO142-10 > DA48 = DO142-10 > DO142-10 >> L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 >
- IgG1b12: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could 2G12, IgG1b12, 2F5 and 447-52D [Connor et al.(1998)] were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses
- to the deleted protein than to wild type [Binley et al.(1998)] a structure closely approximating full length folded monomer - CD4BS MAbs 15e, F91 and IgG1b12 bound better deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces IgG1b12: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a
- depends on residues in V2 [Fouts et al.(1998)] = 205-42-15 > 15e = 21h = F91, and the only thing notably distinguishing about neutralizing IgG1b12 is that it the neutralizing effect – rank order of CD4BS antibodies oligomer binding is IgG1b12 = 2G6 = 205-46-9 > 205-43-16are not - conclusions of this paper contrast with [Parren et al.(1998)] - authors propose a model where 205-46-9 and IgG1b12: Binds JRSF oligomer with high affinity, as do 205-46-9 and 2G6, but IgG1b12 is neutralizing, the other two 2G6 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts
- IgG1b12: Induces Complement-mediated lysis in MN but not primary isolates primary isolates are refractive to CML [Takefman et al.(1998)]
- IgG1b12: UK Medical Research Council AIDS reagent: ARP3065
- IgG1b12: NIH AIDS Research and Reference Reagent Program: 2640

MAb ID	Location	WEAU	Sequence	Neutralizing Immunogen	n Species(Isotype)
580 DO8i	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, Research Inst. La Jolla, CA References: [Parren et al.(1998)] NOTES: DO8i: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding at form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA4 oligomeric form and neutralization were correlated for both Fabs and MAbs — author determined by the fraction of Ab sites occupied on a virion irrespective of the epitope	gp120(dis) arch Insitute, L)] Ab binding affit DA48 > L17) v 2-10 > Loop 2 bization were co of Ab sites occu	20(CD4BS dis) gp120(dis) DISCONTINUOUS nor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical earch Inst. La Jolla, CA eerch Eerches: [Parren et al.(1998)] TES: DO8i: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	and J. Pyati, R. W. Johnson Pharmaceutical p 2 > 3B3 > b12 = DO8i > b11 > b3 > b binding affinity to the mature oligomeric b14 > DA48 > b3 > b13) and binding to bs – authors suggest that neutralization is the epitope [Parren et al.(1998)]	Fab, human narmaceutical b11 > b3 > re oligomeric and binding to utralization is [98)]
581 DA48	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, Research Inst. La Jolla, CA References: [Parren et al.(1998)] NOTES:	gp120(dis) arch Insitute, L	DISCONTINUOUS a Jolla, CA, also J. Geltowsky a	ınd J. Pyati, R. W. Johnson Pharmaceutical	Fab, human narmaceutical
	DA48: The rank order of F b14 > b13 > DO142-10 > form (3B3 > b12 > DO142 oligomeric form and neutral determined by the fraction of the part of th	Ab binding affi DA48 > L17) v 2-10 > Loop 2 bization were confided by the sites occur	DA48: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	p 2 > 3B3 > b12 = DO8i > b11 b binding affinity to the mature ol b14 > DA48 > b3 > b13) and b bs – authors suggest that neutral f the epitope [Parren et al.(1998)]	· b11 > b3 > re oligomeric nd binding to utralization is 98)]
582 b3	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, Research Inst. La Jolla, CA References: [Parren et al.(1997b), Parren et al.(1998)] NOTES:	gp120(dis) arch Insitute, L b), Parren et al.	DISCONTINUOUS a Jolla, CA, also J. Geltowsky a (1998)]	ınd J. Pyati, R. W. Johnson Pharmaceutical	Fab, human narmaceutical
	 b3: Neutralizes TCLA strai b3: The rank order of FAb b13 > D0142-10 > DA form (3B3 > b12 > D0142 oligomeric form and neutral determined by the fraction of the control of the control	ns, but not primbinding affinity binding affinity 48 > L17) was 2-10 > Loop 2 lization were coff Ab sites occur	b3: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)] b3: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	(b)] > 3B3 > b12 = DO8i > b11 > b3 > b14 binding affinity to the mature oligomeric b14 > DA48 > b3 > b13) and binding to abs – authors suggest that neutralization is f the epitope [Parren et al.(1998)]	l > b3 > b14 re oligomeric nd binding to utralization is 98)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
583 b11	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA References: [Parren et al.(1998)] NOTES: • b11: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	gp120(dis) search Insitute, I 8)] Ab binding affir DA48 > L17) 12-10 > Loop 2 alization were c of Ab sites occ	20(CD4BS dis) gp120(dis) DISCONTINUOUS nor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical earch Inst. La Jolla, CA erences: [Parren et al.(1998)] TES: b11: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	and J. Pyati, R. W p 2 > 3B3 > b1: b binding affinity b14 > DA48 > Abs – authors sug f the epitope [Pau	i, R. W. Johnson Pharmaceutical > b12 = DO8i > b11 > b3 > affinity to the mature oligomeric 448 > b3 > b13) and binding to ors suggest that neutralization is pe [Parren et al.(1998)]	Fab, human tical 3 > eeric g to on is
584 b6	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA References: [Parren et al.(1997b), Parren et al.(1998)] NOTES: • b6: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)] • b6: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	gp120(dis) search Insitute, I 7b), Parren et al 7b), Parren et al ins, but not prin binding affinit binding affinit 448 > L17) wa 2-10 > Loop 2 alization were c of Ab sites occ	onor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical esearch Inst. La Jolla, CA eferences: [Parren et al.(1997b), Parren et al.(1998)] OTES: • b6: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)] • b6: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	L and J. Pyati, R. W 7b)] 7b)] 2 > 3B3 > b12 = binding affinity b14 > DA48 > Abs – authors sug f the epitope [Par	i, R. W. Johnson Pharmaceutical b12 = DO8i > b11 > b3 > b14 bfinity to the mature oligomeric A48 > b3 > b13) and binding to ors suggest that neutralization is pe [Parren et al.(1998)]	Fab, human tical b14 b16 b17 b18
585 b13	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyat Research Inst. La Jolla, CA References: [Parren et al.(1995), Parren et al.(1998)] NOTES: • b13: Fab b13 was used as a control in a hu-PBL SCID mouse study – animals v infection by IgG1b12, somewhat by Fab b12, but not by b13 [Parren et al.(1995), Parren et al.(1998)]	gp120(dis) eearch Insitute, I 5), Parren et al.(5), Parren et al.(as a control in a sewhat by Fab bhoinding affir DA48 > L17) 12-10 > Loop 2 alization were cof Ab sites occ	20(CD4BS dis) gp120(dis) DISCONTINUOUS or: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical earch Inst. La Jolla, CA erences: [Parren et al.(1995), Parren et al.(1998)] TES: b13: Fab b13 was used as a control in a hu-PBL SCID mouse study – animals were protected from HIV-1 SF2 infection by IgG1b12, somewhat by Fab b12, but not by b13 [Parren et al.(1995), Parren & Burton(1997)] b13: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = D08i > b11 > b14 > b13 > D0142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > D0142-10 > Loop 2 > b11 > L17 > b6 > D08i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]	and J. Pyati, R. W animals were p. l.(1995), Parren & p 2 > 3B3 > b1. b binding affinity b14 > DA48 > Abs – authors sug	i, R. W. Johnson Pharmaceutical were protected from HIV-1 SF2 arren & Burton(1997)] > b12 = DO8i > b11 > b3 > affinity to the mature oligomeric 448 > b3 > b13) and binding to ors suggest that neutralization is	Fab, human tical SF2 SF2 g to gg to n is

	Advante and annual guillante and annual annu
586 b14	gp120(CD4BS dis) gp120(dis) JISCONTINUOUS Donor: D. Burton, Scripps Research Insitute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA References: [Parren et al.(1998)] NOTES: h14: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 >
	• b14: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
587 F91	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS Donor: J. Robinson, University of Connecticut, Storrs References: [Moore & Ho(1993), Moore et al.(1994b), Moore & Sodroski(1996), Fouts et al.(1997), Mondor et al.(1998), Parren et al.(1998), Binley et al.(1998), Fouts et al.(1998)] NOTES:
	 F91: Called F-91 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)] F91: Has strong cross-reactivity with gp120 monomers from most subtypes, A-F [Moore et al.(1994b)] F91: Unusual pattern of reciprocal enhancement with several anti-V2 and V3 directed MAbs – reciprocal inhibition of other CD4BS MAbs [Moore & Sodroski(1996)]
	 F91: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – F91 bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)] F91: Weak inhibition of binding of Hx10 to CD4 positive or negative cells, weakly neutralizing [Mondor et al.(1998)] F91: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren et al.(1998)]
	 F91: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (\(\Delta \) V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MAbs 15e, F91 and IgG1b12 bound better to the deleted protein than to wild type [Binley et al.(1998)] F91: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer

MADID	Location	WEAU	Sequence	Neutranzing	ımmunogen	Species(isotype)
588 НТ6	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L (weak) Donor: Ciba-Geigy AG Basel, Switzerland, and Tanox Biosystems, Houston, Texas References: [Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997), Fouts et al.(1998)] NOTES:	gp120(dis) , Switzerland, a 94b), Moore et a	DISCONTINUOUS nd Tanox Biosystems, Housto ll.(1995a), Fouts et al.(1997), l	L (weak) nn, Texas Fouts et al.(1998)]	HIV-1 infection	human
	 HT6: HT5, HT6, and HT7 are also HT6: Despite highly cross-reactive IIIB and MN [Moore et al.(1995a)] 	7 are also knowr s-reactive bindin .(1995a)]	HT6: HT5, HT6, and HT7 are also known as 205-43-1, 205-42-15, and 205-46-9, respectively [Fouts et al.(1998)] HT6: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN [Moore et al.(1995a)]	d 205-46-9, respect adapted viral strains	ively [Fouts et al.(19 s, only weakly neutra	98)] lizes
	 HT6: 205-46-9 was cross et al.(1994b)] 	-reactive across	HT6: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was not quite as extensively cross-reactive [Moore et al.(1994b)]	ot quite as extensive	bly cross-reactive [M	[oore
	 HT6: MAbs IgG1b12, H inhibit gp120-sCD4 intera 	T5, HT6, and I ctions, but only	HT6: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts et al.(1997)]	ng to monomeric g outs et al.(1997)]	p120, bind equally	well,
	contrast with [Parren et al.(1998)] [Fouts et al.(1998)]	.(1998)] [Fouts	contrast with [Parren et al.(1998)] [Fouts et al.(1998)]	Hot Hennamzing –	COLCIUSIONS OF THIS I	зареі
589 HT5	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L (weak) Donor: Ciba-Geigy AG (Basel, Switzerland), and Tanox Biosystems, Houston, Texas References: [Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997), Fouts et al.(1998)] NOTES:	gp120(dis) !, Switzerland), 94b), Moore et a	DISCONTINUOUS and Tanox Biosystems, Housdand Tan	L (weak) ton, Texas Fouts et al.(1998)]	HIV-1 infection	human
	 HT5: HT5, HT6, and HT7 are also HT5: Despite highly cross-reactive IIIB and MN [Moore et al.(1995a)] 	7 are also knowr s-reactive bindin .(1995a)]	HT5: HT5, HT6, and HT7 are also known as 205-43-1, 205-42-15, and 205-46-9, respectively [Fouts et al.(1998)] HT5: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN [Moore et al.(1995a)]	l 205-46-9, respecti adapted viral strains	vely [Fouts et al.(199s, only weakly neutra	98)] dizes
	 HT5: 205-46-9 was cross- [Moore et al.(1994b)] 	reactive across c	HT5: 205-46-9 was cross-reactive across clades A-F, 205-43-1 very cross-reactive but not quite as extensive 205-46-9 [Moore et al.(1994b)]	s-reactive but not qu	uite as extensive 205-	46-9
	 HT5: MAbs IgG1b12, H inhibit gp120-sCD4 intera 	T5, HT6, and I ctions, but only	HT5: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts et al.(1997)]	ng to monomeric gouts et al.(1997)]	p120, bind equally	well,
	 HT5: HT5 and HT6 bind JRSF oligomer but with low contrast with [Parren et al.(1998)] [Fouts et al.(1998)] 	JRSF oligomer (1998)] [Fouts	HT5: HT5 and HT6 bind JRSF oligomer but with low affinity, and are not neutralizing – conclusions of this paper contrast with [Parren et al.(1998)] [Fouts et al.(1998)]	not neutralizing –	conclusions of this p	vaper

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
590 HT7	gp120(CD4BS dis) gp120(dis) DISCONTINUOUS L (IIIB) Donor: Ciba-Geigy AG (Basel, Switzerland), and Tanox Biosystems, Houston, Texas References: [Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997), Fouts et al.(1998)] NOTES: • HT7: HT5, HT6, and HT7 are also known as 205-43-1, 205-42-15, and 205-46-9, respec • HT7: Despite highly cross-reactive binding to many primary and T-cell adapted viral str: well, with sporadic weak neutralization of other isolates [Moore et al.(1995a)] • HT7: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was cross-reactive, but not et al.(1994b)] • HT7: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric; inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts et al.(1997)] • HT7: Binds JRSF oligomer with high affinity, at least as high as IgG1b12, but IgG1b12 i	gp120(dis) l, Switzerland), at 4b), Moore et al 4b), Moore et al 7 are also known s-reactive bindin neutralization of reactive across creactive across creative	gp120(dis) gp120(dis) DISCONTINUOUS L (IIIB) HIV-1 infection nor: Ciba-Geigy AG (Basel, Switzerland), and Tanox Biosystems, Houston, Texas ferences: [Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997), Fouts et al.(1998)] YTES: HT7: HT5, HT6, and HT7 are also known as 205-43-1, 205-42-15, and 205-46-9, respectively [Fouts et al.(1998)] HT7: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only neutralizes IIIB well, with sporadic weak neutralization of other isolates [Moore et al.(1995a)] HT7: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was cross-reactive, but not quite as extensive [Moore et al.(1994b)] HT7: MAbs [gG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only [gG1b12 neutralizes JRFL [Fouts et al.(1997)] HT7: Binds JRSF oligomer with high affinity, at least as high as [gG1b12, but IgG1b12 is neutralizing, H7 is not –	L (IIIB) on, Texas outs et al.(1998); 205-46-9, respecadapted viral str 995a)] s-reactive, but not g to monomeric g to monomeric outs et al.(1997)] 12, but IgG1b12 i	HIV-1 infection 98)] spectively [Fouts et al.(1998)] strains, only neutralizes IIIB not quite as extensive [Moore ric gp120, bind equally well, 77)] 12 is neutralizing, H7 is not –	human 98)] IIIB oore well, aot –
	[Fouts et al.(1998)]		[Fouts et al.(1998)]			
221 MAY 22	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994), Moore & Sodroski(1996)]	%P120(chs)	roski(1996)]	t	gp120)-complex	шшшс
	 MAG 55: Amino acid substitutions that reduce bindi Y/E, 421 K/L, 470 P/L, 475 M/S, 477 D/V – neutral MAG 55: Called #55 – binding reciprocally inhibit MAbs – binding enhanced by anti-V3 MAb 110.5 an anti-V3 and -V2 MAbs. [Moore & Sodroski(1996)] 	stitutions that rec 75 M/S, 477 D/V inding reciproca by anti-V3 MAI Moore & Sodros	MAG 55: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 470 P/L, 475 M/S, 477 D/V – neutralizes MN, IIIB and RF [Kang et al.(1994)] MAG 55: Called #55 – binding reciprocally inhibited by other anti-CD4 binding site MAbs, and by some C1-C5 MAbs – binding enhanced by anti-V3 MAb 110.5 and anti-V2 MAbs G3-136 and G3-4 – enhances binding of many anti-V3 and -V2 MAbs. [Moore & Sodroski(1996)]	7, 257 T/R, 368 D RF [Kang et al.(19) 94 binding site M 3-136 and G3-4 –	/R or T, 370 E/R or Q, 994)] [Abs, and by some Clenhances binding of n	, 384 1-C5 nany
592 MAG 72	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	T	sCD4-(rHXB2 gp120)-complex	murine
	 Donor: C. Y. Kang or Dr. Hariharam, IDEC Pharmaceuticals Corp, La Jolla, CA References: [Kang et al.(1994), Ditzel et al.(1997)] NOTES: MAG 72: Also called L72 MAG 72: Amino acid substitutions that reduce binding 10 fold: 257 T/R or A or Q, 384 Y/E, 421 K/L, 477 D/V – neutralizes MN, IIIB and RF [Kang et al. MAG 72: Called L72 – used to bind gp120 to solid phase to select MAbs et al.(1997)] 	iharam, IDEC Pl.), Ditzel et al.(19), Ditzel et al.(19), stitutions that recent that the stitutions that recent the stitutions that recent the stitutions that recent the stitutions that the stitutions that recent the stitutions is also below the stitution of th	 In the content of the conte	, CA t or A or G, 262 N et al.(1994)] Abs from a phag	VT, 368 D/R or T, 370 se selection library [D	E/R itzel

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
593 MAG 86	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	T	sCD4-(rHXB2	murine
	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)] NOTES: • MAG 86: Amino acid subst VE A21 KA A70 DA A77	itutions that re	nor: C. Y. Kang, IDEC Inc erences: [Kang et al.(1994)] TES: MAG 86: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 30 V.F. 421 K.F. 470 B.F. 477 D.W. neutralizas MN HIR and BETKang et al.(1994))	, 257 T/R, 368 D.	68 D/R or T, 370 E/R or Q, 384	384
594 MAG 96	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	T	sCD4-(rHXB2	murine
	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)]				gp120)-complex	
	 MAG 96: Amino acid substitutions that I neutralization of IIIB [Kang et al.(1994)] 	titutions that reg et al.(1994)]	MAG 96: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, neutralization of IIIB [Kang et al.(1994)]		368 D/R or T, 370 E/R – weak	eak
595 MAG 116	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	T	sCD4-(rHXB2	murine
	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)]				8p120/compacy	
	 MAG 116: Amino acid sub 384 Y/E, 421 K/L – neutral 	ostitutions that izes MN, IIIB	MAG 116: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L – neutralizes MN, IIIB and RF [Kang et al.(1994)]	S/Y, 257 T/R, 368	3 D/R or T, 370 E/R or	Q,
596 MAG 3B	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	Z	sCD4-(rHXB2	murine
	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)]				Sprzo) comprex	
	 MAG 3B: Amino acid subs T, 370 E/R or Q, 381 E/P, 3 	titutions that re 84 Y/E, 421 K	MAG 3B: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R or A or G, 262 N/T, 368 D/R or T, 370 E/R or Q, 381 E/P, 384 Y/E, 421 K/L, 475 M/S, 477 D/V [Kang et al.(1994)]	Y, 257 T/R or A or al.(1994)]	or G, 262 N/T, 368 D/R	Cor
597 MAG 12B	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2	murine
	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)] NOTES:				gp120)-complex	
	 MAG 12B: Amino acid substitutions that reduce binding 477 D/V – weak neutralization of IIIB [Kang et al.(1994)] 	ostitutions that ion of IIIB [Ka	MAG 12B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 477 D/V – weak neutralization of IIIB [Kang et al.(1994)]	[/R, 368 D/R or]	f, 370 E/R or Q, 384 Y	/E,

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
598 830D	gp120(CD4BS dis) References: [Wyatt et al.(1998)] NOTES:	gp120(dis)]	DISCONTINUOUS	T		
	 830D: Summary of the implications of the crystal is known about mutations that reduce NAb binding interference with CD4 binding [Wyatt et al.(1998)] 	lications of the nat reduce NAb ing [Wyatt et al	830D: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt et al.(1998)]	gp120 bound of neutralizati	to CD4 and 17b with won by CD4BS Ab is dir	hat
599 MAG 29B	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	T	sCD4-(rHXB2 gp120)-complex	murine
	 Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)] NOTES: MAG 29B: Amino acid substitutions that reduce binding 10 fold: 2 386 N/Q, 421 K/L – weak neutralization of IIIB [Kang et al.(1994)] 	stitutions that r	nor: C. Y. Kang, IDEC Inc & Frences: [Kang et al.(1994)] TES: MAG 29B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R 386 N/Q, 421 K/L – weak neutralization of IIIB [Kang et al.(1994)]		or T, 370 E/R or Q, 384 Y/E	/́Е,
600 120-1B1	gp120(CD4BS dis) Donor: Virus Testing Systems Corp., Houston, TX References: [Watkins et al.(1993)]	Corp., Houston,	DISCONTINUOUS TX	T		human
	• 120-1B1: A neutralization e neutralizing sera – 120-1B1	scape mutant (F	120-1B1: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – 120-1B1 was not affected by this mutation [Watkins et al.(1993)]	growth of HXB al.(1993)]	2 in the presence of broa	dly
601 MAG 6B	gp120(dis)	gp120(dis)	DISCONTINUOUS	Z	sCD4-(rHXB2 gp120)-complex	murine
	Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)] NOTES: • MAG 6B: Amino acid subst T. 370 E/R or O. 381 E/P. 38	titutions that res	nor: C. Y. Kang, IDEC Inc errences: [Kang et al.(1994)] TIES: MAG 6B: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R or T. 370 E/R or O. 381 E/P. 384 Y/E. 421 K/L. 475 M/S. 477 D/V [Kang et al.(1994)]		G or A, 262 N/T, 368 D/R or	or
602 P43110	gp120(dis) gp120(dis) DISCONTINUOUS Donor: Advanced Biosciences (Kensington, MD) References: [di Marzo Veronese et al.(1992), VanCott et al.(1995)]	gp120(dis) (Kensington, M e et al.(1992), V	DISCONTINUOUS (D) VanCott et al.(1995)]		unk	
	• P43110: Does not recognize	ed denatured fo	• P43110: Does not recognized denatured form of the gp120 protein [VanCott et al.(1995)]	ott et al.(1995)		

	603 17b	MAb ID
Ponor: Advanced Biosciences (Kensington, MD) References: [Thali et al.(1993), Moore et al.(1993c), Thali et al.(1994), Beretta & Dalgleish(1994), Wyatt et al.(1995) Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Wu et al.(1996), Trkola et al.(1996a) Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Weinberg et al.(1997), Ditzel et al.(1997), Cao et al.(1997), Wyat	gp120(CD4i dis)	Location
s (Kensington 3), Moore et al loore & Sodro(1997), Li et	gp120(dis)	WEAU Sequence
, MD) l.(1993c), Thali et al.(1994), sski(1996), Poignard et al.(al.(1997), Weinberg et al.(19	gp120(dis) DISCONTINUOUS	Sequence
Beretta & Dalgle 1996a), Wu et al. 97), Ditzel et al.(1	L P (weak)	Neutralizing
ish(1994), Wyatt et al. (1996), Trkola et al. (1997), Cao et al.(1997)	HIV-1 infection	Immunogen
l.(1995), (1996a),), Wyatt	human	Species(Isotype)

NOTES:

Sullivan et al.(1998), Binley et al.(1998)]

• 17b: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs

et al.(1997), Parren et al.(1997b), Kwong et al.(1998), Wyatt et al.(1998), Moore & Binley(1998), Rizzuto et al.(1998),

- 17b: Epitope is better exposed upon CD4 binding to gp120 competes with 15e and 21h, anti-CD4 binding site sensitivity to neutralization [Thali et al.(1993)] MAbs – 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 433 A/L, 438 P/R and 475 M/S confer decreased
- 17b: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding of 17b [Moore et al.(1993c)]
- 17b: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d,

21h and 15e) [Thali et al.(1994)]

- 17b: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 17b in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 - similar effect observed for 48d and A32 [Wyatt
- 17b: Binds with higher affinity to monomer and oligomer, slow association rate, poor neutralization of lab strain this is in contrast to 48d, which has very different kinetics [Sattentau & Moore(1995)]
- 17b: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) anti-V3 MAb 5G11 enhances binding, as do C1-C4 discontinuous epitopes A32 and 2/11c - enhances binding of some anti-V2 MAbs [Moore & Sodroski(1996)]
- 17b: Binding did not result in significant gp120 dissociation from virion, in contrast to 48d, although the the gp41 epitope of MAb 50-69 was exposed [Poignard et al.(1996a)]
- 17b: MIP-1 α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 binding of 17b blocks this inhibition [Wu et al.(1996)]
- 17b: Neutralizes JR-FL inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola
- 17b: A low avidity antibody as assessed by urea elution
- was not present, 17b only bound monomer [Fouts et al.(1997)] oligomeric Env binding - 17b bound monomer, oligomer, and neutralized JRFL in the presence of sCD4, but if sCD4 17b: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with
- 17b has synergistic response in combination with anti-V3 MAb 694/98-D [Li et al.(1997)] 17b: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env
- 17b: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the CanOA V3 peptide, suggesting CanOA V3 is a conformer that mimics the 48d - it does not bind to 17b, distinguishing the epitopes [Weinberg et al.(1997)]

- 17b: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to a CD4BS MAb F105 or sCD4 [Cao et al.(1997)]
- 17b: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding in conjunction with amino acids 31-93 in C1, but binding was restored in the presence of sCD4 [Wyatt et al.(1997)] partial reexposure if sCD4 was bound - could not bind to HXBc2 gp120 if the 19 C-term amino acids were deleted
- 17b: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- on 17b and basic on gp120 [Kwong et al.(1998)] chain - the center of the binding region has hydrophobic interactions, and the periphery charge interactions, acidic V1/V2 stem – the contact area is small for an Ab-antigen interactive surface, and dominated in the Ab by the heavy binds to the "bridging sheet" of gp120, an antiparallel β sheet region, contacting residues from the C4 region and the 17b: 17b FAb was co-crystallized with a gp120 core and CD4, and it's binding site can be directly visualized – 17b
- 17b: Summary of the implications of the crystal structure of a gp120 core bound to CD4 and 17b, combined with what may mask the CD4i Ab binding site, and that the V2 loop may be repositioned upon CD4 binding [Wyatt et al.(1998)] which may be occupied by the V3 loop in a complete gp120 molecule – the authors propose that the V2 and V3 loops gp120 are mostly in the heavy chain of the Ab, and there is a gap between 17b's light chain and the partial gp120 residues in gp120 that contact 17b are 202T and 434M – the contact points for 17b with the crystallized incomplete 419, I 420, K 421, Q 422, I 423, W 427, Y 435, P 438, M 475 of HXBc2 (IIIB) reduce binding – the only variable with chemokine receptor binding − mutations in 88N, 117K, 121K, 256S, 257T, N262, △ V3, E370, E381, F 382, R is known about mutations that reduce NAb binding to gp120 - probable mechanism of neutralization is interference
- 17b: Moore and Binley provide a commentary on the papers by [Rizzuto et al.(1998)], [Wyatt et al.(1998)] and bound virus, thus making it a poor NAb for primary isolates [Moore & Binley(1998)] that CD4 needs to bind to gp120 first to make the 17b epitope accessible and it may be stericly blocked in the CD4 [Kwong et al. (1998)] – they point out 17b shares binding elements in gp120 with chemokine receptor molecules, and
- chemokine receptors, supporting a common region in gp120 in chemokine-receptor interaction [Rizzuto et al.(1998)] 419, I/R 420, Q/L 422, Y/S 435, I/S 423, K/D 121 and K/D 421-17b can neutralize HIV-1 strains that use different interaction and interaction of gp120 and CCR5 – mutations in residues that reduced 17b by \geq 70% binding were R/D 17b: Site directed mutagenesis of a WU2 protein with the V1-V2 loops deleted revealed key residues for 17b-gp120
- 17b: sCD4 induces 17b binding in primary isolates and TCLA strains amino acids that reduce the efficiency of conformation [Sullivan et al.(1998)] range of temperatures, consistent with the energy of CD4 binding being sufficient to drive the V1/V2 loop into a new probably weak due to poor exposure of the epitope – 17b epitope exposure upon sCD4 binding can occur over a wide binding were determined and found also to compromise syncytia formation and viral entry - V1V2 deletion or sCD4 binding can expose the 17b epitope for both HXBc2 and macrophage tropic YU2 – neutralizing potency of 17b is
- structure closely approximating full length folded monomer CD4i MAbs 17b and 48d bound better to the deleted deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a 17b: A panel of MAbs was shown to bind with similar or greater affinity and similar competition profiles to a protein than to wild type [Binley et al.(1998)]

MAb ID 48d	
Location WEAU Sequence Neutralizing gp120(CD4i dis) gp120(dis) DISCONTINUOUS L P (weak) Donor: J. Robinson, University of Connecticut, Storrs References: [Thali et al.(1993), Moore & Ho(1993), Moore et al.(1993c), Thali et al.	
WEAU gp120(dis) tiy of Connect 93), Moore &	
WEAU Sequence gp120(dis) DISCONTINUOUS y of Connecticut, Storrs 3), Moore & Ho(1993), Moore et al.(1993)	
Neutralizing L P (weak) 93c), Thali et al	
HIV-1 infection hu L(1994), Moore et al.(1994b	
Species(Isotype) human($\operatorname{IgG}_{1\kappa}$) (1994b),	

NOTES

• 48d: Also called 4.8d and 4.8D

et al.(1998), Yang et al.(1998), Binley et al.(1998)]

et al. (1997), Wyatt et al. (1997), Parren et al. (1997b), Wyatt et al. (1998), Mondor et al. (1998), Parren et al. (1998), Sullivan et al.(1996a), Trkola et al.(1996a), Binley et al.(1997), Li et al.(1997), Weinberg et al.(1997), Lee et al.(1997), Ugolini D'Souza et al. (1995), Sattentau (1995), Wyatt et al. (1995), Sattentau & Moore (1995), Moore & Sodroski (1996), Poignard

- 48d: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs
- 48d: Epitope is better exposed upon CD4 binding to gp120 competes with ICR 39.13, 15e and 21h, anti-CD4 sensitivity to neutralization [Thali et al.(1993)] binding site MAbs - inhibited by anti-CD4BS MAb ICR 39.13g and linear anti-C4 MAbs G3-42 and G3-508 - 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 421 K/L, 433A/L, 438 P/R and 475 M/S confer decreased
- 48d: Called 4.8d Neutralizes IIIB reactive with SF-2 gp120 does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- of 17b [Moore et al.(1993c)] 48d: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding
- 48d: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 21h, 15e and 17b) [Thali et al.(1994)]
- 48d: Poor cross-reactivity with gp120 from most clades [Moore et al.(1994b)]
- 48d: Called 4.8D Found to neutralize MN, but not JRCSF, two B subtype primary isolates, or a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs[D'Souza et al.(1995)]
- 48d: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence of sCD4 et al.(1995)] involves the V1/V2 loops, with more significant involvement of V2 - similar effect observed for 17b and A32 [Wyatt
- 48d: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau et al.(1995)]
- 48d: Binds with similar affinity to monomer and oligomer, moderate association rate, potent neutralization this is in contrast to 17b, which has very different kinetics [Sattentau & Moore(1995)]
- 48d: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) anti-C1-C4 discontinuous epitope MAbs A32 and 2/11c enhance binding - reciprocal enhanced binding with some anti-V2 MAbs [Moore & Sodroski(1996)]
- 48d: Binding resulted in gp120 dissociation from virion, mimicking sCD4, and exposure of the gp41 epitope of MAb 50-69, in contrast to CD4BS MAbs [Poignard et al.(1996a)]

- 48d: Neutralizes JR-FL slightly inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]
- 48d: A low avidity antibody as assessed by urea elution
- 48d: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env - all Ab combinations tested showed synergistic neutralization - 48d has synergistic response with MAbs 694/98-D (anti-V3) and F105 [Li et al.(1997)]
- 48d: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the Can0A V3 peptide, suggesting Can0A V3 is a conformer that mimics the 48d, (but not 17b), epitope [Weinberg et al.(1997)]
- 48d: Prefers CD4-gp120 complex to gp120 alone, but does not enhance fusion, in contrast to MAb CG10, in fact it inhibits syncytium formation [Lee et al.(1997)]
- showed some correlation except 2F5) [Ugolini et al.(1997)] 48d: Viral binding inhibition by 48d was strongly correlated with neutralization (all other neutralizing MAbs tested
- 48d: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt et al.(1997)]
- 48d: Neutralizes TCLA strains, but not primary isolates [Parren et al.(1997b)]
- 48d: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is chemokine receptor binding - CD4 binding increases exposure of epitope due to V2 loop movement - 88N, 117K, 475 mutations in HXBc2 (IIIB) decrease binding [Wyatt et al.(1998)] known about mutations that reduce NAb binding – probable mechanism of neutralization of 48d is interference with 121K, 256S, 257T, N262, delta V3, E370, E381, F 382, R 419, I 420, K 421, Q 422, I 423, W 427, Y 435, P 438, M
- 48d: Inhibits binding of Hx 10 to both CD4 positive and CD4 negative HeLa cells [Mondor et al.(1998)]
- suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope 48d: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors [Parren et al.(1998)]
- 48d: CD4i MAbs 17b and 48d compete with MAb CG10, and the binding sites may overlap MAb A32 enhances binding of 17b, 48d and CG10 [Sullivan et al.(1998)]
- 48d: A neutralization assay was developed based on heminested PCR amplification of the LTR (HNPCR) LTRon tests with 6 MAbs and 5 isolates [Yang et al.(1998)] HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based
- 48d: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a protein than to wild type [Binley et al.(1998)] structure closely approximating full length folded monomer - CD4i MAbs 17b and 48d bound better to the deleted deglycosylated or variable loop deleted core gp120 protein (\Delta V1, V2, and V3), thus such a core protein produces a
- 48d: NIH AIDS Research and Reference Reagent Program: 1756

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
605 A32	gp120(CD4i C1-C4 dis)	gp120(dis)	DISCONTINUOUS	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_1)$
	Ponor: J. Robinson, Tulane University, LA References: [Moore et al.(1994b), Wyatt et al.(1995), Moore & Ho(1995), Moore & Sodroski(1996), Wu et al.(1996), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997), Burton & Montefiori(1997), Wyatt et al.(1997), Boots et al.(1997), Parren et al.(1997b), Sullivan et al.(1998), Binley et al.(1998)] NOTES:	uversity, LA 4b), Wyatt et a al.(1997), Foo), Sullivan et a	l.(1995), Moore & Ho(1995) uts et al.(1997), Burton & P l.(1998), Binley et al.(1998)]	, Moore & Sodros Montefiori(1997),	ki(1996), Wu et al.(1 Wyatt et al.(1997), 1	996), 3oots
	• A32: Reacted with virtually known [Moore et al.(1994b)]	y every gp120)]	A32: Reacted with virtually every gp120 monomer of every clade tested, most conserved gp120 monomer epitope known [Moore et al.(1994b)]	ed, most conserve	d gp120 monomer ep	и́tope
	 A32: Epitope is better exp studies using a V1/V2 delet V1/V2 loops, with more sign 	osed upon CDaion mutant den	A32: Epitope is better exposed upon CD4 binding to gp120 – binding of A32 enhances binding of 48d and 17b – studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 [Wvatt et al.(1995)]	of A32 enhances iing of 48d in the p	binding of 48d and bresence sCD4 involve	17b – es the
	 A32: Review: epitope is di A32: Reciprocal inhibition 	stinct from CD of binding of a	A32: Review: epitope is distinct from CD4BS MAbs, 48d and 17b, and 2G12 [Moore & Ho(1995)] A32: Reciprocal inhibition of binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of anti-C1, -C5, -C4, -V3 and -C1, -C	d 2G12 [Moore & ti-CD4 binding si	Ho(1995)] te MAbs – induces bij	nding
	 are unique among known h A32: Not neutralizing – bii hi, ihited by cm120 cCD4 a 	uman and rode nds domains th	are unique among known human and rodent MAbs [Moore & Sodroski(1996)] A32: Not neutralizing – binds domains that interact with gp41 – MIP-1 α binding to CCR-5 expressing cells can be inhibited by cm120 sCDA and binding of A32 does not block this inhibition (What all (1996))	[(1996)] 1α binding to CCI	R-5 expressing cells c	an be
	 A32: Does not neuranze JK-FL, or any suam suongry MIP-1/β-CCR-5 competition study [Trkola et al.(1996a)] A32: A low guidity optibody of occorded by proposition 	n study [Trkola	A32: Does not neutralize JN-rL, or any strain strongty – partial infliction of gp120 interaction with CCN-3 in a MIP-1\beta-CCR-5 competition study [Trkola et al.(1996a)] A32: A lear avidity antibody as assessed by upon alution	non or gp 120 m	TETACHOII WILL CCK-	o III a
	 A32: Study shows neutralization is not pred oligomeric env binding – A32 bound monor A32: Review (Burton & Montafori/1007)1 	zation is not pre	A32: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – A32 bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]	RFL monomeric g _l or neutralize JRFI	o120, but is associated [Fouts et al.(1997)]	with
	 A32: Binds efficiently to specification binding [Wyatt et al.(1997)] 	sgp120 but noi	A32: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt et al.(1997)]	esting its gp120 e	pitope is blocked by	gp41
	 A32: Does not neutralize T A37: Ahs that recognize of 	CLA strains or	A32: Does not neutralize TCLA strains or primary isolates [Parren et al.(1997b)] A37: Abs that recognize discontinuous enitones can identify mimotones from a phage pentide display library —	վ.(1997b)] nnes from a nhao	s pentide display lihr	VIR
	A32 has a unique epitope recognized, with a consens	involving most	A32 has a unique epitope involving mostly C2 but C1 and C4 contribute – six quite variable phage inserts were recognized, with a consensus of LPWYN – a central Trp was the most conserved element, consistent with W427	bute – six quite v st conserved elem	ariable phage inserts ent, consistent with V	were V427
	 being an important residue for binding gp120 [Boots et al.(1997)] A32: Enhances binding of CD4i MAbs 17b and 48d, and a MAb ge 	for binding gp CD4i MAbs 17	being an important residue for binding gp120 [Boots et al.(1997)] A32: Enhances binding of CD4i MAbs 17b and 48d, and a MAb generated in response to gp120-CD4 complex CG10	ted in response to a	gp120-CD4 complex (7G10

A32: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces

a structure closely approximating full length folded monomer [Binley et al.(1998)]

MAb ID L	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
606 2/11c g	gp120(C1-C4 dis) gp120(dis) Donor: J. Robinson, Tulane University, LA	gp120(dis) versity, LA	DISCONTINUOUS		HIV-1 infection	human
607 N70-2.3a	NOTES: NOTES: 2/11c: Also called 211c, 2.11c, 211/c and 2-11c 1/11c: Inhibits binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – ind and CD4i MAbs (48d and 17b) – similar reactivity pattern to A32, but less cross-reac and 211/c are unique among known human and rodent MAbs [Moore & Sodroski(199) 2/11c: Called 211c – does not neutralize JR-FL nor block gp120 interaction with competition study [Trkola et al.(1996a)] 2/11c: Study shows neutralization is not predicted by MAb binding to JRFL monorr with oligomeric env binding – 2/11c bound monomer, did not bind oligomer or neutral 2/11c: Called 2.11c – One of 14 human MAbs tested for ability to neutralize a chimeric HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration 2/11c: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction deleted [Wyatt et al.(1997)] 2/11c: Called 2.11/c – a panel of MAbs were shown to bind with similar or greater aff profiles to a deglycosylated or variable loop deleted core gp120 protein (\(\Delta \text{ V1}, \text{ V2}, and produces a structure closely approximating full length folded monomer [Binley et al.(gp120/272-509 dis) gp120/(272-509 dis) NOTES: NOTES: NOTES: References: [Robinson et al.(1990), Takeda et al.(1992)] NOTES: NOTES: References: [Robinson et al.(1990), Takeda et al.(1992)]	1c, 211/c and 2 ti-C1, -C5, -C4 7b) – similar r known human not neutralize al.(1996a)] by as assessed I zation is not p -2/11c bound 14 human MA alization could sgp120 but not [XBc2 gp120 iz [XBc2 gp120 iz [XBc2 gp120 iz el of MAbs we or variable loop approximating gp120(dis) versity, LA 90), Takeda et Robinson et al.(revences: [Moore & Sodroski(1996), Tikola et al.(1996a), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Binley et al.(1997), Binley et al.(1998)] TES: 2/11c: Also called 2/11c, 2/11c, and 2/11c 2/11c: Inhibits binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of some anti-V2 and CD4 i MAbs (488 and 17b) – similar reactivity pattern to A32, but less cross-reactive and lower affinity – A32 and 2/11c are unique among known human and rodent MAbs [Moore & Sodroski(1996)] 2/11c: Called 2/11c – does not neutralize IR-FL nor block gp120 interaction with CCR-5 in a MIP-1/3-CCR-5 competition study [Tikola et al.(1996a)] 2/11c: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – 2/11c bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)] 2/11c: Study shows neutralization could not be achieved at a maximal concentration of 67 µg/ml [Li et al.(1997)] 2/11c: Called 2/11c – One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 67 µg/ml [Li et al.(1997)] 2/11c: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-74, are deleted [Wyatt et al.(1997)] 2/11c: Called 2/11c – a panel of MAbs were shown to bind with similar or greater affinity and similar competition produces a structure closely approximating full length folded monomer [Binley et al.(1998)] 2/12c. Broad reactivity [Robinson et al.(1992)] TREs: N HIV-1 infection N HIV-1 infection N HIV-1 infection N HIV-1 infection of HV-1 infection – binds a conformational site in the carboxyl half of gp120, distinct from 1.5c [Takeda et al.(1992)]	ie MAbs – inducess cross-reactive Sodroski(1996)] raction with CC search or neutralize ze a chimeric SH concentration of ting its gp120 e n conjunction with V1, V2, and V. Binley et al.(199 N	et al.(1997), Li et al.(1997), huces binding of some anti-V2 tive and lower affinity – A32 (6)] CCR-5 in a MIP-1\beta-CCR-5 eric gp120, but is associated lize JRFL [Fouts et al.(1997)] SHIV-vpu+, which expressed of 67 \(\text{µg/ml}\) [Li et al.(1997)] SHIV-ppe is blocked by gp41 with C1 positions 31-74, are finity and similar competition 1 V3), thus such a core protein 1998)] HIV-1 infection	97), 432 432 47)] seed 97)] y41 are tion tein human(IgG1) half
608 6E10 gp120 (dis)	or Spino, custimer mom income	L rancour or m.	DISCONTINUOUS			

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
609 C31	gp120(unknown) gp120 Donor: ? References: [Boyer et al.(1991)] NOTES: C31: Broadly reactive group specific —	gp120	? N high yield cultivation of human MAb [Boyer	N 1 MAb [Boyer et	HIV-1 infection et al.(1991)]	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
610 P5-3	gp120(unknown) gp120 ? Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Robinson Jr. et al.(1990a), Pincus et al.(1991)] NOTES: P5-3: No enhancing activity for HIV-1 IIIB [Robinson Jr. et al.(1995)] P5-3: Poor immunotoxin activity when coupled to RAC – isotype P5-3: NIH AIDS Research and Reference Reagent Program: 378	gp120 Ichi Matsumot al.(1990a), Pin ity for HIV-1 I activity when h and Referen	20(unknown) gp120 ? nor: Evan Hersh and Yoh-Ichi Matsumoto erences: [Robinson Jr. et al.(1990a), Pincus et al.(1991)] TES: P5-3: No enhancing activity for HIV-1 IIIB [Robinson Jr. et al.(1990a)] P5-3: Poor immunotoxin activity when coupled to RAC – isotype specified as: IgG _{3\(\lambda\)} [Pincus et al.(1991)] P5-3: NIH AIDS Research and Reference Reagent Program: 378)] cified as: IgG _{3λ} [HIV-1 infection [Pincus et al.(1991)]	$\mathrm{human}(\mathrm{IgG}_{1\lambda})$
611 BAT401	gp120(unknown) Donor: ? References: [Fung et al.(1987)]	gp120	?	L	Intact IIIB	$\operatorname{murine}(\operatorname{IgG}_1)$
612 BAT267	gp120(unknown) Donor: ? References: [Fung et al.(1987)]	gp120	.?	L	Inact IIIB	$\operatorname{murine}(\operatorname{IgG}_1)$
613 BAT509	gp120(unknown) Donor: ? References: [Fung et al.(1987)]	gp120	?	L	Inact IIIB	$\operatorname{murine}(\operatorname{IgG}_1)$
614 13.10	gp120(unknown) gp120 Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Lake et al.(1989), Moran et al.(NOTES: • 13.10: Also called No. 13 • 13.10: First HIV-1 specific human-mous et al.(1989)] • 13.10: Heavy (V_H I) and light (V_λ II) chair et al.(1993)] • 13.10: 13.10 is V_H I – V-region heavy chave the value of value of the value of t	gp120 [chi Matsumot], Moran et al.), Moran et al. ic human-mou ight $(V_{\lambda}II)$ cha ight $(V_{\lambda}II)$ cha egion heavy cl IV infected in ch and Referer	gp120(unknown) gp120 ? N HIV-1 infection Ponor: Evan Hersh and Yoh-Ichi Matsumoto References: [Lake et al.(1989), Moran et al.(1993), Wisnewski et al.(1996)] NOTES: • 13.10: Also called No. 13 • 13.10: First HIV-1 specific human-mouse hybridoma that produces a MAb that binds to gp120 and gp160 [Lake et al.(1989)] • 13.10: Heavy (V_H I) and light (V_{λ} II) chain sequenced – no enhancing or neutralizing activity – called No. 13 [Moran et al.(1993)] • 13.10: 13.10 is V_H 1 – V-region heavy chain usage was examined and a bias of enhanced V_H 1 and V_H 4, and reduced V_H 3, was noted among HIV infected individuals [Wisnewski et al.(1996)] • 13.10: NIH AIDS Research and Reference Reagent Program: 377	N MAb that bind: r neutralizing act bias of enhancec 96)]	HIV-1 infection $^{\rm H}$ has to gp120 and gp160 [Lake activity – called No. 13 [Moran sed V_H 1 and V_H 4, and reduced	$ ext{human}(ext{Ig}G_{1\lambda})$ _ake oran

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615 F285 Env(unknown) Donor: Evan Hersh References: [Wisne NOTES: • F285: F285 is V_H3 , was noted	Env(unknown) gp120 ? HIV-1 infection Ponor: Evan Hersh and Yoh-Ichi Matsumoto References: [Wisnewski et al.(1995), Wisnewski et al.(1996)] NOTES: • F285: F285 is $V_H 1 - V$ -region heavy chain usage was examined and a bias of enhanced $V_H 1$ and $V_H 4$, and reduced $V_H 3$, was noted among HIV infected individuals [Wisnewski et al.(1996)]	HIV-1 infection $\operatorname{Ced} \operatorname{V}_H 1$ and $\operatorname{V}_H 4$, and $\operatorname{red} \operatorname{V}_H 1$	$ ext{human}(ext{IgG}_1)$ $ ext{iced}$
616 HBW4 gp120(unknown IIIB) Donor: Evan Hersh a References: [Moran of the content of the cont	gp120(unknown IIIB) gp120 ? HIV-1 infection Ponor: Evan Hersh and Yoh-Ichi Matsumoto References: [Moran et al.(1993), Wisnewski et al.(1995), Wisnewski et al.(1996)] NOTES: • HBW4: Heavy (V_H II) and light (V_{λ} II) chain sequenced [Moran et al.(1993)] • HBW4: HBW4 is V_H 2 – V-region heavy chain usage was examined and a bias of enhanced V_H 1 and V_H 4, and reduced V_H 3, was noted among HIV infected individuals [Wisnewski et al.(1996)]	HIV-1 infection ${ m ^2}$ enhanced ${ m V}_H{ m 1}$ and ${ m V}_H{ m 4}$,	$human(IgG_{1\lambda})$ and
617 multiple gp120(unknown) Fabs	gp120 ?	HIV-1 infection	human
Donor: Evan Hersh and Yoh-Ichi References: [Burton et al.(1991)] NOTES: • A panel of anti-gp120 Fabs w bone marrow from an asympt	 Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Burton et al.(1991)] NOTES: A panel of anti-gp120 Fabs was generated by antigen selection from a random combinatorial library prepared from bone marrow from an asymptomatic individual [Burton et al.(1991)] 	binatorial library prepared f	rom
618 multiple gp120(unknown) MAbs	gp120 ?	gp120 complexed with MAb M77	murine
Donor: Evan Hersh and Yoh-Ichi M References: [Denisova et al.(1996)] NOTES: • When anti-V3 MAb M77 was epitopes, as well as an array of I are mentioned elsewhere in this GV2H4, GV6E6, GV1F7, GV GV1E10, GV5E3, GV5B9, GV	 Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Denisova et al.(1996)] NOTES: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes, as well as an array of MAbs to discontinuous epitope – 10 of 36 MAbs were mapped to linear epitopes and are mentioned elsewhere in this database, the others are: GV5H1, GV4D5, GV4G10, GV1A8, GV10H5, GV8E11, GV2H4, GV6E6, GV1F7, GV1G9, GV4G5, GV6B12, GV1E8, GV2B7, GV1B11, GV6H5, GV6G2, GV6B5, GV1E10, GV5E3, GV5B9, GV5F4, GV6G4, GV1A12, GV5C11, GV6B6, GV3C10 [Denisova et al.(1996)] 	mulated many MAbs to linear e mapped to linear epitopes and 0, GV1A8, GV10H5, GV8E11 11, GV6H5, GV6G2, GV6B5) [Denisova et al.(1996)]	near and 311, 3B5,

MAb ID Location	623 polyclonal gp120	Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Wagner et al.(1998)]	NOTES: • A VLP is a non-infe VLPs bound to eithe case, and Ab respor whole gp120, not V challenge with SHIV	624 polyclonal gp120(IIIB)	Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Wagner et al.(1998)] NOTES:	▶ DNA vaccinations of	with Th1-like secret et al.(1997)]	with Th1-like secret et al.(1997)] 625 polyclonal gp120	polyclonal gp1 Dor Ref NO
WEAU	gp120	Yoh-Ichi Matsumoto al.(1998)]	ectious virus-like par er gp120 or V3+CD2 se to gag and gp120 3+CD4 – despite the chimeric challenge	gp120	Yoh-Ichi Matsumoto al.(1998)]		î BALBc mice with \imath ion of γ interferon ar	ion of γ interferon arginal gp120	f BALBc mice with a fon of γ interferon ar gp120 Yoh-Ichi Matsumott [1997)]
Sequence		9	A VLP is a non-infectious virus-like particle self-assembled from HIV Pr55 gag – macaques were immunized with VLPs bound to either gp120 or V3+CD4 linear domains – gag and env CTL specific CTL were stimulated in each case, and Ab response to gag and gp120 and was elicited, but the gp120 neutralizing response occurred only with whole gp120, not V3+CD4 – despite the CTL and Ab response, immunized macaques were infected by intervenous challenge with SHIV chimeric challenge stock [Wagner et al.(1998)]				nor: Evan Hersh and Yoh-Ichi Matsumoto erences: [Wagner et al.(1998)] TES: TES: DNA vaccinations of BALBc mice with a gp120 or gp160 DNA vaccine elicited a strong T cell proliferative response with Th1-like secretion of γ interferon and IL-2, with little or no IL-4, as well as antigen specific gp120 Abs [Shiver et al.(1997)]	n gp120 or gp160 DNA vacc nd IL-2, with little or no IL-	1 gp120 or gp160 DNA vacc nd IL-2, with little or no IL-
Neutralizing	Y		IIV Pr55 gag – mac env CTL specific C gp120 neutralizing 1 nunized macaques v				ine elicited a strong 4, as well as antiger	ine elicited a strong 4, as well as antiger	ine elicited a strong 4, as well as antiger L
Immunogen	HIV-1 Pr55gag VLP with anchored gp120 or V3+CD4 linear domains	linear domains	aques were immunize TL were stimulated i esponse occurred only vere infected by inter		gp120 or gp160 DNA vaccine	gp120 or gp160 DNA vaccine	gp120 or gp160 DNA vaccine T cell proliferative re	gp120 or gp160 DNA vaccine T cell proliferative re 1 specific gp120 Abs [DNA gag/pol, vif, and CMN160 vaccine	gp120 or gp160 DNA vaccine T cell proliferative re 1 specific gp120 Abs [DNA gag/pol, vif, and CMN160 vaccine
Species(Isotype)	Macaca mulatta		xd with n each y with venous		murine	murine	murine sponse Shiver	murine sponse [Shiver murine	murine sponse Shiver murine