Table 10:  $\mathbf{gp41}$ 

			ğ			
MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
626 5F3	gp41(526-543 BH10)	gp41(15-33)	AAGSTMGAASMTLTVQ- ARQ	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria <b>References:</b> [Buchacher et al.(1994)] <b>NOTES:</b>	l. Microbiol., Vie 1994)]	nna, Austria			
	• 5F3: Human MAb gener et al.(1994)]	ated by electrofi	Human MAb generated by electrofusion of PBL from HIV-1+ volunteers 1994)]		with CB-F7 cells [Buchacher	er
627 25C2	gp41(526-543 BH10)	gp41(15-33)	AAGSTMGAASMTLTVQ-ARQ	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX <b>References:</b> [Buchacher et al.(1992), Buchacher et al.(1994), Sattentau et al.(1995)] <b>NOTES:</b>	l. Microbiol., Vie 1992), Buchache	nna, Austria and Viral Testing r et al.(1994), Sattentau et al.(1	Systems, Houst 995)]	.on, TX	
	<ul> <li>25C2: Human MAb generated by electrofusion of PBL fro and monomeric gp41, and gp160 [Buchacher et al.(1994)]</li> <li>25C2: Called IAM 41-25C2 – Binding domain overlaps binding is enhanced by sCD4 – binding region defined as:</li> </ul>	ted by electrofus gp160 [Buchache C2 – Binding dor D4 – binding regi	25C2: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells – binds oligomeric and monomeric gp41, and gp160 [Buchacher et al.(1994)] 25C2: Called IAM 41-25C2 – Binding domain overlaps sites that are critical for gp120-gp41 association gStM – binding is enhanced by sCD4 – binding region defined as: gp41(21-38 BH10) [Sattentau et al.(1995)]	teers with CB-Fical for gp120- 10) [Sattentau e	'B-F7 cells – binds oligomeric 120-gp41 association g <b>StM</b> – tau et al.(1995)]	I 6
628 24G3	gp41(526-543 BH10)	gp41(15-33)	AAGSTMGAASMTLTVQ-ARQ	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria <b>References:</b> [Buchacher et al.(1992), Buchacher et al.(1994)] <b>NOTES:</b>	l. Microbiol., Vie 1992), Buchache	nna, Austria r et al.(1994)]			
	• 24G3: Human MAb gene et al.(1994)]	rated by electron	24G3: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers et al.(1994)]		with CB-F7 cells [Buchacher	er
629 1A1	gp41(526-543 BH10)	gp41(15-33)	AAGSTMGAASMTLTVQ-ARQ	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria References: [Buchacher et al.(1994)]  NOTES:  1 A 1 · Human MAh generated using FRV transformation	l. Microbiol., Vie 1994)] red using FRV fr	nor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria erences: [Buchacher et al.(1994)] TES:	-1+ volunteers	(Ruchacher et al (1994))	
630 $\alpha$ (566-586)	gp41(566-586 BRU)	gp41(51-71)	AQQHLLQLTVWGIKQLQ- ARII		HIV-1 infection	human
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria <b>References:</b> [Poumbourios et al.(1992)]	l. Microbiol., Vie d.(1992)]	nna, Austria			

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
631 PC5009	gp41(577-596 BRU)	gp41(62-81)	GIKQLQARILAVERYLK- DOO		rgp160	murine
	<ul> <li>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria References: [Poumbourios et al.(1992)]</li> <li>NOTES:</li> <li>PC5009: Recognized only monomeric gp41 [Poumbourios et al.(1992)]</li> </ul>	ol. Microbiol., Vie al.(1992)] monomeric gp41	nna, Austria [Poumbourios et al.(1992)]			
632 polyclonal	gp41(577-596 BRU)	gp41(62-81)	GIKQLQARILAVERYLK- DOO		HIV-1 infection	human plasma
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria <b>References:</b> [Poumbourios et al.(1992)] <b>NOTES:</b>	ป. Microbiol., Vie al.(1992)]	nna, Austria			
	• $\alpha$ (577-596): Affinity puri	fied from HIV-1+ <sub>J</sub>	$\bullet$ $\alpha$ (577-596): Affinity purified from HIV-1+ plasma – preferentially bind oligomer [Poumbourios et al.(1992)]	gomer [Poumb	ourios et al.(1992)]	
633 polyclonal	gp41(583-604)	gp41(69-89)	RILAVERYLKDQQLLGI- WGCS	Z	desialylated HIV-1 gp160	rabbit sera
	<ul> <li>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria References: [Benjouad et al.(1993)]</li> <li>NOTES:</li> <li>MAbs raised against desialylated HIV-1 gp160 cross-reenitone in gn41 [Renjouad et al.(1993)]</li> </ul>	ol. Microbiol., Vie 1993)] alylated HIV-1 gp	<ul> <li>In the statinger, Inst. Appl. Microbiol., Vienna, Austria</li> <li>In the statinger, Inst. Appl. Microbiol., Vienna, Austria</li> <li>In the state of th</li></ul>		immunodominant conserved	, g
	داماسامسر	1 Ct m.(1775)]				
634 polyclonal	gp41(584-602) gp41(70-87) ILAVERYLKDQQLL  Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria  References: [Petrov et al.(1990)]  NOTES:  • Immunodominant and broadly reactive peptide [Petrov et al.(1990)]	gp41(70-87) bl. Microbiol., Vie 0)] adly reactive pepti	ILAVERYLKDQQLLGIWG nna, Austria de [Petrov et al.(1990)]	Z	HIV-1 infection	human sera
635 V10-9	gp41(586-620 IIIB)	gp41(70-103)	ILAVERYLKDQQLLGIW- GCSGKLICTTAVPWNAS	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria <b>References:</b> [Robinson Jr. et al.(1990a), Robinson Jr. et al.(1990b)]	ol. Microbiol., Vie al.(1990a), Robins	nna, Austria on Jr. et al.(1990b)]			
	• V10-9: Antibody dependent [Robinson Jr. et al.(1990a)]	nt enhancement (A )]	V10-9: Antibody dependent enhancement (ADE) of HIV-1 IIIB infectivity, synergistically enhanced by MAb 120-16 [Robinson Jr. et al.(1990a)]	synergistically	enhanced by MAb 120-1	6
	1					

• NO	Do Ref	638 41-6 gp4	Do Rei NO	637 polyclonal gp4	636 86 gp4  Doy Ret et a NO  •	MAb ID Loc
NOTES:  • 41-6: Poor cross-reactivity with HIV-2 peptide CAFRQVC – slightly more reactive with LGLIWG	<b>Donor:</b> Evan Hersh and Yoh-Ichi Matsumoto <b>References:</b> [Oldstone et al.(1991)]	gp41(598-609)	Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Shafferman et al.(1989)] NOTES:  • Immunogenic domain useful for diagnostics [Shafferman et al.(1989)]	gp41(74-94?)	<ul> <li>gp41(586-620 IIIB)</li> <li>gp41(69-103)</li> <li>RILAVERYLKDQQL WGCSGKLICTTAVP WGCSGKLICTTAVP WGCSGKLICTTAVP WGCSGKLICTTAVP WGCSGKLICTTAVP References: [Sugano et al.(1988), Robinson Jr. et al.(1990a), Robinso et al.(1993), Wisnewski et al.(1996), Mitchell et al.(1990a), Robinso et al.(1993), Wisnewski et al.(1996), Mitchell et al.(1998)]</li> <li>NOTES: <ul> <li>86: Also called No. 86</li> <li>86: Also called No. 86</li> <li>86: Reacts with gp41 and also reacted weakly with gp120 [Sugano et al.(1990a)]</li> <li>86: Reacts with gp41 and also reacted weakly with gp120 [Sugano et al.(1990a)]</li> <li>86: Peptide 586-620 blocks complement mediated ADE [Robinson et al.(1993)]</li> <li>86: Heavy (V<sub>H</sub>1) and light (V<sub>κ</sub>1) chain sequenced – enhancing activation but very different activity [Moran et al.(1993)]</li> <li>86: 86 is V<sub>H</sub>1 – V-region heavy chain usage was examined and a bias was noted among HIV infected individuals [Wisnewski et al.(1996)]</li> <li>86: Mutations in BH10 gp160, W596Y and T605A, as well as delegant GKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50-to date bind to the immunodominant region 579-613 [Mitchell et al. 66: NIH AIDS Research and Reference Reagent Program: 380</li> </ul> </li> </ul>	Location
y with HIV-2 pept	chi Matsumoto 991)]	gp41(88-94)	chi Matsumoto .(1989)] ful for diagnostics	gp41	gp41(69-103) chi Matsumoto >88), Robinson Jr. [1996), Mitchell et also reacted weakl nhancement (ADE is complement me ivity when coupled to (V <sub>K</sub> I) chain seque [Moran et al.(1993) neavy chain usage ected individuals [190] 160, W596Y and binding of enhanci ond Reference Rea,	WEAU
TES: 41-6: Poor cross-reactivity with HIV-2 peptide CAFRQVC – slightly more reactive		CSGKLIC	[Shafferman et al.(1989)]	ERYLKDQLLGIWGCSGK- LIC	pg41(586-620 IIIB)  gp41(69-103)  RILAVERYLKDQQLLGI- WGCSGKLICTTAVPWNAS  Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Sugano et al.(1988), Robinson Jr. et al.(1990a), Robinson Jr. et al.(1990b), Pincus et al.(1991), Moran et al.(1993), Wisnewski et al.(1996), Mitchell et al.(1998)]  NOTES:  • 86: Also called No. 86  • 86: Reacts with gp41 and also reacted weakly with gp120 [Sugano et al.(1988)]  • 86: Antibody dependent enhancement (ADE) of HIV-1 IIIB infectivity in the presence of complement [Robinson Jr. et al.(1990a)]  • 86: Poptide 586-620 blocks complement mediated ADE [Robinson Jr. et al.(1990b)]  • 86: Peptide 586-620 blocks complement mediated ADE [Robinson Jr. et al.(1990b)]  • 86: Heavy (V <sub>H</sub> 1) and light (V <sub>s</sub> 1) chain sequenced – enhancing activity – similar germline sequence to MAb S1-1, but very different activity [Moran et al.(1993)]  • 86: 86 is V <sub>H</sub> 1 – V-region heavy chain usage was examined and a bias of enhanced V <sub>H</sub> 1 and V <sub>H</sub> 4, and reduced V <sub>H</sub> 3, was noted among HIV infected individuals [Wisnewski et al.(1996)]  • 86: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605-609 (TTAVP) and 597-609 (GCS-GKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50-69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579-613 [Mitchell et al.(1998)]  • 86: NIH AIDS Research and Reference Reagent Program: 380	Sequence
re reactive with				·	S  et al.(1990b), Pi  988)]  he presence of c  l.(1990b)]  ted to be aa 579-  similar germline  hanced V <sub>H</sub> 1 and  hanced V <sub>H</sub> 1 and  of 605-609 (TTA  d 246-D – 5/6 en	Neutralizing
with LGLIWGCSGKLIC and	,	peptide LGLIWGC-SGKLIC (aa 598-609)		HIV-1 infection	HIV-1 infection b), Pincus et al.(1991), Moran e of complement [Robinson Jr. a 579-603 [Pincus et al.(1991)] rmline sequence to MAb S1-1, t1 and V <sub>H</sub> 4, and reduced V <sub>H</sub> 3, (TTAVP) and 597-609 (GCS-5/6 enhancing MAbs identified	Immunogen
pd		$murine(IgG_{2b})$		human sera	human( $\operatorname{IgG}_{1\kappa}$ ) an  Jr. Jr. 3, 3, e.d.	Species(Isotype)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
639 4	gp41(598-609)	gp41(88-94)	CSGKLIC		peptide LGLIWGC-SGKLIC (aa 598-609)	$murine(\mathrm{IgG}_{2b})$
	<b>Donor:</b> Evan Hersh and Yoh-Ichi Matsumoto <b>References:</b> [Oldstone et al.(1991)] <b>NOTES:</b>	Yoh-Ichi Matsumoto t al.(1991)]				
	<ul> <li>4: There is another MAb with this I</li> <li>4: Poor cross-reactivity with HIV-2 CAFRQVC [Oldstone et al.(1991)]</li> </ul>	MAb with this ID that rity with HIV-2 peptide e et al.(1991)]	4: There is another MAb with this ID that reacts with integrase [Oldstone et al.(1991), Bizub-Bender et al.(1994)] 4: Poor cross-reactivity with HIV-2 peptide CAFRQVC – slightly more reactive with longer HIV-2 peptide NSWG-CAFRQVC [Oldstone et al.(1991)]	one et al.(1991), Big e reactive with long	Bizub-Bender et al.(1994)] mger HIV-2 peptide NSWG	7"
640 75	gp41(598-609)	gp41(88-94)	CSGKLIC		peptide LGLIWGC-SGKLIC (aa 598-609)	rat(IgG)
	<b>Donor:</b> Evan Hersh and Yoh-Ichi Matsumoto <b>References:</b> [Oldstone et al.(1991)] <b>NOTES:</b>	Yoh-Ichi Matsumoto t al.(1991)]				
	<ul> <li>75: Poor cross-reactivity with HT CAFRQVC [Oldstone et al.(1991)]</li> </ul>	tivity with HIV-2 pep ne et al.(1991)]	75: Poor cross-reactivity with HIV-2 peptide CAFRQVC – more reactive with longer HIV-2 peptide NSWG-CAFRQVC [Oldstone et al.(1991)]	eactive with longe	r HIV-2 peptide NSWG	7
641 68.1	gp41(598-609)	gp41(88-94)	CSGKLIC		peptide LGLIWGC-SGKLIC (aa 598-609)	murine(IgM)
	<b>Donor:</b> Evan Hersh and Yoh-Ichi Matsumoto <b>References:</b> [Oldstone et al.(1991)] <b>NOTES:</b>	Yoh-Ichi Matsumoto t al.(1991)]				
	• 68.1: Cross-reactive and HIV-2 peptide N	68.1: Cross-reactive with HIV-2 peptide CAFRQVC – more re and HIV-2 peptide NSWGCAFRQVC [Oldstone et al.(1991)]	68.1: Cross-reactive with HIV-2 peptide CAFRQVC – more reactive with longer HIV-1 and HIV-2 peptide NSWGCAFRQVC [Oldstone et al.(1991)]		peptide LGLIWGCSGKLIC	
642 68.11	gp41(598-609)	gp41(88-94)	CSGKLIC		peptide LGLIWGC- SGKLIC (aa 598- 609)	murine(IgM)
	<b>Donor:</b> Evan Hersh and Yoh-Ichi Matsumoto <b>References:</b> [Oldstone et al.(1991)] <b>NOTES:</b>	Yoh-Ichi Matsumoto t al.(1991)]				
	• 68.11: Cross-reactive and HIV-2 peptide N	68.11: Cross-reactive with HIV-2 peptide CAFRQVC – more reand HIV-2 peptide NSWGCAFRQVC [Oldstone et al.(1991)]	68.11: Cross-reactive with HIV-2 peptide CAFRQVC – more reactive with longer HIV-1 and HIV-2 peptide NSWGCAFRQVC [Oldstone et al.(1991)]		peptide LGLIWGCSGKLIC	()

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
643 115.8	gp41(598-609)	gp41(82-94)	LGLIWGCSGKLIC		peptide LGLIWGC- SGKLIC (aa 598- 609)	murine(IgM)
	<b>Donor:</b> Evan Hersh and Yoh-Ichi Matsumoto <b>References:</b> [Oldstone et al.(1991)]	Ichi Matsumoto [991)]			007)	
	NOTES:  • 115.8: Poor reactivity w  CAFRQVC – disulfide bo	ith CSGKLIC – and between cyste	TES:  115.8: Poor reactivity with CSGKLIC – reacts well with longer HIV-2 peptide CAFRQVC – disulfide bond between cysteines required [Oldstone et al.(1991)]		NSWGCAFRQVC as well as	as
644 M-22	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide	$\mathrm{murine}(\mathrm{IgG}_{2b})$
	Donor: ? References: [Yamada et al.(1991)]	991)]			(aa 598-609)	
	<ul> <li>M-22: Strongest reaction</li> <li>[Yamada et al.(1991)]</li> </ul>	of 12 anti-HIV-1 g	M-22: Strongest reaction of 12 anti-HIV-1 gp41 MAbs to a cellular 43-kDa protein found in rat and human astrocytes [Yamada et al.(1991)]	protein found in	rat and human astrocyt	es
645 M-24	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide	$murine(IgG_1)$
	Donor: ? References: [Yamada et al.(1991)]	991)]			(aa 598-009)	
	<ul><li>M-24: Strongly reacted</li><li>[Yamada et al.(1991)]</li></ul>	with a cellular 43	M-24: Strongly reacted with a cellular 43-kDa protein found in rat and human astrocytes as well as with gp41 [Yamada et al.(1991)]	human astrocy	tes as well as with gp-	41
646 M-28	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide	$murine(IgG_1)$
	Donor: ? References: [Yamada et al.(1991)]	991)]			(44 398-009)	
	• M-28: Strongly reacted [Yamada et al.(1991)]	with a cellular 4:	M-28: Strongly reacted with a cellular 43-kDa protein found in rat and human astrocytes as well as with gp41 [Yamada et al.(1991)]	human astrocy	tes as well as with gp-	41
647 M-2	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide	$\operatorname{murine}(\operatorname{IgG}_{2b})$
	Donor: ? References: [Yamada et al.(1991)]	991)]			(aa 598-609)	
	<ul> <li>M-2: Strongly reacted winet al. (1991)]</li> </ul>	h a cellular 43-kL	M-2: Strongly reacted with a cellular 43-kDa protein found in rat and human astrocytes as well as with gp41 [Yamada et al.(1991)]	n astrocytes as w	ell as with gp41 [Yamac	da

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
648 M-11	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: ? References: [Yamada et al.(1991)] NOTES: • M-11: Strongly reacted with [Yamada et al.(1991)]	991)] with a cellular 43	or: ? erences: [Yamada et al.(1991)] TES: M-11: Strongly reacted with a cellular 43-kDa protein found in rat and human astrocytes as well as with gp41 [Yamada et al.(1991)]	d human astrocy	tes as well as with gp4	1
649 M-13	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	$\mathrm{murine}(\mathrm{IgG}_{2b})$
	Donor: ? References: [Yamada et al.(1991)]	991)]				
	• M-13: Reacted with a c et al.(1991)]	ellular 43-kDa pro	M-13: Reacted with a cellular 43-kDa protein found in rat and human astrocytes as et al.(1991)]		well as with gp41 [Yamada	ia
650 M-25	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	$murine(IgG_1)$
	Donor: ? References: [Yamada et al.(1991)] NOTES:	991)]				
	• M-25: Reacted with a c et al.(1991)]	ellular 43-kDa pro	M-25: Reacted with a cellular 43-kDa protein found in rat and human astrocytes as et al.(1991)]		well as with gp41 [Yamada	ົລ
651 M-1	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	$\mathrm{murine}(\mathrm{IgG}_{1or2b})$
	Donor: ? References: [Yamada et al.(1991)] NOTES:	991)]				
	• M-1: Unlike M-22, did n	ot react to 43-kDa	M-1: Unlike M-22, did not react to 43-kDa protein found in rat and human astrocytes	_	Yamada et al.(1991)]	
652 M-4	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	$\mathrm{murine}(\mathrm{IgG}_{2b})$
	Donor: ? References: [Yamada et al.(1991)] NOTES:	991)]		1		
	• M-4: Unlike M-22, did n	ot react to 43-kDa	M-4: Unlike M-22, did not react to 43-kDa protein found in rat and human astrocytes	_	Yamada et al.(1991)]	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
653 M-6	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	$murine(IgG_{2b})$
	<b>Donor:</b> ? <b>References:</b> [Yamada et al.(1991)] <b>NOTES:</b> • M-6: Unlike M-22, did not rea	91)] t react to 43-kDa	nor: ?  ferences: [Yamada et al.(1991)]  TES:  M-6: Unlike M-22, did not react to 43-kDa protein found in rat and human astrocytes [Yamada et al.(1991)]	astrocytes [Yar	nada et al.(1991)]	
654 M-29	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	$murine(IgG_1)$
	Donor: ? References: [Yamada et al.(1991)] NOTES: • M-29: Unlike M-22, did not re	91)] ot react to 43-kD	nor: ?  ferences: [Yamada et al.(1991)]  TES:  M-29: Unlike M-22, did not react to 43-kDa protein found in rat and human astrocytes [Yamada et al.(1991)]	n astrocytes [Ya	amada et al.(1991)]	
655 M-36	gp41(598-609)	gp41(83-94)	LGIWGCSGKLIC		HIV-1 gp41 peptide (aa 598-609)	$\mathrm{murine}(\mathrm{Ig}\mathrm{G}_1)$
	Donor: ? References: [Yamada et al.(1991)] NOTES: • M-36: Unlike M-22, did not re	91)] ot react to 43-kL	nor: ?  lerences: [Yamada et al.(1991)]  TES:  M-36: Unlike M-22, did not react to 43-kDa protein found in rat and human astrocytes [Yamada et al.(1991)]	n astrocytes [Ya	amada et al.(1991)]	
656 1B8.env	gp41(594-605 HXB2) gp4 <b>Donor:</b> ? <b>References:</b> [Banapour et al.(1987)] <b>NOTES:</b>	gp41(84-94) 987)]	GIWGCSGKLIC	Z	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{2\lambda})$
657 polyclonal	gp41(598-609)	gp41(84-91)	GIWGCSGK		HIV-1 infection	human
	<ul> <li>Donor: ?</li> <li>References: [Poumbourios et al.(1992)]</li> <li>NOTES:</li> <li>α(598-609): Affinity purified from F</li> <li>bourios et al.(1992)]</li> </ul>	ul.(1992)] ed from HIV-1+	nor: ?  erences: [Poumbourios et al.(1992)]  TES: $\alpha$ (598-609): Affinity purified from HIV-1+ plasma – immunodominant region, binds bourios et al.(1992)]		oligomer and monomer [Poum-	T

MAD III	Location	VY EAC	Seductice	Surging month	magorina	Species(Isotype)
658 clone 3	gp41 gp41(87-96) GCSGK Donor: ? References: [Cotropia et al.(1992), Cotropia et al.(1996)] NOTES:	gp41(87-96)	GCSGKLICTT al.(1996)]	Т	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_1)$
	<ul> <li>clone 3: Core binding domain gcsgkLIC – lack of serologi in infants ([Broliden et al.(1989)]) [Cotropia et al.(1992)]</li> <li>clone 3: Inhibits replication of three diverse HIV-1 labora et al.(1996)]</li> </ul>	ain gcsgkLIC – la 1989)]) [Cotropia n of three diverse	clone 3: Core binding domain gcsgkLIC – lack of serological activity to this region correlates with rapid progression in infants ([Broliden et al.(1989)]) [Cotropia et al.(1992)] clone 3: Inhibits replication of three diverse HIV-1 laboratory strains, as well as an AZT-resistant isolate [Cotropia et al.(1996)]	is region correla well as an AZT-	ıtes with rapid progressi resistant isolate [Cotrop	on via
659 polyclonal	gp41(601-616) gp41(84-99) GIWGCSGKLICTTAV Donor: ?  References: [Petrov et al.(1990)]  NOTES:  Immunodominant and broadly reactive peptide [Petrov et al.(1990)]	gp41(84-99)  ))]  idly reactive pept	GIWGCSGKLICTTAVP ide [Petrov et al.(1990)]	Z	HIV-1 infection	human sera
660 41-7	gp41(605-611)  Donor: ?  References: [Bugge et al.(1990)]  NOTES:  • 41-7: Sera from 6/6 HIV-1 et al.(1990)]	gp41(88-94)  ))]  '-1 positive, but	11(605-611) gp41(88-94) CSGKLIC N nor:? ferences: [Bugge et al.(1990)] TES: 41-7: Sera from 6/6 HIV-1 positive, but no HIV-2 positive, individuals interfered et al.(1990)]		HIV-1 infection with 41-7 binding [Bugge	$ ext{human}( ext{Ig} ext{G}_{1\kappa})$ ge
661 2A2/26	gp41(584-606 BRU)	gp41(69-91)	RILAVERYLKDQQLLGI- WGCSGK		viral gp41	murine(IgG)
	<ul> <li>Donor: ?</li> <li>References: [Poumbourios et al.(1992), Poumbourios et al.(1995)]</li> <li>NOTES:</li> <li>2A2/26: Immunodominant region, binds both oligomer and monor</li> <li>2A2/26: △ 550-561 (△ LLRAIEAQQHLL), a region important</li> <li>(550-561 +571-581) abrogates binding [Poumbourios et al.(1995)]</li> </ul>	d.(1992), Poumboregion, binds booking the Linding Inc.	nor:?  ierences: [Poumbourios et al.(1992), Poumbourios et al.(1995)]  TES:  2A2/26: Immunodominant region, binds both oligomer and monomer [Poumbourios et al.(1992)]  2A2/26: △ 550-561 (△ LLRAIEAQQHLL), a region important for oligomer formation diminishes binding, △  2A2/26: A 550-561 (△ LRAIEAQQHLL), a region important for oligomer formation diminishes binding, △	umbourios et al	t al.(1992)] ation diminishes binding,	<b>&gt;</b>

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
662 98-43	gp41(579-604 HXB2)	gp41(69-94)	RILAVERYLKDQQLLGI- WGCSGKLIC	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{2\kappa})$
	Donor: ?  References: [Pinter et al.(1989), Gorny et al.(1989), Tyler et al.(1990), Xu et al.(1991)]  NOTES:  98.43: Reacts equally well with oligomer and monomer [Pinter et al.(1989)]	, Gorny et al.(1)	989), Tyler et al.(1990), Xu et :	al.(1991)]		
	<ul> <li>98-43: Reacts equally well with oligomer and monomer [Pinter et al.(1)</li> <li>98-43: Poor ADCC (in contrast to MAb 120-16, gp41(644-663)) [Tyler</li> <li>98-43: 579-604 binds in the immunodominant region [Xu et al.(1991)]</li> <li>98-43: NIH AIDS Research and Reference Reagent Program: 1241</li> </ul>	with oligomer a rast to MAb 12 immunodomin and Reference	98-43: Reacts equally well with oligomer and monomer [Pinter et al.(1989)] 98-43: Poor ADCC (in contrast to MAb 120-16, gp41(644-663)) [Tyler et al.(1990)] 98-43: 579-604 binds in the immunodominant region [Xu et al.(1991)] 98-43: NIH AIDS Research and Reference Reagent Program: 1241	al.(1990)]		
663 181-D	gp41(591-597 HXB2) <b>Donor:</b> ?	gp41(81-87)	QLLGIWG	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{2\kappa})$
	References: [Xu et al.(1991), Robinson Jr. et al.(1991), Eddleston et al.(1993), Forthal et al. NOTES:	binson Jr. et al.	(1991), Eddleston et al.(1993),		l.(1995), Fontenot et al.(1995)]	95)]
	<ul> <li>181-D: Fine mapping indicates core is LLGIW [Xu et al.(1991)]</li> <li>181-D: No enhancing or neutralization activity [Robinson Jr. et al. 181-D: Called SZ-181.D [Eddleston et al.(1993)]</li> <li>181-D: No neutralizing, no ADCC, and no viral enhancing activities.</li> </ul>	ites core is LLC utralization active ddleston et al.(1 ADCC, and no	181-D: Fine mapping indicates core is LLGIW [Xu et al.(1991)] 181-D: No enhancing or neutralization activity [Robinson Jr. et al.(1991)] 181-D: Called SZ-181.D [Eddleston et al.(1993)] 181-D: No neutralizing, no ADCC, and no viral enhancing activity [Forthal et al.(1995)]	al et al.(1995)]		
664 240-D	gp41(592-600 HXB2) <b>Donor:</b> ?	gp41(82-90)	LLGIWGCSG	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	References: [Xu et al.(1991), Robinson Jr. et al.(1991), Spear et al.(1993), Binley et al.(1996), Wisnewski et al.(1996), Mitchell et al.(1998)] NOTES:	obinson Jr. et a l et al.(1998)]	l.(1991), Spear et al.(1993), Bi	nley et al.(1996)	1996), Wisnewski et al.(1995),	95),
	<ul> <li>240-D: Fine mapping indicates core is IWG [Xu et al.(1991)]</li> <li>240-D: No neutralizing activity, some enhancing activity [Rol</li> <li>240-D: Did not mediate deposition of complement componen</li> <li>240-D: Binds to a linear epitope located in the Cluster I regio</li> </ul>	ntes core is IWC vity, some enhance osition of comproper located in	240-D: Fine mapping indicates core is IWG [Xu et al.(1991)] 240-D: No neutralizing activity, some enhancing activity [Robinson Jr. et al.(1991)] 240-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear et al.(1993)] 240-D: Binds to a linear epitope located in the Cluster I region – binding of 50-69 and 240-D inhibited by Figure 1.	al.(1991)] infected cells [S of 50-69 and 240	ells [Spear et al.(1993)] d 240-D inhibited by Fabs A1,	A1,
	<ul> <li>240-D: Called F240: F240</li> <li>V<sub>H</sub>4, and reduced V<sub>H</sub>3, wz</li> <li>240-D: Mutations in BH10</li> <li>(GCSGKLICTTAVP), abro</li> </ul>	in $V_H 3 - V$ -reg is noted among gp160, W596 gate binding of	240-D: Called F240: F240 in $V_H3 - V_T$ 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)] 240-D: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605-609 (TTAVP) and 597-609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50-69, and 246-D – 5/6 enhancing MAbs	amined and a bias onewski et al.(1996)] tions of 605-609 (60-69, and 246-D –	as of enhanced $V_H 1$ 6)] 9 (TTAVP) and 597- 0 – 5/6 enhancing M	and -609 Abs
	<ul> <li>dentined to date bind to the immunodominant region 5/9-613 [Mitto</li> <li>240-D: NIH AIDS Research and Reference Reagent Program: 1242</li> </ul>	and Reference	identified to date bind to the immunodominant region 5/9-613 [Mitchell et al.(1998)] 240-D: NIH AIDS Research and Reference Reagent Program: 1242	a1.(1998)]		

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
665 D61	gp41(592-608 HXB2) <b>Donor:</b> ? <b>References:</b> [Earl et al.(1994).	gp41(82-98) , Richardson Jr e	gp41(592-608 HXB2) gp41(82-98) LLGIWGCSGKLICTTAV Donor: ? References: [Earl et al.(1994), Richardson Jr et al.(1996), Weissenhorn et al.(1996), Earl		dimeric Env et al.(1997)]	murine
	<ul> <li>D61: Linear gp41 epitope in the cluster I region – human sera blocl extent for gp41 MAbs D20, D43, D61, and T4 [Richardson Jr et a D61: Does not precipitate gp41(21-166), but due to a structural ctwo cysteines – the authors propose that this region may change confusion state of the HIV-1 glycoprotein [Weissenhorn et al.(1996)]</li> <li>D61: Binding maps to region 597-613: WGCSGKLICTTAVPWN residues – this antibody, along with human MAb 246-D, can be block (M10, D41, D54, T4, T6, T9, T10 and T35) – members of this coindividuals [Earl et al.(1997)]</li> </ul>	in the cluster I re 0, D43, D61, and 0, D43, D61, and gp41(21-166), I s propose that thi glycoprotein [We ion 597-613: Wo ong with human T9, T10 and T35	D61: Linear gp41 epitope in the cluster I region – human sera blocked binding in oligomeric ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 [Richardson Jr et al.(1996)] D61: Does not precipitate gp41(21-166), but due to a structural difference in the disulfide bonding region near the two cysteines – the authors propose that this region may change conformation during the activation of the membrane fusion state of the HIV-1 glycoprotein [Weissenhorn et al.(1996)] D61: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues – this antibody, along with human MAb 246-D, can be blocked by any of a group of 8 conformational MAbs (M10, D41, D54, T4, T6, T9, T10 and T35) – members of this competition group are blocked by sera from HIV-1+ individuals [Earl et al.(1997)]	ng in oligomeric in the disulfide on during the ac unodominant re uny of a group o group are bloc	omeric ELISA assay to a similar sulfide bonding region near the the activation of the membrane nant region containing two Cysroup of 8 conformational MAbs e blocked by sera from HIV-1+	milar r the rane Cys [Abs
666 D49	gp41(597-613) <b>Donor:</b> ?	gp41(82-98)	LLGIWGCSGKLICTTAV		dimeric Env	murine
	<ul> <li>References: [Earl et al.(1994), Earl et al.(1997)]</li> <li>NOTES:</li> <li>D49: Binding maps to region 597-613: WG-residues [Earl et al.(1997)]</li> </ul>	Earl et al.(1997) ion 597-613: W	erences: [Earl et al.(1994), Earl et al.(1997)]  FES:  D49: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodomiresidues [Earl et al.(1997)]	unodominant re	nant region containing two Cys	Cys
667 T32	gp41(597-613) gp41(82-98) <b>Donor:</b> ? <b>References:</b> [Earl et al.(1994), Earl et al.(1997)] <b>NOTES:</b>	gp41(82-98) , Earl et al.(1997	LLGIWGCSGKLICTTAV )]		tetrameric Env	murine
	• T32: Binding maps to regiresidues [Earl et al.(1997)]	ion 597-613: Wo	T32: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodomin residues [Earl et al.(1997)]	unodominant re	nant region containing two Cys	Cys
668 T34	gp41(597-613) gp41(82-98) <b>Donor:</b> ? <b>References:</b> [Earl et al.(1994), Earl et al.(1997)] <b>NOTES:</b>	gp41(82-98) , Earl et al.(1997	LLGIWGCSGKLICTTAV		tetrameric Env	murine
	• T34: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys	ion 597-613: Wo				)

Donor: Susan Zolla-Paner, NY, IWA Center, NY, IWY  References: [Xu et al.(1991), Robinson Jr. et al.(1991). Spear et al.(1993), Eddleston et al.(1993), Forthal et al.(1995), Manca et al.(1995), Saarloos et al.(1995), Earl et al.(1997)]  NOTES:  • 246-D: Fine mapping indicates core is LLGI [Xu et al.(1991)]  • 246-D: Fine mapping indicates core is LLGI [Xu et al.(1991)]  • 246-D: Called SC-246.D [Eddleston of complement component C3 on HIV infected cells unless cells were pre-incubated with sCD4 [Spear et al.(1993)]  • 246-D: No neutralizing activity, both ADCC and viral enhancing activity [Forthal et al.(1991)]  • 246-D: No neutralizing activity, both ADCC and viral enhancing activity [Forthal et al.(1995)]  • 246-D: Virions complexed to gp41 Ab facilitate presentation of p66 RI epitopes to Th cells [Manca et al.(1995)]  • 246-D: Nb-mediated activation of complement on HIV+ cells is higher than Ab independent activation – what has been termed "Ab independent" in fact results in part from IgM in normal human serum that is HIV-cross-reactive [Saarloos et al.(1995)]  • 246-D: Mutations in BHI0 gp160, W596Y and T605A, as well as deletions of 605-609 (TTAVP) and 597-609 (GCSGKLICITIAVP), abrogate binding of enhancing MAbs 86, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69, and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69) and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69) and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69) and 246-D – 5/6 enhancing MAbs (36, 240D, 50-69) and 246-	
SE N. R. D. SE	669 246-D
p41(HXB2)  p41(HXB2)  p41(79-90)  p41(T9-90)  p41(T9-9	
gp41(68-102) ARILAVERYLKDQQLLG- IWGCSGKLICTTAVPWNA	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
672 1F11	gp41(579-613 BH10)	gp41(68-102)	ARILAVERYLKDQQLLG- IWGCSGKLICTTAVPWNA	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria <b>References:</b> [Buchacher et al.(1992), Buchacher et al.(1994)] <b>NOTES:</b>	ppl. Microbiol., Vier 1.(1992), Buchacher	ına, Austria et al.(1994)]			
	• 1F11: Generated by elec	trofusion of PBL frc	• 1F11: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher et al.(1994)]	ith CB-F7 cells	Buchacher et al.(199	
673 4D4	gp41(579-613 BH10)	gp41(68-102)	ARILAVERYLKDQQLLG- IWGCSGKLICTTAVPWNA	Z	HIV-1 infection	$\text{human}(\text{Ig}G_{1\lambda})$
	Donor: H. Katinger, Inst. Apreferences: [Buchacher et a NOTES:	ppl. Microbiol., Vier 1.(1992), Buchacher	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX <b>References:</b> [Buchacher et al.(1992), Buchacher et al.(1994), Chen et al.(1994b), Sattentau et al.(1998), NOTES:	Systems, Houst)), Sattentau et	uston, TX et al.(1995)]	
	<ul> <li>4D4: Generated by elect</li> </ul>	rofusion of PBL fro	• 4D4: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher et al.(1994)]	ith CB-F7 cells	Buchacher et al.(199	(4)]
674 3D9	gp41(579-613 BH10)	gp41(68-102)	ARILAVERYLKDQQLLG- IWGCSGKLICTTAVPWNA	Z	HIV-1 infection	$\mathrm{human}(\mathrm{IgG}_{1\kappa})$
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria <b>References:</b> [Buchacher et al.(1992), Buchacher et al.(1994)]	ppl. Microbiol., Vier 1.(1992), Buchacher	nna, Austria et al.(1994)]			
	• 3D9: Generated by elect	rofusion of PBL fro	3D9: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher et al.(1994)]	ith CB-F7 cells	[Buchacher et al.(199	4)]
675 4G2	gp41(579-613 BH10)	gp41(68-102)	ARILAVERYLKDQQLLG- IWGCSGKLICTTAVPWNA	Z	HIV-1 infection	$\text{human}(\text{Ig}G_{1\kappa})$
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria <b>References:</b> [Buchacher et al.(1992), Buchacher et al.(1994)] <b>NOTES:</b>	ppl. Microbiol., Vier 1.(1992), Buchacher	ına, Austria et al.(1994)]			
	• +Oz. Ocherated by circle	TOTASION OF F DE 110	TO2. Obtained by discussion of the from the $r_1$ positive voluneous with $c_{D-1}$ , some [indicutative of $a_1(1) = 7$ ]	Int CD-17 CCIIs	Epacinaciici et ai.(177	(+)]
676 4B3	gp41(579-613 BH10)	gp41(68-102)	ARILAVERYLKDQQLLG- IWGCSGKLICTTAVPWNA	Z	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\lambda})$
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria <b>References:</b> [Buchacher et al.(1992)]. Buchacher et al.(1994)	onl Microbiol Vie	ına Austria			
	NOTES.	1.(1992), Buchacher	References: [Buchacher et al.(1992), Buchacher et al.(1994), Chen et al.(1994b)]	9)]		

677 50-69	MAb ID
gp41(579-603 BH10)	MAb ID Location
gp41(69-93)	WEAU
RILAVERYLKDQQLLGI- WGCSGKLI	U Sequence
Z	Neutralizing
HIV-1 infection	Immunogen
$\operatorname{human}(\operatorname{IgG}_{2\kappa})$	Species(Isotype)
	gp41(579-603 BH10) gp41(69-93) RILAVERYLKDQQLLGI- N HIV-1 infection h WGCSGKLI

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

## ZCIEV:

- 50-69: Combined with deglycosylated A chain of ricin is toxic to lines of HIV-infected T cells (H9) and monocytes (U937) [Till et al.(1989)]
- 50-69: Reacts preferentially with gp160 oligomer, compared to gp41 monomer [Pinter et al.(1989)]
- 50-69: Kills HIV-infected cells when coupled to deglycosylated ricin A chain [Gorny et al.(1989)]
- 50-69: The epitope is affected by the conformation conferred by the two cysteines at amino acids 598 and 604 [Xu et al.(1991)]
- 50-69: Enhances HIV-1 infection in vitro synergizes with huMAb 120-16 in vitro to enhance HIV-1 infection to level approaching that found in polyclonal anti-HIV serum [Robinson Jr. et al.(1991)]
- 50-69: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 50-69: Called SZ-50.69 binds to an epitope within aa 579-613 [Eddleston et al.(1993)]
- 50-69: Did not mediate deposition of complement component C3 on HIV infected cells unless cells were pre-incubated with sCD4 - complement mediated virolysis of MN and IIIB in the presence of sCD4 [Spear et al.(1993)]
- 50-69: Epitope described as Cluster I, 601-604, conformational does not neutralize IIIB or synergize neutralization by anti-V3 MAb 447-52D or by CD4 BS MAbs [Laal et al.(1994)]
- 50-69: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the et al.(1995)] leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen
- 50-69: Preferentially binds oligomer binding increased after pretreatment of infected cells with sCD4 binding domain overlaps site that is critical for gp120-gp41 association, avEry [Sattentau et al.(1995)]
- 50-69: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca et al.(1995)]
- 50-69: Does not neutralize HIV-1 LAI [McDougal et al.(1996)]
- 50-69: Prebinding of anti-V3, and CD4i MAbs 48d and 17b, but not anti-V2 neutralizing MAbs, expose the 50-69 epitope [Poignard et al.(1996a)]
- 50-69: Binds to a linear epitope located in the Cluster I region binding of 50-69 and 240-D inhibited by Fabs A1. A4, M8B, M26B, M12B and T2 [Binley et al.(1996)]
- 50-69: Used to test exposure of gp41 upon sCD4 binding [Klasse & Sattentau(1996)]
- 50-69: Binding of anti-gp120 MAbs IgG1b12 or 654-30D does not mediate significant exposure of the gp41 epitopes for MAbs 2F5 and 50-69 [Stamatatos et al.(1997)]

677 cont.

- 50-69: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library 50-69 maps to an immunodominant domain in gp41 - three groups of peptides were selected, one which seems most closely related to gp41 sequence peptide consensus is WGCxx(RK)( $x_n$ )LxC – the analogous gp41 sequence WGCSGKLIC is present in most M group clades, except D with a common L to H substitution [Boots et al.(1997)]
- 50-69: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605-609 (TTAVP) and 597-609 T605 as minimal epitope [Mitchell et al.(1998)] identified to date bind to the immunodominant region 579-613 - identifies non-contiguous W596-G597-C598...C604 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50-69, and 246-D - 5/6 enhancing MAbs
- 50-69: NIH AIDS Research and Reference Reagent Program: 531

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
678 Fab A1	gp41(584-609 LAI)	gp41(69-98)	RILAVERYLKDQQLLGI- WGCSGKLICTTAV	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	Donor: Susan Zolla-Pazner, NYU, NY References: [Binley et al.(1996)] NOTES:	NYU, NY 96)]				
	• Fab A1: Binds to Cluster I region – c regions sequenced [Binley et al.(1996)]	r I region – comp y et al.(1996)]	Fab A1: Binds to Cluster I region – competes with MAbs 240-D and 50-69 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	50-69 – conform	ation sensitive – var	iable
679 Fab A4	gp41(584-609 LAI)	gp41(69-98)	RILAVERYLKDQQLLGI- WGCSGKLICTTAV	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	<b>Donor:</b> Susan Zolla-Pazner, NYU, NY <b>References:</b> [Binley et al.(1996)] <b>NOTES:</b>	96)]				
	• Fab A4: Binds to Cluster I region – c regions sequenced [Binley et al.(1996)]	r I region – comp y et al.(1996)]	Fab A4: Binds to Cluster I region – competes with MAbs 240-D and 50-69 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	50-69 – conform	ation sensitive – var	iable
680 Fab M8B	gp41(584-609 LAI)	gp41(69-98)	RILAVERYLKDQQLLGI- WGCSGKLICTTAV	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	<b>Donor:</b> Susan Zolla-Pazner, NYU, NY <b>References:</b> [Binley et al.(1996)]	96)]				
	NOTES:				•	•
	• Fab M8B: Binds to Clust	ter I region – com	Fab M8B: Binds to Cluster I region – competes with MAbs 240-D and 50-69 – conformation sensitive – variable	50-69 – conform	nation sensitive – var	iable
	regions sequenced [Binley et al.(1996)]	y et al.(1996)]	4			

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
681 Fab M26B	gp41(584-609 LAI)	gp41(69-98)	RILAVERYLKDQQLLGI- WGCSGKLICTTAV	N	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$
	<ul> <li>Donor: Susan Zolla-Pazner, NYU, NY References: [Binley et al.(1996)]</li> <li>NOTES:</li> <li>Fab M26B: Binds to Cluster I region – regions sequenced [Binley et al.(1996)]</li> </ul>	YU, NY 6)] er I region – con et al.(1996)]	nor: Susan Zolla-Pazner, NYU, NY ferences: [Binley et al.(1996)]  NTES: Fab M26B: Binds to Cluster I region – competes with MAbs 240-D and 50-69 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	50-69 – conform	ation sensitive – vari	able
682 Fab T2	gp41(584-609 LAI)	gp41(69-98)	RILAVERYLKDQQLLGI- WGCSGKLICTTAV	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	Donor: Susan Zolla-Pazner, NYU, NY References: [Binley et al.(1996)] NOTES:	YU, NY 6)]				
	• Fab T2: Binds to Cluster I region – coregions sequenced [Binley et al.(1996)]	I region – comp et al.(1996)]	Binds to Cluster I region – competes with MAbs 240-D and $50-69$ – conformation sensitive – variable sequenced [Binley et al.(1996)]	0-69 – conform	ation sensitive – varia	able
683 Fab M12B	gp41(584-609 LAI)	gp41(69-98)	RILAVERYLKDQQLLGI- WGCSGKLICTTAV	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	Donor: Susan Zolla-Pazner, NYU, NY References: [Binley et al.(1996)] NOTES:  • Fab M12B: Binds to Cluster I region – regions sequenced [Binley et al.(1996)]	YU, NY 6)] er I region – con et al.(1996)]	or: Susan Zolla-Pazner, NYU, NY erences: [Binley et al.(1996)] TES: Fab M12B: Binds to Cluster I region – competes with MAbs 240-D and 50-69 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	50-69 – conform	nation sensitive – vari	able
684 41.4	gp41(584-609)	gp41(69-98)	RILAVERYLKDQQLLGI- WGCSGKLICTTAV			
	<b>Donor:</b> Jan McClure, Bristol-Myers Squibb Pharmaceutical Res Inst, Seattle, WA <b>References:</b> [Pincus & McClure(1993)] <b>NOTES:</b>	Лyers Squibb Ph re(1993)]	armaceutical Res Inst, Seattle,	WA		
	<ul> <li>41.4: Binds to peptide weakly, but conformational – MAb was couple 30-fold [Pincus &amp; McClure(1993)]</li> </ul>	kly, but to gp160 s coupled to ricire (1993)]	41.4: Binds to peptide weakly, but to gp160 with higher affinity than 41.1, and cross-competes with 41.1 – probably conformational – MAb was coupled to ricin A chain (RAC) – sCD4 enhances the efficacy of immunotoxins in vitro 30-fold [Pincus & McClure(1993)]	and cross-comp	of immunotoxins in v	ably itro

MAb ID	Location	WEAU	Sequence	Neutralizing Ir	Immunogen	Species(Isotype)
685 41-1	gp41(584-609)	gp41(69-98)	RILAVERYLKDQQLLGI- WGCSGKLICTTAV	18	gp160	$\mathrm{murine}(\mathrm{IgG}_{1\kappa})$
	<b>Donor:</b> Jan McClure, Bristol-Myers Sq <b>References:</b> [Gosting et al.(1987), Dalgl Pincus et al.(1996), Pincus et al.(1998)] <b>NOTES:</b>	tol-Myers Squibb Ph (1987), Dalgleish et a et al.(1998)]	Donor: Jan McClure, Bristol-Myers Squibb Pharmaceutical Res Inst, Seattle, WA References: [Gosting et al.(1987), Dalgleish et al.(1988), Pincus et al.(1991), Pincus & McC. Pincus et al.(1996), Pincus et al.(1998)] NOTES:	WA incus & McClure(19	lure(1993), Mani et al.(1994),	÷
	<ul> <li>41-1: This antibody gp41(584-609) [Mani et gp41(735-752 IIIB) [Dalgleish et al.(1988)]</li> <li>41-1: Also called 41.1, although possibly no region with similar names (dash versus period 41-1: Broadly reactive [Gosting et al.(1987)]</li> </ul>	p41(584-609) [Mani Palgleish et al.(1988) , although possibly i mes (dash versus per [Gosting et al.(1987	41-1: This antibody gp41(584-609) [Mani et al.(1994)] seems to have been named the gp41(735-752 IIIB) [Dalgleish et al.(1988)] 41-1: Also called 41.1, although possibly not, the literature is confusing because two region with similar names (dash versus period) are listed as murine and human 41-1: Broadly reactive [Gosting et al.(1987)]	en named the same because two gp41 Numan	e same as a different MAb to gp41 MAbs that bind to this	is to
	<ul> <li>41-1: This antibody se</li> <li>41-1: Efficacious as</li> <li>et al (1991)1</li> </ul>	ems to have been nam an immunotoxin wh	41-1: This antibody seems to have been named the same as a different MAb to gp41(735-752) [Dalgleish et al.(1988)] 41-1: Efficacious as an immunotoxin when coupled to RAC – gave linear epitope as gp160 579-603 [Pincus et al.(1991)]	b to gp41(735-752) [] linear epitope as gp	752) [Dalgleish et al.(1988)] as gp160 579-603 [Pincus	)] )3
	<ul> <li>41-1: Called 41.1, an immunotoxins in vitro</li> <li>41-1: Did not requiret al.(1994)]</li> </ul>	d described as a hun 30-fold – MAb was the C-C disulfide	41-1: Called 41.1, and described as a human MAb – cross-competes with 41.4 – sCD4 enhances the efficacy of immunotoxins in vitro 30-fold – MAb was coupled to ricin A chain (RAC) – [Pincus & McClure(1993)] 41-1: Did not require the C-C disulfide bridge and loop formation, can bind simultaneously with 9-11 [Mani et al.(1994)]	rith 41.4 – sCD4 enl C) – [Pincus & McCl an bind simultaneou	hances the efficacy of ure(1993)] usly with 9-11 [Mar	of ni
	<ul> <li>41-1: Called 41.1, and desc by linking Env MAbs to rici binding [Pincus et al.(1996)]</li> </ul>	d described as a hur to ricin A – immunc [1996]]	41-1: Called 41.1, and described as a human MAb, binding 579-604 – a panel of immunotoxins was generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]	- a panel of immuno out killing was not d	otoxins was generate irectly proportional t	to
686 9-11	gp41(584-609)	gp41(69-94)	RILAVERYLKDQQLLGI- WGCSGKLIC	15	gp160	$murine(IgG_1)$
	Donor: Jan McClure, Bristol-M: References: [Mani et al.(1994)] NOTES:	tol-Myers Squibb Ph 994)]	Donor: Jan McClure, Bristol-Myers Squibb Pharmaceutical Res Inst, Seattle, WA References: [Mani et al.(1994)] NOTES:	WA		
	• 9-11: required the C-C	disulfide bridge and	• 9-11: required the C-C disulfide bridge and loop formation, can bind simultaneously with 41-1 [Mani et al.(1994)]	ultaneously with 41-	1 [Mani et al.(1994)]	
687 polyclonal	gp41(589-596) g <b>Donor:</b> Jan McClure, Bristol-Myo <b>References:</b> [Klasse et al.(1991)] <b>NOTES:</b>	gp41 (72-79) tol-Myers Squibb Ph [1991)]	gp41(589-596) gp41 (72-79) AVERYLKD  Donor: Jan McClure, Bristol-Myers Squibb Pharmaceutical Res Inst, Seattle, WA  References: [Klasse et al.(1991)]  NOTES:		HIV-1 infection	human sera
	<ul> <li>Substitutions and deletions in peptide 583-599 were systematically studied – alterations in AVERYLKD abrogated</li> </ul>	tions in peptide 583-	COO orrotamatically studio			

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
688 polyclonal	gp41(583-599) gp Donor: Jan McClure, Bristol-Myer References: [Klasse et al.(1993b)] NOTES:  • 42 HIV-1 positive human sera w strongly with WT, weakly with et al.(1993b)]	gp41 (66-82) Myers Squibb Pha 93b)] sera were tested aga with A589T – 31 r	gp41(583-599) gp41 (66-82) LQARILAVERYLKDQQL HIV-1 infection Donor: Jan McClure, Bristol-Myers Squibb Pharmaceutical Res Inst, Seattle, WA References: [Klasse et al.(1993b)] NOTES: • 42 HIV-1 positive human sera were tested against WT peptide, and peptide with substitution 589 A to T: 11/42 reacted strongly with WT, weakly with A589T – 31 reacted weakly with parental, even more weakly with substituted [Klasse et al.(1993b)]	WA vith substitution : ven more weakly	HIV-1 infection 589 A to T: 11/42 reacte with substituted [Klass	human sera ed se
689 9G5A	gp41(596-599 IIIB)	gp41 (81-84) QLLG	QLLG		Anti-idiotype against M38	murine(IgM)
	<ul> <li>Donor: Jan McClure, Bristol-Myers Squibb Pharmaceutical References: [Lopalco et al.(1993), Beretta &amp; Dalgleish(1994)]</li> <li>NOTES:</li> <li>9G5A: Anti-idiotype to gp120 C terminus (C5 region) MA</li> </ul>	Myers Squibb Pha 993), Beretta & Da p120 C terminus (	<ul> <li>Donor: Jan McClure, Bristol-Myers Squibb Pharmaceutical Res Inst, Seattle, WA References: [Lopalco et al.(1993), Beretta &amp; Dalgleish(1994)]</li> <li>NOTES:</li> <li>9G5A: Anti-idiotype to gp120 C terminus (C5 region) MAb M38 [Lopalco et al.(1993)]</li> </ul>	WA  o et al.(1993)]		
690 3D6	gp41 (604-617 BH10) <b>Donor:</b> H. Katinger, Inst. Ap <b>References:</b> [Felgenhauer et a <b>NOTES:</b>	gp41(89-103) pl. Microbiol., Vie l.(1990), He et al.(1	gp41(604-617 BH10) gp41(89-103) SGKLICTTAVPWNAS  Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, References: [Felgenhauer et al.(1990), He et al.(1992), Chen et al.(1994b), Sattentau et al. NOTES:	HIV- Systems, Houston, TX ntau et al.(1995), Wisn	HIV-1 infection Houston, TX (1995), Wisnewski et al.(1996)]	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	<ul> <li>3D6: Sequence of cDNA encoding V-regions [Felger</li> <li>3D6: Fab fragment crystal structure [He et al.(1992)]</li> <li>3D6: This MAb binds to HIV gp41, and to a 43 kd pr</li> </ul>	encoding V- regional structure [He et a HIV gp41, and to	3D6: Sequence of cDNA encoding V-regions [Felgenhauer et al.(1990)] 3D6: Fab fragment crystal structure [He et al.(1992)] 3D6: This MAb binds to HIV gp41, and to a 43 kd protein found in human T, B and	n T, B and mono	monocyte cell lines, proposed	Ģ.
	<ul> <li>3D6: Called IAM 41-3D6: binding in overlaps site that is critical for gp120-gp</li> <li>3D6: 3D6 is V<sub>H</sub>3 – V-region heavy chair V<sub>H</sub>3, was noted among HIV infected in</li> </ul>	l et al.(19940)] 06: binding increa ld for gp120-gp41 a gion heavy chain us IIV infected individ	molecular minicry [Chen et al. (1994b)] 3D6: Called IAM 41-3D6: binding increased after pretreatment of infected cells with sCD4 – binding domain overlaps site that is critical for gp120-gp41 association, cttaV [Sattentau et al. (1995)] 3D6: 3D6 is $V_H 3 - V$ -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)]		with sCD4 – binding domain $\mid$ and $\mid$ $\mid$ and $\mid$ $\mid$ and $\mid$ $\mid$ and reduced	ğ ii.

MAb ID	MAb ID Location	WEAU	Sequence	Neutralizing Immunogen	Immunogen	Species(Isotype)
693 98-6	gp41(644-663 HXB2)	gp41(134-153)	gp41(134-153) SLIEESQNQQEKNEQEL- LEL	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{2\kappa})$
	<b>Donor:</b> H. Katinger, Inst. Ap	opl. Microbiol., Vier	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX	Systems, Housto	ouston, TX	

et al.(1993), Tani et al.(1994), Laal et al.(1994), Chen et al.(1995), Forthal et al.(1995), Manca et al.(1995), Sattentau et al.(1995), Wisnewski et al.(1996)] Andris et al. (1992), Sattentau & Moore (1991), Robinson Jr. et al. (1991), Xu et al. (1991), Eddleston et al. (1993), Spear **References:** [Finiter et al.(1989), Gorny et al.(1989), Till et al.(1989), Robinson Jr. et al.(1990a), Tyler et al.(1990),

## VOIES:

- 98-6: Reacts preferentially with gp160 oligomer, compared to gp41 monomer [Pinter et al.(1989)]
- 98-6: Kills HIV-infected cells when coupled to deglycosylated ricin A chain [Gorny et al.(1989)]
- 98-6: Toxic to HIV-infected T cells (H9) and monocytes (U937) when coupled to deglycosylated A chain of ricin [Till et al.(1989)]
- 98-6: No neutralizing or enhancing activity for HIV-1 IIIB [Robinson Jr. et al.(1990a)]
- 98-6: Serves as target for antibody-dependent cellular cytotoxicity, ADCC [Tyler et al.(1990)]
- 98-6: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 98-6: No neutralizing or enhancing activity [Robinson Jr. et al.(1991)]
- 98-6: Appeared to be specific for a conformational or discontinuous epitope [Xu et al.(1991)]
- 98-6: Called SZ-98.6 binds to a conformational domain within aa 644-663 of gp41, and reacts with astrocytes, as do 167-7 and ND-15G1 [Eddleston et al.(1993)]
- 98-6: Did not mediate deposition of complement component C3 on HIV infected cells, binding enhanced by sCD4 [Spear et al.(1993)]
- 98-6: This MAb was expressed as a surface anti-gp41 monoclonal antibody receptor for gp41 on a CD4-negative specifically enhanced viral replication [Tani et al.(1994)] by retroviral constructs was expressed on these cells, they acquired the ability to replicate HIV-1, and slg/gp41 B-cell line. Transfected cells could bind HIV envelope, but could not be infected by HIV-1. When CD4 delivered
- 98-6: Epitope described as Cluster II, 644-663, conformational does not neutralize IIIB or synergize neutralization by anti-V3 MAb 447-52D or by CD4 BS MAbs [Laal et al.(1994)]
- 98-6: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen
- 98-6: No neutralizing activity, positive ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]
- 98-6: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca et al.(1995)]
- 98-6: Preferentially recognizes oligomeric form of gp41 enhanced binding to HIV-1 infected cells at 37 degrees relative to 4 degrees - addition of sCD4 enhances binding [Sattentau et al.(1995)]
- 98-6: 98-6 is  $V_H4 V$ -region heavy chain usage was examined and a bias of enhanced  $V_H1$  and  $V_H4$ , and reduced  $V_H$ 3, was noted among HIV infected individuals [Wisnewski et al.(1996)]
- 98-6: NIH AIDS Research and Reference Reagent Program: 1240

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
694 167-7	gp41(644-663)	gp41(134-153)	SLIEESQNQQEKNEQEL-		HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{2\lambda})$
	<b>Donor:</b> H. Katinger, Inst. Appl. Microbiol., Vienna, References: [Xu et al.(1991), Eddleston et al.(1993)] NOTES:	.ppl. Microbiol., Vien ), Eddleston et al.(19	Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX References: [Xu et al.(1991), Eddleston et al.(1993)] NOTES:	Systems, Housto	n, TX	
	<ul> <li>167-7: Specific for a conformational epitope [Xu et al.(1991)]</li> <li>167-7: Called SZ-167.7 – binds to a conformational domain v as do 98-6 and ND-15G1 [Eddleston et al.(1993)]</li> </ul>	nformational epitope ' – binds to a conform ' l [Eddleston et al.(19)	167-7: Specific for a conformational epitope [Xu et al.(1991)] 167-7: Called SZ-167.7 – binds to a conformational domain within aa 644-663 of gp4 as do 98-6 and ND-15G1 [Eddleston et al.(1993)]		1, and reacts with astrocytes,	s,
695 ND-15G1	gp41(644-663 HXB2)	gp41(134-153)	gp41(134-153) SLIEESQNQQEKNEQEL- LEL		HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	Donor: ? References: [Eddleston et al.(1993)] NOTES: • ND-15G1: Mapped to the conful formula (167-7 [Eddleston et al.(1993)]	I.(1993)] the conformational e	<ul> <li>nor: ?</li> <li>erences: [Eddleston et al.(1993)]</li> <li>TES:</li> <li>ND-15G1: Mapped to the conformational epitope within aa 644-663, and reacts with astrocytes, as do 98-6 and 167-7 [Eddleston et al.(1993)]</li> </ul>	d reacts with ast	rocytes, as do 98-6 an	<u>d</u>
696 167-D	gp41(644-663 HXB2)	gp41(134-153)	gp41(134-153) SLIEESQNQQEKNEQEL- LEL	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\lambda})$
	<b>Donor:</b> ? <b>References:</b> [Spear et al.(1993), Forthal et al.(1995), Manca et al.(1995)] <b>NOTES:</b>	93), Forthal et al.(19	95), Manca et al.(1995)]			
	<ul> <li>167-D: Did not mediat virolysis of MN and III</li> <li>167-D: No neutralizing</li> <li>167-D: Virions complex</li> </ul>	e deposition of complete deposition of complete B in the presence of seactivity, no ADCC acted to gp41 Ab facility	167-D: Did not mediate deposition of complement component C3 on HIV infected cells – complement virolysis of MN and IIIB in the presence of sCD4 [Spear et al.(1993)] 167-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] 167-D: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca et a	/ infected cells - activity [Forthal topes to Th cells	cells – complement mediated orthal et al.(1995)] h cells [Manca et al.(1995)]	ğ

	697 2F5 g	MAb ID Location
Houston, TX, USA  References: [Buchacher et al.(1992), Muster et al.(1993), Allaway et al.(1993), Klasse et al.(1993a), Purtscher et al.(1994)  Laal et al.(1994), Buchacher et al.(1994), D'Souza et al.(1994), Conley et al.(1994b), Thali et al.(1994), Chen et al.(1994b)  Muster et al.(1994), Beretta & Dalgleish(1994), D'Souza et al.(1995), Trkola et al.(1995), Sattentau et al.(1995)  Moore & Ho(1995), Neurath et al.(1995), Kessler 2nd et al.(1995), Calarota et al.(1996), McKeating(1996), Poignard et al.(1996), Sattentau(1996), Conley et al.(1996), Pincus et al.(1996), McKeating et al.(1996), Stoiber et al.(1996)	gp41(662-667 BH10) gp41(152-157) ELDKWA LP HIV-1 infection hun <b>Donor:</b> Hermann Katinger, U. of Bodenkultur, or Polymun Scientific Inc., Vienna, Austria; Viral Testing Systems,	Location
(1992), Muster et tal.(1994), D'Sou & Dalgleish(199 et al.(1995), Kes	gp41(152-157) ELDKWA U. of Bodenkultur, or Polym	WEAU
al.(1993), Allaway et al.(1993), Za et al.(1994), Conley et al.(1994), D'Souza et al.(1995), Talarol (1996) Pincus et al.(1996) M	ELDKWA ır, or Polymun Scientific Inc	Sequence
), Klasse et al.(1994b), Thali et 1994b), Thali et Irkola et al.(1961), ta et al.(1996), To Keating et al.	L P c., Vienna, Au	Neutralizing
1993a), Purtscher et al. (1994) al. (1994), Chen et al. (1994b) 395), Sattentau et al. (1995) McKeating (1996), Poignard (1996), Striber et al. (1996)	HIV-1 infection stria; Viral Testing Sy	g Immunogen
(1994), 994b), 1995), ignard	$\begin{array}{c} \text{human}(\operatorname{Ig} G_{3\kappa}) \\ \text{'stems,} \end{array}$	Species(Isotype)

## NOLES

- 2F5: Also called IAM 2F5, IAM-41-2F5, IAM2F5
- 2F5: DKWA defined as the core sequence highly conserved epitope neutralizing MAb [Buchacher et al.(1992),

& Montefiori(1997), Earl et al.(1997), Andrus et al.(1998), Mondor et al.(1998), Connor et al.(1998), Yang et al.(1998) Moore & Trkola(1997), Mascola et al.(1997), Stamatatos et al.(1997), Turbica et al.(1997), Ugolini et al.(1997), Burton

Purtscher et al. (1996), Schutten et al. (1997), D'Souza et al. (1997), Mo et al. (1997), Li et al. (1997), Kessler II et al. (1997),

Trkola et al. (1998), Fouts et al. (1998), Ernst et al. (1998), Takefman et al. (1998), Li et al. (1998)]

- 2F5: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)]
- tion efficiency of 2F5 is not affected [Klasse et al.(1993a)] changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs - neutraliza-2F5: Called IAM-41-2F5 – reports MAb to be  $IgG_1$  – the gp41 mutation 582(Ala to Thr) results in conformational
- 2F5: Broadly reactive neutralizing activity, ELDKWA is relatively conserved neutralized 2 primary isolates [Purtscher et al.(1994)]
- 2F5: Failed to show synergy with anti-CD4 binding site IIIB neutralizing antibodies [Laal et al.(1994)]
- 2F5: MAb generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher et al.(1994)]
- 2F5: Included in a multi-lab study for antibody characterization binding and neutralization assay comparison [D'Souza et al.(1994)]
- min for gp41 core D(K/R)W Ab resistant isolate had the sequence KLDNWA [Conley et al.(1994b)] 2F5: Called IAM-41-2F5 – neutralized lab and primary isolates –  $t_{1/2}$  dissociation 122 min for the peptide, and 156
- to neutralize [Thali et al.(1994)] 2F5: gp41 mutation (582 A/T) that reduces neutralization of anti-CD4 binding site MAbs does not alter 2F5's ability
- and RF neutralizing sera in immunized mice [Muster et al.(1994)] 2F5: 2F5 epitope ELDKWA inserted into an immunogenic loop in influenza virus hemagglutinin can elicit IIIB, MN
- most labs in a multi-laboratory study involving 11 labs [D'Souza et al.(1995)] 2F5: Found to neutralize MN, JRCSF, and two B subtype primary isolates, but not a D subtype primary isolate, by
- 2F5: Cross-clade primary virus neutralizing activity LDKW defined as the core epitope [Trkola et al.(1995)]

- 2F5: Called IAM 41-2F5 exposed in the presence of gp120 on the cell surface, while most of gp41 is masked binds proximal to transmembrane region [Sattentau et al.(1995)]
- 2F5: Review: binds to the only generally accepted strong neutralizing epitope outside of gp120, one of only 3 MAbs cluster [Moore & Ho(1995)] and John Moore, per comm 1996 with strong broad activity against primary viruses, the others are 2G12 and IgG1b12 - unique member of epitope
- 2F5: MAb binding decreases the accessibility or alters the conformation of the gp41 fusion domain and of gp120 domains, including the binding site for the CD4 cell receptor [Neurath et al.(1995)]
- MAb IgG1b12 (Called BM12) [Kessler 2nd et al.(1995)] 2F5: Broad cross-clade neutralization of primary isolates - additive neutralization in combination with anti-CD4BS
- 2F5: Only 4/20 Argentinian and 3/43 Swedish HIV+ sera reacted with LLELDKWASL sera reacting with peptides peptide QELLELDKWA [Calarota et al.(1996)] found to be more important for polyclonal sera AB binding, 670-675 WNWFDI - 2F5 bound most strongly to the that contained ELDKWA tended to have high neutralization titers - the region carboxyl terminal to EDLKWA was
- 2F5: ELDKWAS is in a gp41 binding region for the negative regulator of complement factor H (CFH) Abs to facilitating HIV destruction by complement [Stoiber et al. (1996)] HIV generally do not cause efficient complement-mediated lysis, but binding of 2F5 can interfere with CHF binding
- 2F5: Primary isolates from clade A, B, and E are neutralized by 2F5 neutralization requires the LDKW motif neutralization resistant isolates or 2F5 selected variants all had substitutions in the D or K [Purtscher et al.(1996)]
- 2F5: Neutralizes HXB2, primary isolates, and chimeric virus with gp120 from primary isolates in an HXB2 back ground [McKeating et al.(1996)]
- 2F5: Review: one of three MAbs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard et al.(1996b)]
- broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 2F5: Review: only four epitopes have been described which can stimulate a useful neutralizing response to a [Sattentau(1996)]
- 2F5: 2F5 was infused into two chimpanzees which were then given an intravenous challenge with a primary HIV-1 [Conley et al.(1996)] acute-phase virus replication and can be influential after the Ab can no longer be detected in the peripheral circulation indicating that preexisting, neutralizing antibodies (passively administered or actively elicited) affect the course of isolate – both became infected, but with delayed detection and prolonged decrease in viral load relative to controls,
- but killing was not directly proportional to binding [Pincus et al.(1996)] 2F5: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing
- 2F5: Called IAM 2F5 antibody mediated enhancement or inhibition seemed to be determined by isolate rather phenotype, and directly proportional to its affinity to monomeric HIV-1 gp160 [Schutten et al.(1997)] than antibody specificity - in this study, only 2F5 inhibited the entry of all the viruses studied, irrespective of their
- 2F5: Of three neutralizing MAbs (257-D, IgG1b12, and 2F5), 2F5 was the only one to inhibit the entry of all viruses studied, both SI and NSI, with a potency proportional to its affinity for monomeric gp126 [Schutten et al.(1997)]

697 cont.

- 2F5: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half were neutralized, and ALDKWQ and ALDKWA were susceptible to neutralization [D'Souza et al.(1997)] neutralizing susceptibility had the sequences ALGQWA or ELDTWA instead of EDLKWA - 7/9 primary isolates of the 9 primary test isolates at a concentration of  $< 25 \mu g$  per ml for 90% viral inhibition – the isolates with no 2F5
- 2F5: A JRCSF variant that was selected for IgG1b12 resistance remained sensitive to MAbs 2G12 and 2F5, for combination therapy [Mo et al.(1997)]
- 2F5: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB synergistic response with MAbs 694/98-D (anti-V3), 2G12, b12, and F105 [Li et al.(1997)] env - strong neutralizer of SHIV-vpu+ - all Ab combinations tested showed synergistic neutralization - 2F5 has
- primary isolates [Kessler II et al.(1997)] 2F5: IgG1b12 was more potent with greater breadth than MAb 2F5 in an infection reduction assay including 35
- should consider including constructs that may enhance exposure of these MAbs' epitopes [Moore & Trkola(1997)] 2F5: Review: MABs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic - homologous MAbs to these are rare in humans and vaccine strategies
- 2F5: Binding of anti-gp120 MAbs IgG1b12 or 654-30D does not mediate significant exposure of the gp41 epitopes for MAbs 2F5 and 50-69 [Stamatatos et al.(1997)]
- et al.(1997)] be synergistic to have the greatest breadth and magnitude of response against 15 clade B primary isolates [Mascola 2F5: Using concentrations of Abs achievable in vivo, the triple combination of 2F5, 2G12 and HIVIG was found to
- 2F5: Used to standardize polyclonal response to CD4 BS [Turbica et al.(1997)]
- 2F5: The only MAb out of a large panel to show no correlation between Viral binding inhibition and neutralization [Ugolini et al.(1997)]
- to virus, given the neutralizing titres [Burton & Montefiori(1997)] only gp41 MAb that is neutralizing, it reacts with many non-B clade viruses and has a paradoxically weak binding 2F5: This review summarizes results about 2F5: it binds extracellularly, near the transmembrane domain, it is the
- infection [Andrus et al.(1998)] 2F5: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIV-1 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
- gp41 would typically project less from the surface of the virion and so be unable to interfere with attachment [Parren 2F5: This MAb and the results of [Ugolini et al.(1997)] are discussed - the authors propose that an Ab bound to
- 2F5: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could no 2G12, IgG1b12, 2F5 and 447-52D [Connor et al.(1998)] achieve 90% neutralization of the primary virus by which the individuals were ultimately infected - these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs

697 cont.

- 2F5: 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
- 2F5: A wide range of neutralizing titers was observed that was independent of co-receptor usage 2F5 was the most potent of the MAbs tested [Trkola et al.(1998)]
- 2F5: Points out that 2G12 and 2F5, potent neutralizing antibodies, were identified by screening for cell surface (oligomeric envelope) reactivity [Fouts et al.(1998)]
- 2F5: The ELDKWA epitope was inserted into the antigenic site B of influenza hemagglutinin and expressed on particularly specific immunogenic constructs - PELDKWAPP was a high affinity form selected by FACS [Ernst isolate the clone that displayed the epitope with the most markedly increased binding capacity for 2F5, to identify baculovirus infected insect cells, flanked by 3 additional random amino acids, xELDKWAxx - FACS was used to
- 2F5: Induces complement-mediated lysis in MN but not primary isolates primary isolates are refractive to CML [Takefman et al.(1998)]
- 2F5: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontin-F105 (CD4 BS) [Li et al.(1998)] uous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb
- 2F5: UK Medical Research Council AIDS reagent: ARP3063
- 2F5: NIH AIDS Research and Reference Reagent Program: 1475

MAb ID	Location	WEAU	Sequence Ne	Neutralizing	Immunogen	Species(Isotype)
698 polyclonal	gp41(662-667 BH10)	gp41(152-157)	ELDKWA	T	chimeric influenza virus/ELDKWA	murine(IgG,IgA)
	<ul> <li>Donor: Hermann Katinger, U. of Bodenkultur, or P Houston, TX, USA</li> <li>References: [Muster et al.(1994), Muster et al.(1995)]</li> <li>NOTES:</li> <li>Sustained ELDKWA specific IgA response in muc</li> </ul>	U. of Bodenkultur, 994), Muster et al.(1 ecific IgA response in	<ul> <li>Donor: Hermann Katinger, U. of Bodenkultur, or Polymun Scientific Inc., Vienna, Al Houston, TX, USA</li> <li>References: [Muster et al.(1994), Muster et al.(1995)]</li> <li>NOTES:</li> <li>Sustained ELDKWA specific IgA response in mucosa of immunized mice [Muster et</li> </ul>	nna, Austria; Vir ister et al.(1995)]	ustria; Viral Testing Systems, al.(1995)]	
699 B30	gp41(720-734 BH10)	gp41(210-224)	HLPIPRGPDRPEGIE		mis-folded LAI rgp160	$murine(IgG_1)$
	<ul><li>Donor: Gearoge Lewis</li><li>References: [Abacioglu et al.(1994)]</li><li>NOTES:</li><li>B30: Epitope boundaries mapped</li></ul>	I.(1994)] s mapped by peptide	or: Gearoge Lewis  rences: [Abacioglu et al.(1994)]  TES:  B30: Epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]	_		
700 B31	gp41(727-734 BH10)	gp41(217-224)	PDRPEGIE		mis-folded LAI rgp160	$murine(IgG_1)$
	Donor: Gearoge Lewis References: [Abacioglu et al.(1994)] NOTES:  • B31: Epitope boundaries mapped	I.(1994)] s mapped by peptide	<ul> <li>or: Gearoge Lewis</li> <li>erences: [Abacioglu et al.(1994)]</li> <li>TES:</li> <li>B31: Epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]</li> </ul>	J		
701 B33	gp41(727-734 BH10)	gp41(217-224) PDRPEGIE	PDRPEGIE	Z	Baculovirus- expressed mis- folded rgp160 IIIB:NL43, MicroGenSys	$\operatorname{murine}(\operatorname{IgG}_1)$
	Donor: Gearoge Lewis References: [Abacioglu et al.(1994), Bristow et al.(1994)] NOTES:  • B33: There are two MAbs in the literature named B33	I.(1994), Bristow et a bs in the literature na s manned by nentide	or: Gearoge Lewis  erences: [Abacioglu et al.(1994), Bristow et al.(1994)]  IES:  B33: There are two MAbs in the literature named B33. See also gp120, LAI 123-142  B33: Enitone boundaries manned by pentide scanning InG1 [Abacioglu et al.(1994)]		[Bristow et al.(1994)]	

MAb ID	Location	WEAU	Sequence	Neutralizing	
702 C8	gp41(727-732 BH10)	gp41(217-222)	PDRPEG	Z	mis-folded LAI rgp160
	Donor: Gearoge Lewis References: [Pincus & Mc NOTES:  • C8: Immunotoxin of C	Clure(1993), Pincus of the coupled to ricin-A	Donor: Gearoge Lewis  References: [Pincus & McClure(1993), Pincus et al.(1993), Abacioglu et al.(1994)]  NOTES:  • C8: Immunotoxin of C8 coupled to ricin-A does not mediate cells killing, and is not affected by sCD4 [Pincus & McClure(1993)]	(1994)] ng, and is not affe	ted by sCD4 [Pincus
	<ul> <li>C8: Immunotoxin of C McClure(1993)]</li> <li>C8: Ab response in III the dominant response binding this region do r</li> <li>C8: Epitope boundaries</li> </ul>	8 coupled to ricin-A 3 lab workers was commong vaccinees was ot neutralize, bind to mapped by peptide	C8: Immunotoxin of C8 coupled to ricin-A does not mediate cells killing, and is not affected by sCD4 [Pincus & McClure(1993)]  McClure(1993)]  C8: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – C8 was used as a control – the dominant response among vaccinees was to this mid-gp41 region, but not among the infected lab workers – Abs binding this region do not neutralize, bind to infected cells, nor serve as immunotoxins [Pincus et al.(1993)]  C8: Epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]	ng, and is not affective recipients – C8 that among the infimmunotoxins [Pin 994)]	s - C8 was used as a control – the infected lab workers – Abs ns [Pincus et al.(1993)]
703 88-158/02	gp41(732-752 IIIB)  Donor: Gearoge Lewis  References: [Niedrig et al.(1992a)]  NOTES:   88-158/02: Mild inhibition of	gp41(222-237) 1992a)] tion of <i>in vitro</i> activ	1(732-752 IIIB) gp41(222-237) GIEEEGGERDRDRSIR rgp41 IIIB  nor: Gearoge Lewis erences: [Niedrig et al.(1992a)]  TES: 88-158/02: Mild inhibition of in vitro activity at high MAb concentrations – profound enhancing activity at low	ions – profound e	rgp41 IIIB
704 88-158/022	gp41(732-752 IIIB) gp4  Donor: Gearoge Lewis  References: [Niedrig et al.(1992a)]  NOTES:  • 88-158/022: Mild inhibition of concentrations – significant reactions	gp41(222-237) 1992a)] ition of <i>in vitro</i> acticant reactivity to viri	1(732-752 IIIB) gp41(222-237) GIEEEGGERDRDRSIR rgp41 IIIB  nor: Gearoge Lewis erences: [Niedrig et al.(1992a)]  TES:  88-158/022: Mild inhibition of <i>in vitro</i> activity at high MAb concentrations – profound enhancing activity at low concentrations – significant reactivity to virion – domain non-immunogenic in humans [Niedrig et al.(1992a)]		or: Gearoge Lewis rences: [Niedrig et al.(1992a)]  TES:  88-158/02: Mild inhibition of <i>in vitro</i> activity at high MAb concentrations – profound enhancing activity at le concentrations – significant reactivity to virion – domain non-immunogenic in humans [Niedrig et al.(1992a)]
705 88-158/079	gp41(732-752 IIIB)  Donor: Gearoge Lewis  References: [Niedrig et al.(1992a)]	gp41(222-237) 1992a)]	gp41(222-237) GIEEEGGERDRDRSIR	tions – profound e nic in humans [Nie	hancing activity at ledrig et al.(1992a)] rgp41 IIIB hancing activity at ledrig et al.(1992a)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
706 B8	gp41(733-741 BH10)	gp41(223-231)	IEEEGGERD	Z	mis-folded LAI rgp160	$\operatorname{murine}(\operatorname{IgG}_1)$
	<b>Donor:</b> Gearoge Lewis <b>References:</b> [Pincus et al.(1993), Abacioglu et al.(1994)] <b>NOTES:</b>	993), Abacioglu et al	.(1994)]			
	<ul> <li>B8: Ab response in IIII the dominant response a binding this region do n</li> <li>B8: Epitope boundaries</li> </ul>	lab workers was co mong vaccinees was ot neutralize, bind to mapped by peptide s	B8: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – B8 was used as a compared the dominant response among vaccinees was to this mid-gp41 region, but not among the infected lab worker binding this region do not neutralize, bind to infected cells, nor serve as immunotoxins [Pincus et al.(1993)] B8: Epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]	recipients – B8 t among the inf nunotoxins [Pin	– B8 was used as a control – he infected lab workers – Abs s [Pincus et al.(1993)]	Ø
707 LA9 (121-	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	?	murine(IgM)
101)	<b>Donor:</b> Gearoge Lewis <b>References:</b> [Evans et al.(1989)]	89)]				
708 ED6	gp41(735-752 IIIB)  Donor: Gearoge Lewis  References: [Evans et al.(1989)]	gp41(218-235) 89)]	DRPEGIEEEGGERDRDRS	N	?	murine(IgM)
709 1575	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Poliovirus/gp41 peptide chimera	murine
	<ul> <li>Donor: Gearoge Lewis</li> <li>References: [Evans et al.(1989), Vella et al.(1993), Buratti et al.(1997)]</li> <li>NOTES: <ul> <li>1575: Neutralizing activity, less broad than 1577 [Evans et al.(1989)</li> <li>1575: Core epitope: IEEE – neutralized IIIB, but not RF or MN [Vella et al.(1989)</li> <li>1575: Study shows that MAb 1575 can recognize the IEEE sequence p17 protein – motif is conserved in both regions in different HIV-1 or p17 protein – motif is conserved in both regions in different HIV-1 or p18 p18 p19 p19 p19 p19 p19 p19 p19 p19 p19 p19</li></ul></li></ul>	789), Vella et al.(1993) Tity, less broad than 1 TE – neutralized IIIB MAb 1575 can recog	or: Gearoge Lewis  rences: [Evans et al.(1989), Vella et al.(1993), Buratti et al.(1997)]  IES:  1575: Neutralizing activity, less broad than 1577 [Evans et al.(1989)]  1575: Core epitope: IEEE – neutralized IIIB, but not RF or MN [Vella et al.(1993)]  1575: Study shows that MAb 1575 can recognize the IEEE sequence in both gp41, and in the HPG30 region of the p17 protein – motif is conserved in both regions in different HIV-1 clades [Buratti et al.(1997)]	.(1993)] h gp41, and in t	the HPG30 region of th	O
	<b>*</b>	,	,			
710 1576	gp41(735-752 IIIB)  Donor: Gearnoge Lewis	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Poliovirus/gp41 peptide chimera	murine
	<ul> <li>Donor: Gearoge Lewis</li> <li>References: [Vella et al.(1993)]</li> <li>NOTES:</li> <li>1576: Not neutralizing [Vella et al.(1993)]</li> </ul>	Vella et al.(1993)]				

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
711 1577	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Poliovirus/gp41 peptide chimera	murine
	<ul> <li>Donor: Morag Ferguson (NIBSC)</li> <li>References: [Evans et al.(1989), D'Souza et al.(1991), Vella et al.(1993)]</li> <li>NOTES: <ul> <li>1577: Raised against IIIB peptide chimera – neutralized African and A</li> <li>1577: Non-neutralizing in this multi-lab study [D'Souza et al.(1991)]</li> <li>1577: Core epitope: ERDRD – could neutralize HIV IIIB and HIV R</li> <li>1577: UK Medical Research Council AIDS reagent: ARP317</li> </ul> </li> </ul>	BSC)  89), D'Souza et al.(1  3 peptide chimera – r in this multi-lab stud  DRD – could neutral arch Council AIDS 1	or: Morag Ferguson (NIBSC)  Prences: [Evans et al.(1989), D'Souza et al.(1991), Vella et al.(1993)]  Pres:  1577: Raised against IIIB peptide chimera – neutralized African and American HIV-1 lab st 1577: Non-neutralizing in this multi-lab study [D'Souza et al.(1991)]  1577: Core epitope: ERDRD – could neutralize HIV IIIB and HIV RF [Vella et al.(1993)]  1577: UK Medical Research Council AIDS reagent: ARP317		lab strains [Evans et al.(1989)] 993)]	<u> </u>
712 1578	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Poliovirus/gp41 peptide chimera	murine
	<ul> <li>Donor: Morag Ferguson (NIBSC)</li> <li>References: [Evans et al.(1989), Vella et al.(1993)]</li> <li>NOTES:</li> <li>1578: No neutralizing activity – epitope may be</li> <li>1578: Core epitope: IEEE – in this study, neutralizing activity – epitope may be</li> </ul>	BSC)  89), Vella et al.(1993) tivity – epitope may E – in this study, ne	or: Morag Ferguson (NIBSC)  erences: [Evans et al.(1989), Vella et al.(1993)]  FES:  1578: No neutralizing activity – epitope may be formed by regions from both poliovirus and HIV [Evans et al.(1989)]  1578: Core epitope: IEEE – in this study, neutralized IIIB, but not RF or MN [Vella et al.(1993)]	poliovirus and HIV [ N [Vella et al.(1993)]	HIV [Evans et al.(1989)]	
713 1899	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Poliovirus/gp41 peptide chimera	murine
	Donor: Morag Ferguson (NIBSC) References: [Vella et al.(1993)] NOTES:  • 1899: Could neutralize HIV IIIB and HIV RF [Vella et al.(1993)]	BSC) (3)] HIV IIIB and HIV R	F [Vella et al.(1993)]			
714 1579	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	N	Poliovirus/gp41 peptide chimera	murine
	Donor: Morag Ferguson (NIBSC)  References: [Vella et al.(1993)]  NOTES:  1579: Core enitone: IEEE – neutralized IIIR, but not RE or MN [Vella et al.(1993)]	BSC) (3)]				

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
715 1583	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Poliovirus/gp41	murine
	Donor: Morag Ferguson (NIBSC) References: [Evans et al.(1989), Vella et al.(1993), Sattentau et al.(1995)] NOTES:	SC) 9), Vella et al.(1993	)), Sattentau et al.(1995)]		рериае спинета	
	<ul> <li>1583: Neutralizing activity, less broad than 1577 [Evans et al.(1989)]</li> <li>1583: Core epitope: ERDRD – Could neutralize HIV IIIB but not HI</li> <li>1583: Cytoplasmic domain, epitope not exposed at the surface of HIV</li> </ul>	y, less broad than 1 RD – Could neutra n, epitope not expc	<ul> <li>1583: Neutralizing activity, less broad than 1577 [Evans et al.(1989)]</li> <li>1583: Core epitope: ERDRD – Could neutralize HIV IIIB but not HIV RF [Vella et al.(1993)]</li> <li>1583: Cytoplasmic domain, epitope not exposed at the surface of HIV-1 infected cells [Sattentau et al.(1995)]</li> </ul>	Vella et al.(1993)] cted cells [Sattent	93)] :entau et al.(1995)]	
716 1907	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Poliovirus/gp41	murine
	Donor: Morag Ferguson (NIBSC) References: [Vella et al.(1993)]	SC) )]			peptide chimera	
	NOTES: • 1907: Could not neutralize HIV IIIB, RF or MN [Vella et al.(1993)]	e HIV IIIB, RF or l	MN [Vella et al.(1993)]			
717 1908	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Poliovirus/gp41	murine
	Donor: Morag Ferguson (NIBSC) References: [Evans et al.(1989), Vella et al.(1993), Sattentau et al.(1995)]	SC) 9), Vella et al.(1993	)), Sattentau et al.(1995)]		peptide chimera	
	<ul> <li>1908: Neutralized IIIB, but not RF or MN [Vella et al.(1993)]</li> <li>1908: Cytoplasmic domain, epitope not exposed at the surface</li> </ul>	nt not RF or MN [Vn, epitope not expo	1908: Neutralized IIIB, but not RF or MN [Vella et al.(1993)] 1908: Cytoplasmic domain, epitope not exposed at the surface of HIV-1 infected cells [Sattentau et al.(1995)]	cted cells [Satt	entau et al.(1995)]	
718 1909	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Poliovirus/gp41	murine
	Donor: Morag Ferguson (NIBSC) References: [Vella et al.(1993)]	SC) )]			рериче спинста	
	• 1909: Neutralized HIV IIIB but not HIV RF [Vella et al.(1993)]	B but not HIV RF	[Vella et al.(1993)]			
719 41-1	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Peptide 735-752	$\mathrm{murine}(\mathrm{Ig} M_\kappa)$
	Donor: Morag Ferguson (NIBSC) References: [Dalgleish et al.(1988)] NOTES:	SC) [988)]			E 6	
	<ul> <li>41-1: This antibody gp41(735-752 IIIB) [Dalgleish et al.(1988)] seem MAb to gp41(584-609) [Mani et al.(1994)]</li> <li>41-1: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish et al.(1988)]</li> </ul>	(735-752 IIIB) [Da 4ani et al.(1994)] 11 not HIV-2 strain	41-1: This antibody gp41(735-752 IIIB) [Dalgleish et al.(1988)] seems to have been MAb to gp41(584-609) [Mani et al.(1994)] 41-1: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish et al.(1988)]		named the same as a different	nt

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
720 41-2	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Peptide 735-752	$\mathrm{murine}(\mathrm{Ig}\mathrm{M}_\kappa)$
	Donor: Morag Ferguson (NIBSC) References: [Dalgleish et al.(1988)] NOTES:  • 41-2: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish et al.(1988)]	3SC) (1988)] but not HIV-2 strain	s [Dalgleish et al.(1988)]			
721 41-3	gp41(735-752 IIIB)	gp41(218-235)	DRPEGIEEEGGERDRDRS	Z	Peptide 735-752	$\mathrm{murine}(\mathrm{Ig}\mathbf{M}_\kappa)$
	Donor: Morag Ferguson (NIBSC) References: [Dalgleish et al.(1988)] NOTES:  • 41-3: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish et al.(1988)]	3SC) (1988)] but not HIV-2 strain	s [Dalgleish et al.(1988)]			
722 4E10	gp41(824-830 BH10) gr <b>Donor:</b> Morag Ferguson (NIBSC) <b>References:</b> [Buchacher et al.(199	gp41(313-319) 3SC) .(1992), Buchacher	gp41(824-830 BH10) gp41(313-319) AEGTDRV  Donor: Morag Ferguson (NIBSC)  References: [Buchacher et al.(1992), Buchacher et al.(1994), D'Souza et al.(1994)]	N 94)]	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{3\kappa})$
	<ul> <li>4E10: MAbs generated b II proteins – anti-class II</li> <li>4E10: Included in a mu [D'Souza et al.(1994)]</li> </ul>	y electrofusion of PI Abs are only found lti-lab study for an	4E10: MAbs generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells – also binds to MHC class II proteins – anti-class II Abs are only found in HIV-1 positive people [Buchacher et al.(1994)] 4E10: Included in a multi-lab study for antibody characterization, binding and neutralization assay comparison [D'Souza et al.(1994)]	h CB-F7 cells – nacher et al.(19 ig and neutrali	cells – also binds to MHC class al.(1994)] eutralization assay comparison	
723 DZ	gp41(827-860 BRU) <b>Donor:</b> ?	gp41(312-345)	VAEGTDRVIEVVQGACR- AIRHIPRRIRQGLERIL?	L	rec vaccinia gp160 IIIB	$\operatorname{human}(\operatorname{IgG}_{1\lambda})$
	References: [Boyer et al.(1991)] NOTES:  • DZ: Weakly neutralizing IIII RF [Boyer et al.(1991)]	IIIB – binds to pept	res: [Boyer et al.(1991)]  TES:  DZ: Weakly neutralizing IIIB – binds to peptides 827-843 and 846-860 of BRU – reacted specifically with IIIB and RF [Boyer et al.(1991)]	3RU – reacted s	pecifically with IIIB and	
724 Chessie 8	gp41(cytoplasmic domain)	gp41	VAEGTDRVIEVVQGACR- AIRHIPRRIRQGLERIL?			murine(IgG)
	References: [Lewis et al.(1991), Poumbourios et al.(1995) NOTES:  • Chessie 8: Used to precipitate gp160 in immunoblots in glycoprotein as an immunogen [Rovinski et al.(1995)]	)1), Poumbourios et itate gp160 in immu nogen [Rovinski et a	References: [Lewis et al.(1991), Poumbourios et al.(1995), Rovinski et al.(1995)]  NOTES:  Chessie 8: Used to precipitate gp160 in immunoblots in a study examining the feasibility of using unprocessed gp160 glycoprotein as an immunogen [Rovinski et al.(1995)]	e feasibility of	using unprocessed gp160	

727 126-50 gp41(dis ]	726 T30 gp41(dis) Donor: ? Reference NOTES: • T30: glycc HIV-	725 K14 gp41(dis)  Donor: ?  Reference et al.(1997)  NOTES:  K14:  with sera f  K14:  et al.(4)  K14:  F14:  F14:  F14:  F14:	MAb ID Location
gp41 (dis HXB2)  Donor: ?  References: [Robinson Jr. et al NOTES:  • 126-50: No enhancing acti • 126-50: Serves as target fo	gp41(dis) gp41(dis) D Donor: ? References: [Earl et al.(1994), Earl et al.(1997)] NOTES:  • T30: binds to the region 580 to 640, but does glycosylation of Asn 616 – no other antibod HIV+ individuals [Earl et al.(1997)]	gp41(dis) gp41(dis) DISCONT Donor:?  References: [Teeuwsen et al.(1990), Schutten et al.(1981)] et al.(1997)]  NOTES:  • K14: Did not bind to peptides spanning gp41, but it d with HIV-2 – competition experiments showed this sera from Europe and Africa [Teeuwsen et al.(1990)]  • K14: Reduced affinity for both SI and NSI viruses re et al.(1995b)]	ion
gp41(dis) .(1990a), Tyler .ity for HIV-1 : antibody-dependralizing acti	gp41(dis) Earl et al.(199) to 640, but do no other antib	gp41(dis) 1990), Schutte 1990), Schutte les spanning gr experiments sh a [Teeuwsen e ooth SI and NS	WEAU
gp41(dis HXB2) gp41(dis) DISCONTINUOUS N  Donor: ?  References: [Robinson Jr. et al.(1990a), Tyler et al.(1990), Robinson Jr. et al.(1991), Xv  NOTES:  • 126-50: No enhancing activity for HIV-1 IIIB [Robinson Jr. et al.(1990a)]  • 126-50: Serves as target for antibody-dependent cellular cytotoxicity ADCC [Tyler 6  • 126-50: No enhancing or neutralizing activity [Robinson Jr. et al.(1991)]	I(dis) gp41(dis) DISCONTINUOUS N  or: ? erences: [Earl et al.(1994), Earl et al.(1997)]  TES: T30: binds to the region 580 to 640, but does not bind to peptides spanning this region glycosylation of Asn 616 – no other antibody tested inhibited binding, but binding of HIV+ individuals [Earl et al.(1997)]	I(dis)  gp41(dis)  gp41(dis)  DISCONTINUOUS  N  1  erences: [Teeuwsen et al.(1990), Schutten et al.(1995a), Schutten et al.(1995b), Schutten et al.(1996), Schutten et al.(1997)]  TES:  K14: Did not bind to peptides spanning gp41, but it does not react with env deletion mutant 643-692 – does not react with HIV-2 – competition experiments showed this was an immunodominant conserved epitope in HIV-1 positive sera from Europe and Africa [Teeuwsen et al.(1990)]  K14: Reduced affinity for both SI and NSI viruses relative to MAb MN215, failed to neutralize SI strain [Schutten et al.(1995b)]  K14: In a study of NSI and SI virus neutralization, K14 did not influence viral entry [Schutten et al.(1997)]	Sequence
N al.(1991), Xu ei al.) a)] DCC [Tyler et :	N ng this region – but binding cou	N al.(1995b), Sch and deletion mu minant conserv v215, failed to 1 ce viral entry [S	Neutralizing
HIV-1 infection  1 et al.(1991)]  et al.(1990)]	tetrameric Env	Schutten et al.(1996), Schutten mutant 643-692 – does not react erved epitope in HIV-1 positive to neutralize SI strain [Schutten [Schutten et al.(1997)]	Immunogen
$\operatorname{human}(\operatorname{IgG}_{2\kappa})$	murine nked from	$\begin{array}{c} human(IgG_1) \\ \\ utten \\ \\ react \\ \\ sitive \\ \\ \\ utten \end{array}$	Species(Isotype)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
728 T4	gp41(dis IIIB)	gp41(dis)	DISCONTINUOUS	T	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)
	Donor: ? References: [Earl et al.(19 Otteken et al.(1996)] NOTES:	94), Broder et al.(1	<b>Donor:</b> ? <b>References:</b> [Earl et al.(1994), Broder et al.(1994), Richardson Jr et al.(1996), Weissenhorn et al.(1996), Earl et al.(1997), Otteken et al.(1996)] <b>NOTES:</b>	6), Weissenhorn et	al.(1996), Earl et al.(199	97),
	<ul> <li>T4: one of five MAbs (T4, T6, T9, T10 and T35) in a co epitope in gp41 and is oligomer specific – neutralizes IIIB :</li> <li>T4: Does not bind to soluble monomeric gp41(21-166) that the oligomer gp140, as does T6 [Weissenhorn et al.(1996)]</li> </ul>	(T4, T6, T9, T10 oligomer specific oluble monomeric does T6 [Weisser	T4: one of five MAbs (T4, T6, T9, T10 and T35) in a competition group that bind to a conformation-dependent epitope in gp41 and is oligomer specific – neutralizes IIIB and SF2 [Broder et al.(1994)] T4: Does not bind to soluble monomeric gp41(21-166) that lacks the fusion peptide and membrane anchor, only to the oligomer gp140, as does T6 [Weissenhorn et al.(1996)]	group that bind to roder et al.(1994)] fusion peptide and	a conformation-depend membrane anchor, only	lent y to
	<ul> <li>T4: This antibody, along with 7 others (M10, D D61, and the human MAb 246-D, which both bit of these antibodies are oligomer dependent – all c of this competition group are blocked by sera fro</li> <li>MAbs T4 and T6 bind only to oligomer, and pull requiring one to two hours [Otteken et al.(1996)]</li> </ul>	ng with 7 others (Ab 246-D, which oligomer depende up are blocked by only to oligomer, Otteken et al	T4: This antibody, along with 7 others (M10, D41, D54, T6, T9, T10 and T35), can block the linear murine MAb D61, and the human MAb 246-D, which both bind to the immunodominant region near the two Cys in gp41 – most of these antibodies are oligomer dependent – all of the MAbs are reactive with ten different HIV-1 strains – members of this competition group are blocked by sera from HIV-1+ individuals [Earl et al.(1997)] MAbs T4 and T6 bind only to oligomer, and pulse chase experiments indicate that the epitope is very slow to form, requiring one to two hours [Otteken et al.(1996)]	and T35), can bluinant region near we with ten differe s [Earl et al.(1997) indicate that the ep	ock the linear murine M the two Cys in gp41 – m nt HIV-1 strains – memb  j pitope is very slow to for	[Ab 10st 1ers rm,
729 D12	gp41(dis IIIB)	gp41(dis)	DISCONTINUOUS	L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)
	Donor: ? References: [Broder et al. NOTES:	1994), Richardson	<b>Donor:</b> ? <b>References:</b> [Broder et al.(1994), Richardson Jr et al.(1996), Earl et al.(1997), Otteken <b>NOTES:</b>	_	et al.(1996)]	
	<ul> <li>D12: One of 18 MA preferentially, but not</li> <li>D12: This antibody was in a client provider of the preference of the preferenc</li></ul>	os (e. g. D4 and exclusively, to oligous blocked more st	D12: One of 18 MAbs (e. g. D4 and D40) that bind to a conformation-dependent epitope in gp41 that bind preferentially, but not exclusively, to oligomers – neutralizes IIIB and SF2 [Broder et al.(1994)] D12: This antibody was blocked more strongly by human sera than other anti-gp41 MAbs (D20, D43, D61, and T4) in a light profession and the strong of the strong stro	mation-dependent SF2 [Broder et al.ı ner anti-gp41 MAt	epitope in gp41 that b (1994)] s (D20, D43, D61, and	nind T4)
	<ul> <li>n a oligometric ELISA assay [Richardson Jr et al. (1996)]</li> <li>D12: MAbs D10 and D12 are very easily blocked by hun</li> <li>D12: MAbs D4, D10, D11, D12, and D41 all bind only binding to noncleavable gp160 revealed that these MAbs min [Otteken et al. (1996)]</li> </ul>	assay [Kichardso assay [Kichardso D12 are very easily D11, D12, and E e gp160 revealed	In a oligometric ELISA assay [Richardson Jr et al.(1996)]  D12: MAbs D10 and D12 are very easily blocked by human sera from HIV+ individuals [Earl et al.(1997)]  D12: MAbs D4, D10, D11, D12, and D41 all bind only to complete oligomer – pulse label experiments of MAb binding to noncleavable gp160 revealed that these MAbs bound with a delay, epitopes forming with a half life of 30 min [Otteken et al.(1996)]	n HIV+ individuals oligomer – pulse ı delay, epitopes fo	[Earl et al.(1997)] label experiments of M rming with a half life of	fAb 330

732 126-6	731 D16	730 DI	MAb ID
gp41(dis HXB2) gp41(dis) DISCONTINUOU! Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Robinson Jr. et al.(1990a), Robinson Jr. et al.(197)] et al.(1995), Binley et al.(1996), Earl et al.(1997)] NOTES:  126-6: No enhancing activity for HIV-1 IIIB [Robinson Jr. et al.(1996)] 126-6: No enhancing or neutralizing activity [Robinson Jr. et al.(1991)] 126-6: Specific for a conformational epitope [Xu et al.(1991)] 126-6: Called SZ-126.6 [Eddleston et al.(1993)] 126-6: One of several anti-gp41 MAbs that bind to a gp41-relucine zipper domain of gp41, showing that the construct has et al.(1995)] 126-6: Discontinuous epitope recognizing residues between (T3, M12, M15, S6, S8, S9, S10 block binding [Binley et al.(1995)] 126-6: NIH AIDS Research and Reference Reagent Program:	gp41(dis IIIB) gp41(dis) DISO  Donor: ?  References: [Earl et al.(1994), Weissenhorn et al.(1)  NOTES:  • D16: Precipitates both oligomeric gp140 and membrane anchor, along with MAbs D16, D38,  • D16: One of eleven MAbs (D16, D17, D31, D36)  dependent and that can block the binding of the with 9/10 HIV-1 strains all except HIV-1 ADA, of binding (ELLE to DLLA) [Earl et al.(1997)]	gp41(dis IIIB) g  Donor: ?  References: [Otteken et al.(1996)]  NOTES:  D1: MAbs D1, D16, had T37  to noncleavable gp160 reveale [Otteken et al.(1996)]	Location
gp41(dis) ; NYU Med Cent et al.(1990a), F 996), Earl et al.(19 106), Earl et al.(19 107) ctivity for HIV-1 r neutralizing action formational epit f [Eddleston et al. anti-gp41 MAbs of gp41, showing epitope recognizing spitope recognizing spitope and Referen	gp41(dis)  )4), Weissenhorn  oligomeric gp14  g with MAbs D1, Abs (D16, D17, I  block the binding  all except HIV-1  LLA) [Earl et al.(	gp41(dis) (1996)] ad T37 bind to crevealed that the	WEAU
gp41(dis) DISCONTINUOUS N HIV-1 infection I nor: Susan Zolla-Pazner, NYU Med Center, NY, NY berences: [Robinson Jr. et al.(1990a), Robinson Jr. et al.(1991), Xu et al.(1991), Eddleston et al.(1993), Chen L(1995), Binley et al.(1996), Earl et al.(1997)]  TES:  126-6: No enhancing activity for HIV-1 IIIB [Robinson Jr. et al.(1990a)]  126-6: Specific for a conformational epitope [Xu et al.(1991)]  126-6: Called SZ-126.6 [Eddleston et al.(1993)]  126-6: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen et al.(1995)]  126-6: Discontinuous epitope recognizing residues between 649-668 – designated cluster II – Fabs D5, D11, G1, T3, M12, M15, S6, S8, S9, S10 block binding [Binley et al.(1996)]  126-6: NIH AIDS Research and Reference Reagent Program: 1243	pp41(dis IIIB)  gp41(dis)  dimeric Env  pponor: ?  References: [Earl et al.(1994), Weissenhorn et al.(1996), Earl et al.(1997)]  NOTES:  • D16: Precipitates both oligomeric gp140 and soluble monomeric gp41(21-166)that lacks the fusion peptide and membrane anchor, along with MAbs D16, D38, D40, D41, and D54 [Weissenhorn et al.(1996)]  • D16: One of eleven MAbs (D16, D17, D31, D36, D37, D40, D44, D55, D59, T37, and T45) that are conformation dependent and that can block the binding of the MAb D50 that binds to the linear peptide gp41(642-665) – reactive with 9/10 HIV-1 strains all except HIV-1 ADA, which has the change E659D and E662A that may result in the loss of binding (ELLE to DLLA) [Earl et al.(1997)]	l(dis IIIB)  gp41(dis)  discontinuous  vaccinia expressed oligomeric gp140  IIIB  res:  [Otteken et al.(1996)]  D1: MAbs D1, D16, had T37 bind to oligomeric gp160 equally well – pulse label experiments of MAb binding to noncleavable gp160 revealed that these MAbs bound with a delay, epitopes forming with a half life of 30 min [Otteken et al.(1996)]	Sequence
u et al.(1991), E  a)] binding fusion pul aspects of norm  - designated clu	L  41(21-166)that 1: Weissenhorn et al. 55, D59, T37, and o the linear peptic E659D and E662	I – pulse label en epitopes formin	Neutralizing
HIV-1 infection , Eddleston et al.(1993), Chen n protein designed to study the nmal gp41 conformation [Chen cluster II – Fabs D5, D11, G1,	dimeric Env  at lacks the fusion peptide and t al.(1996)] and T45) that are conformation eptide gp41(642-665) – reactive 662A that may result in the loss	vaccinia expressed oligomeric gp140 IIIB  g with a half life of 30	Immunogen
$\operatorname{human}(\operatorname{IgG}_{2\kappa})$ Chen $\operatorname{Chen}$ $\operatorname{Chen}$ $\operatorname{Chen}$ $\operatorname{Chen}$ $\operatorname{G1}$	murine(IgG) and ation active loss	murine(IgG) nding min	Species(Isotype)

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
733 D43	gp41(dis HXB2) gp41(dis) DISCONTINUOUS Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Earl et al.(1994), Richardson Jr et al.(1996), Earl et al.(1997)] NOTES:	gp41(dis) VYU Med Cente , Richardson Jr	DISCONTINUOUS r, NY, NY et al.(1996), Earl et al.(1997)]		dimeric Env	murine(IgG)
	<ul> <li>D43: This is a linear gp41 epitope, mapping ELISA assay to a similar extent for gp41 MAb</li> <li>D43: Partially conformation dependent – doesn – binding can be blocked by MAbs T3, D38 a strains, not binding to JRFL [Earl et al.(1997)]</li> </ul>	11 epitope, map extent for gp41 on dependent – by MAbs T3, 1 TL [Earl et al.(1	D43: This is a linear gp41 epitope, mapping in the region 635-678 – human sera blocked binding ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 [Richardson Jr et al.(1996)] D43: Partially conformation dependent – doesn't bind to short peptides, but does bind to the region spa – binding can be blocked by MAbs T3, D38 and D45 – MAbs in this competition group reacted w strains, not binding to JRFL [Earl et al.(1997)]		blocked binding in oligomeric n Jr et al.(1996)] 1 to the region spanning 641-683 group reacted with 9/10 HIV-1	meric 1-683 HV-1
734 T3	gp41(dis HXB2) gp41(dis) DISCON Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY References: [Earl et al.(1994), Earl et al.(1997)] NOTES:	gp41(dis) VYU Med Cente , Earl et al.(199	DISCONTINUOUS r, NY, NY 7)]		tetrameric Env	murine(IgG)
	<ul> <li>T3: Partially conformation dependent – doesn'</li> <li>– binding can be blocked by MAbs D43, D38 strains, not binding to JRFL [Earl et al.(1997)]</li> </ul>	n dependent – d by MAbs D43, FL [Earl et al.(1	T3: Partially conformation dependent – doesn't bind to short peptides, but does bind – binding can be blocked by MAbs D43, D38 and D45 – MAbs in this competition strains, not binding to JRFL [Earl et al.(1997)]		to the region spanning 641-683 group reacted with 9/10 HIV-1	1-683 HIV-1
735 Md-1	gp41(dis) gp41(dis) DISCONTINUOUS Donor: R. A. Myers State of Maryland Dept. of Health References: [Myers et al.(1993), Chen et al.(1995), Binley et al.(1996)] NOTES:	gp41(dis) Maryland Dept. 93), Chen et al.(	DISCONTINUOUS of Health 1995), Binley et al.(1996)]	Z	?	$\operatorname{human}(\operatorname{Ig} G_{1\lambda})$
	<ul> <li>Md-1: Also called MD-1</li> <li>Md-1: Called MD-1 – dis [Myers et al.(1993)]</li> <li>Md-1: Called MD-1 – one</li> </ul>	continuous epit	Md-1: Also called MD-1  Md-1: Called MD-1 – discontinuous epitope that binds in the N-terminal region – reacts exclusively with oligomer [Myers et al.(1993)]  Md-1: Called MD-1 – one of several anti-onal 1 Mahs that hind to a grad 1-maltose hinding fusion protein designed to	al region – reacts	exclusively with olig	gomer gomer
	study the leucine zipper de [Chen et al.(1995)]	on several allu- omain of gp41, s	study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen et al.(1995)]	etained aspects of	ts of normal gp41 conformation	nation
	<ul> <li>Md-1: Discontinuous epitope recognizing residues between 563-672, does not recognize cluster I disulfide bridge region – reacts almost exclusively with trimers and tetramers on WB – designated cluster II – Fabs D5, D11, G1, T3, M12, M15, S6, S8, S9, S10 block binding [Binlev et al.(1996)]</li> </ul>	tope recognizin lusively with tri	Md-1: Discontinuous epitope recognizing residues between 563-672, does not recognize cluster I disulfide bridge	does not recognizesignated cluster	e cluster I disulfide l	oridge 1. T3.

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
736 Fab D5	gp41(dis LAI) gp41(dis) DISCON Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996)] NOTES:  • Fab D5: Binds to Cluster II region – competes with	gp41 (dis) faryland Dept.  for of (1996)	941(dis LAI) gp41(dis) DISCONTINUOUS N HIV-1 infection lonor: R. A. Myers State of Maryland Dept. of Health eferences: [Binley et al.(1996)]  OTES:  • Fab D5: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable	N and D50 – confo	HIV-1 infection rmation sensitive – va	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$ riable
737 Fab D11	gp41(dis LAI) gp41(dis) DISCON Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996)] NOTES:  • Fab D11: Binds to Cluster II region – competes with regions sequenced [Binley et al.(1996)]	gp41(dis) faryland Dept. 5)] Il region – com et al.(1996)]	I(dis LAI) gp41(dis) DISCONTINUOUS N HIV-1 infection I nor: R. A. Myers State of Maryland Dept. of Health grences: [Binley et al.(1996)]  TES: Fab D11: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	N l and D50 – confc	HIV-1 infection  rmation sensitive – va	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$ riable
738 Fab G1	gp41(dis LAI) gp41(dis) DISCON Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996)] NOTES:  • Fab G1: Binds to Cluster II region – competes with	gp41(dis) flaryland Dept.  flaryland Dept.  fregion – comp	l(dis LAI) gp41(dis) DISCONTINUOUS N HIV-1 infection  or: R. A. Myers State of Maryland Dept. of Health  erences: [Binley et al.(1996)]  TES: Fab G1: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable	N and $D50$ – confo	HIV-1 infection rmation sensitive – va	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$ riable
739 Fab T3	gp41(dis LAI) gp41(dis) DISCON Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996)] NOTES: • Fab T3: Binds to Cluster II region – competes with regions sequenced [Binley et al.(1996)]	gp41(dis) faryland Dept. 5)] region – compet al.(1996)]	I(dis LAI) gp41(dis) DISCONTINUOUS N HIV-1 infection lor: R. A. Myers State of Maryland Dept. of Health erences: [Binley et al.(1996)]  TES: Fab T3: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	N and D50 – confo	HIV-1 infection rmation sensitive – va-	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$ riable
740 Fab M10	gp41(dis LAI)  Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996), Parren et al.(1997b)]  NOTES:  Fab M10: Binds to Cluster II region – competes with regions sequenced [Binley et al.(1996)]  Fab M10: Does not bind to MN native oligomer, et al.(1997b)]	gp41(dis) faryland Dept. 5), Parren et al. Il region – com et al.(1996)] to MN native c	I(dis LAI)  gp41(dis)  DISCONTINUOUS  N  HIV-1 infection  1  tor: R. A. Myers State of Maryland Dept. of Health  erences: [Binley et al.(1996), Parren et al.(1997b)]  TES:  Fab M10: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]  Fab M10: Does not bind to MN native oligomer, but does bind to both LAI and MN rgp120 and rgp140 [Parren et al.(1997b)]	N I and D50 – confc th LAI and MN	HIV-1 infection  rmation sensitive – var  rgp120 and rgp140 [F	$ ext{human}( ext{Ig}G_{1\kappa})$ riable

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
741 Fab M12	gp41(dis LAI) gp41(dis) DISCON Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996)] NOTES:  • Fab M12: Binds to Cluster II region – competes with regions sequenced [Binley et al.(1996)]	gp41(dis) Maryland Dept. 6)] Il region – comet al.(1996)]	41(dis LAI)  gp41(dis)  DISCONTINUOUS  N  HIV-1 infection  I  ferences: [Binley et al.(1996)]  TES:  Fab M12: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	N l and D50 – confc	HIV-1 infection rmation sensitive – va	$\operatorname{human}(\operatorname{IgG}_{\operatorname{1}_{K}})$ riable
742 Fab M15	gp41(dis LAI) gp41(dis) DISCON Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996)]  NOTES:  • Fab M15: Binds to Cluster II region – competes with regions sequenced [Binley et al.(1996)]	gp41(dis) Maryland Dept. 6)] Il region – comet al.(1996)]	I(dis LAI) gp41(dis) DISCONTINUOUS N HIV-1 infection 1  nor: R. A. Myers State of Maryland Dept. of Health  erences: [Binley et al.(1996)]  TES: Fab M15: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	N l and D50 – confc	HIV-1 infection	$ ext{human}( ext{IgG}_{1\kappa})$ riable
	rogroms sochections [muno)	Ct an(1))0/]				
743 Fab S6	gp41(dis LAI) gp41(dis) DISCON Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996)] NOTES: • Fab S6: Binds to Cluster II region – competes with regions sequenced [Binley et al.(1996)]	gp41(dis) Maryland Dept. 6)] I region – compret al.(1996)]	I(dis LAI) gp41(dis) DISCONTINUOUS N HIV-1 infection I or: R. A. Myers State of Maryland Dept. of Health erences: [Binley et al.(1996)]  TES: Fab S6: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	N and D50 – confo	HIV-1 infection rmation sensitive – va	$ ext{human}( ext{IgG}_{1\kappa})$ uriable
744 Fab S8	gp41(dis LAI) gp41(dis) DISCON Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996)] NOTES:	gp41(dis) Maryland Dept. 6)]	DISCONTINUOUS of Health	Z	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
	• Fab S8: Binds to Cluster II region – cor regions sequenced [Binley et al.(1996)]	I region – comp et al.(1996)]	Fab S8: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	and D50 – confo	rmation sensitive – va	uriable
745 Fab S9	gp41(dis LAI)  gp41(dis) DISCON  Donor: R. A. Myers State of Maryland Dept. of Health  References: [Rinley et al. (1996)]	gp41(dis) Maryland Dept.	DISCONTINUOUS of Health	Z	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$
	NOTES:	6)]				

MAb ID	Location	WEAU	Sequence 1	Neutralizing	Immunogen	Species(Isotype)
746 Fab S10	gp41(dis LAI) gp41(dis) DISCON Donor: R. A. Myers State of Maryland Dept. of Health References: [Binley et al.(1996)] NOTES:  • Fab S10: Binds to Cluster II region – competes with regions sequenced [Binley et al.(1996)]	gp41(dis) laryland Dept.  [5] Iregion – compet al.(1996)]	1 (dis LAI) gp41(dis) DISCONTINUOUS N HIV-1 infection 1 nor: R. A. Myers State of Maryland Dept. of Health Perences: [Binley et al.(1996)]  TES: Fab S10: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	N nd D50 – confc	HIV-1 infection rmation sensitive – vari	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$ iable
747 Fab L2	gp41(dis LAI) gp41(dis) DISCONTINUOUS  Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California References: [Binley et al.(1996), Earl et al.(1997)]  NOTES:  • Fab L2: Binds to Cluster III region – competes with MAb Md-1, but not sensitive – variable regions sequenced [Binley et al.(1996)]	gp41(dis) (Scripps Rese. 5), Earl et al.(19 III region – co sequenced [Bi	I(dis LAI)  gp41(dis)  DISCONTINUOUS  N  nor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California erences: [Binley et al.(1996), Earl et al.(1997)]  TES:  Fab L2: Binds to Cluster III region – competes with MAb Md-1, but not MAbs sensitive – variable regions sequenced [Binley et al.(1996)]	N MAbs ]	HIV-1 infection 1	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$ ation
748 Fab L11	gp41(dis LAI) <b>Donor:</b> P. Perrin and D. Burton (S <b>References:</b> [Binley et al.(1996)] <b>NOTES:</b>	gp41(dis) ı (Scripps Rese 5)]	gp41(dis LAI) gp41(dis) DISCONTINUOUS  Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California  References: [Binley et al.(1996)]  NOTES:	ia N	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$
	• Fab L11: Binds to Cluster III region – competes with MA sensitive – variable regions sequenced [Binley et al.(1996)]	III region – co sequenced [Bi	Fab L11: Binds to Cluster III region – competes with MAb Md-1, but not MAbs sensitive – variable regions sequenced [Binley et al.(1996)]		26-6 and D50 – conformation	ution
749 Fab L1	gp41(dis LAI) gp41(dis) DISCONTINUOUS Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California References: [Binley et al.(1996)] NOTES:	gp41(dis) ı (Scripps Rese. 5)]	DISCONTINUOUS arch Institute, La Jolla, Californ	Z	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$
	• Fab L1: Binds to Cluster III region – competes with MA sensitive – variable regions sequenced [Binley et al.(1996)]	III region – co sequenced [Bi	Fab L1: Binds to Cluster III region – competes with MAb Md-1, but not MAbs sensitive – variable regions sequenced [Binley et al.(1996)]		126-6 and D50 – conformation	ution .
750 Fab G5	gp41(dis LAI) gp41(dis) DISCONTINUOUS  Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California  References: [Binley et al.(1996)]  NOTES:	gp41(dis)  (Scripps Rese  5)]	DISCONTINUOUS arch Institute, La Jolla, Californ	Z	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$
	• Fab G5: Binds to Cluster III region – competes with MA sensitive – variable regions sequenced [Binley et al.(1996)]	III region – co sequenced [Bi	Fab G5: Binds to Cluster III region – competes with MAb Md-1, but not MAbs sensitive – variable regions sequenced [Binley et al.(1996)]		26-6 and D50 – conformation	ntion

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
751 Fab G15	gp41(dis LAI) gp41(dis) DISCONTINUOUS Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California References: [Binley et al.(1996)]	gp41(dis) (Scripps Rese )]	DISCONTINUOUS arch Institute, La Jolla, Califo	rnia N	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$
	• Fab G15: Binds to Cluster III region – competes with MA sensitive – variable regions sequenced [Binley et al.(1996)]	III region – co sequenced [Bi	Fab G15: Binds to Cluster III region – competes with MAb Md-1, but not MAbs 126-6 and D50 – conformation sensitive – variable regions sequenced [Binley et al.(1996)]	t not MAbs 126-0	6 and D50 – conformatic	on
752 Fab A9	gp41(dis LAI) gp41(dis) DISCONTINUOUS Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California References: [Binley et al.(1996)]  NOTES:	gp41(dis) (Scripps Rese )]	DISCONTINUOUS arch Institute, La Jolla, Califo	rnia Z	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} \operatorname{G}_{1\kappa})$
	A9: tive	II region – co sequenced [Bi	Binds to Cluster III region – competes with MAb Md-1, but not MAbs 126-6 and D50 – conformation – variable regions sequenced [Binley et al.(1996)]	not MAbs 126-6	5 and D50 – conformatic	on
753 Fab A12	gp41(dis LAI) gp41(dis) DISCONTINUOUS Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California References: [Binley et al.(1996)]	gp41(dis) (Scripps Rese )]	DISCONTINUOUS arch Institute, La Jolla, Califo	rnia N	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} \operatorname{G}_{1\kappa})$
	• Fab A12: Uncharacterized e	epitope – varia	Fab A12: Uncharacterized epitope – variable regions sequenced [Binley et al.(1996)]	et al.(1996)]		
754 Fab L9	gp41(dis LAI) gp41(dis) DISCONTINUOUS Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California References: [Binley et al.(1996)]	gp41(dis) (Scripps Rese )]	DISCONTINUOUS arch Institute, La Jolla, Califo	rnia Z	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} \operatorname{G}_{1\kappa})$
	<ul> <li>Fab L9: Uncharacterized ep</li> </ul>	itope – variab	Fab L9: Uncharacterized epitope - variable regions sequenced [Binley et al.(1996)]	et al.(1996)]		
755 Fab A2	gp41(dis LAI) gp41(dis) DISCONTINUOUS Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California References: [Binley et al.(1996)]  NOTES:	gp41(dis) (Scripps Rese )]	DISCONTINUOUS arch Institute, La Jolla, Califo	rnia V	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} \operatorname{G}_{1\lambda})$
	• Fab A2: Uncharacterized ep	oitope – variab	Fab A2: Uncharacterized epitope – variable regions sequenced [Binley et al.(1996)]	et al.(1996)]		
756 H2	gp41(dis)  Donor: BioInvent, Lund, Sweden, commercial  References: [Muller et al.(1991)]  NOTES:	gp41(dis) en, commercia )]	DISCONTINUOUS		.9	$\operatorname{human}(\operatorname{IgM}_\kappa)$
	• H2: Anti-idiotypic MAbs (10B3 and 2All) against H2 wer they also react with seropositive sera [Muller et al.(1991)]	0B3 and 2All itive sera [Mul	H2: Anti-idiotypic MAbs (10B3 and 2All) against H2 were generated by immunization of BALB/c mice with H2 – they also react with seropositive sera [Muller et al.(1991)]	y immunization o	of BALB/c mice with H2	

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
757 MO43	gp41(dis)	gp41(dis)	DISCONTINUOUS	Z	in vitro r Env penv9	human(IgM)
	Donor: ? References: [Ohlin et al.(1989)] NOTES: • MO43: Discontinuous epit spanning the transmembrane	89)] epitope involving rane region – this	nor:? erences: [Ohlin et al.(1989)] TES: MO43: Discontinuous epitope involving hydrophobic regions 632-646, 677-681 and 687-691, proximal to and spanning the transmembrane region – this specificity is unusual in HIV-1 positive sera [Ohlin et al.(1989)]	46, <i>677-6</i> 81 and	l 687-691, proximal to a Ohlin et al.(1989)]	nd
758 MO30	gp41(dis)	gp41(dis)	DISCONTINUOUS	Z	in vitro r Env penv9	human(IgM)
	Donor: ? References: [Ohlin et al.(1989)] NOTES:  • MO30: Discontinuous epite spanning the transmembrane	89)] epitope involving rane region – this	nor:?  ferences: [Ohlin et al.(1989)]  TES:  MO30: Discontinuous epitope involving hydrophobic regions 632-646, 677-681 and 687-691, proximal to and spanning the transmembrane region – this specificity is unusual in HIV-1 positive sera [Ohlin et al.(1989)]	46, 677-681 and -1 positive sera [	l 687-691, proximal to a Ohlin et al.(1989)]	nd
759 MO28	gp41(dis)	gp41(dis)	DISCONTINUOUS	Z	in vitro r Env penv9	human(IgM)
	Donor: ? References: [Ohlin et al.(1989)] NOTES: • MO28: Discontinuous epite spanning the transmembrane	89)] epitope involving	nor:? erences: [Ohlin et al.(1989)] TES: MO28: Discontinuous epitope involving hydrophobic regions 632-646, 677-681 and 687-691, proximal to and spanning the transmembrane region – this specificity is unusual in HIV-1 positive sera [Ohlin et al.(1989)]	46, 677-681 and	l 687-691, proximal to a Ohlin et al.(1989)]	nd
760 2A2	gp41(N-term) gp41(n ponor:?  References: [Weissenhorn et al.(1996)] NOTES:	gp41(dis) t al.(1996)]	3	Z	HIV-1 infection	$\operatorname{human}(\operatorname{Ig} G_{1\kappa})$
	<ul> <li>Soluble gp41(21-166) forms a rod like structuone end of the rod [Weissenhorn et al.(1996)]</li> </ul>	orms a rod like st senhorn et al.(19	Soluble gp41(21-166) forms a rod like structure that can be visualized with electron microscopy, and 2A2 binds to one end of the rod [Weissenhorn et al.(1996)]	with electron mi	croscopy, and 2A2 binds	to

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
761 N2-4	gp41 gp41  Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Robinson Jr. et al.(1990a)]  NOTES:  N2-4: No enhancing activity for HIV-1 II  N2-4: NIH AIDS Research and Reference	gp41 (oh-Ichi Matsumoto : et al.(1990a)] activity for HIV-1 II search and Referenc	gp41 ? nor: Evan Hersh and Yoh-Ichi Matsumoto erences: [Robinson Jr. et al.(1990a)] TES: N2-4: No enhancing activity for HIV-1 IIIB [Robinson Jr. et al.(1990a)] N2-4: NIH AIDS Research and Reference Reagent Program: 528	a)] N	HIV-1 infection	$\operatorname{human}(\operatorname{IgG}_{1\kappa})$
762 M25	gp41 gp41 ?  Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [di Marzo Veronese et al.(1985), Watkins et al.(1996)]  NOTES:  • M25: heavy and light chains cloned and sequenced – binding contrast to M77 [Watkins et al.(1996)]	gp41 //Oh-Ichi Matsumoto //ronese et al.(1985) //ronese et al.(1985) //ronese et al.(1996)]	onor: Evan Hersh and Yoh-Ichi Matsumoto eferences: [di Marzo Veronese et al.(1985), Watkins et al.(1996)]  • M25: heavy and light chains cloned and sequenced – binding requires heavy anc contrast to M77 [Watkins et al.(1996)]		purified HTLV-III r light chain in combination, in	$\operatorname{murine}(\operatorname{IgG}_{\kappa})$ 1, in
763 10E9	gp41  Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Papsidero et al.(1988)]  NOTES:  • 10E9: 100/100 HIV+ human sera could i	gp41 /oh-Ichi Matsumotc al.(1988)] human sera could i	gp41 ?  nor: Evan Hersh and Yoh-Ichi Matsumoto erences: [Papsidero et al.(1988)] TES: 10E9: 100/100 HIV+ human sera could inhibit 10E9 binding [Papsidero et al.(1988)]	ero et al.(1988)]	HIV-1 infection	$\operatorname{murine}(\operatorname{Ig} G_1)$
764 31A1	gp41 gp41 ?  Donor: ? References: [Pollock et al.(1989)]  NOTES:  • 31A1: Reacts with both p24 and gp41 [Pollock et al.(1989)]	gp41 l.(1989)] hth p24 and gp41 [P	? ollock et al.(1989)]	Z	in vitro immunization, denatured HIV-1	$\operatorname{human}(\operatorname{IgM}_{\kappa/\lambda})$
765 39A64	gp41 gp41 ?  Donor: ? References: [Pollock et al.(1989)] NOTES:  30864: Peacts with both p24 and gp41 [Pollock et al.(1980)]	gp41 L(1989)]	Dollock et al (1980)	Z	in vitro immunization, denatured HIV-1	$\operatorname{human}(\operatorname{IgM}_{\kappa/\lambda})$

MAb ID 766 39B86	Location gp41	WEAU gp41	Sequence	Neutralizing N		Immunogen  in vitro immunization, denatured HIV-1
	Donor: ? References: [Pollock et al.(1989)] NOTES:  • 39B86: Reacts with both p24 and gp41 [Pollock et al.(1989)]	l.(1989)] oth p24 and gp41	[Pollock et al.(198	9)]	9)]	
767 9303	gp41 gp4 <b>Donor:</b> Du Pont <b>References:</b> [McDougal et al.(1996)]	gp41 st al.(1996)]			Z	Z
768 3H6	gp41  Donor: Du Pont  References: [Pinter et al.(1995)]  NOTES:  • 3H6: There is another MAb  • 3H6: Generated in response	gp41 (1995)] r MAb with this II sponse to virus gro	) that recognizes R	ev [Orsin medium	or: Du Pont erences: [Pinter et al.(1995)] TES: 3H6: There is another MAb with this ID that recognizes Rev [Orsini et al.(1995)] 3H6: Generated in response to virus grown in protein-free medium [Pinter et al.(1995)]	ev [Orsini et al.(1995)] medium [Pinter et al.(1995)]
769 31710B	gp41 gp41  Donor: Du Pont  References: [Alsmadi & Tilley(1998)]  NOTES:  • 31710B: A study of 6 anti-Env MA  MN, SF-2, and RF – bound and di	gp41 Tilley(1998)] anti-Env MAbs an	nd their ability to b	ind or dire	gp41  or: Du Pont  rences: [Alsmadi & Tilley(1998)]  IES:  31710B: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against tar MN, SF-2, and RF – bound and directed lysis against all four strains [Alsmadi & Tilley(	nor: Du Pont èrences: [Alsmadi & Tilley(1998)] TES: 31710B: 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)]