	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
603	4E10	gp160(823–829)	gp41(824–830 BH10)	AEGTDRV	no	HIV-1 infection	human($IgG_3\kappa$)

References: [Buchacher (1992), Buchacher (1994), D'Souza (1994)]

- 4E10: MAbs generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells – also binds to MHC class II proteins – anti-class II Abs are only found in HIV-1 positive people –Buchacher94
- 4E10: Included in a multi-lab study for antibody characterization, binding and neutralization assay comparison –D'Souza94

604	Chim 1	gp160(838–844)	gp120( )	KVVKEIP			humanized chimpanzee(unk)

References: [Pincus & McClure(1993), Pincus (1996)]

- Chim 1: Binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect –Pincus93,Pincus96

605	TH9	Env( )	gp120(CD4BS)		L	?	human($IgG_1\kappa$)

Donor: Michael Fung, Tanox Biosystem, USA

References: [D'Souza (1995), Yang (1998)]

- TH9: Found to neutralize MN, but not JRCSF, two B subtype primary isolates, or a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs–D'Souza95
- TH9: A neutralization assay was developed based on hemi-nested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates –Yang98

606	1202-D	Env(dis)	Env(CD4BS)				human(IgG)

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

References: [Nyambi (1998)]

- 1202-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – CD4-BS Abs tended to bind weakly without clade specificity to virions, but bound well to soluble gp120 – 1202-D did not bind to any B clade viruses, and weakly bound A, C, and G clade isolates – 559/64-D, 558-D and 1202-D had similar reactivities –Nyambi98

607	anti-CD4BS summary	Env(dis)	gp120(CD4BS dis)				( )

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

- Shared components of MAb epitopes and the discontinuous CD4 binding regions included Thr 257, Asp 368, Glu 370, Lys 421 through Trp 427 and Asp 457 –Thali93
- Anti-CD4 binding site antibodies (CD4BS) competitively inhibit CD4 binding to monomeric gp120, and they differ in precise dependence on gp120 residues, but generally require Asp-368 and Glu-370 –Moore96

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
608	2G6	Env(dis)	gp120(CD4BS dis)				( )

Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, or Polymun Scientific Inc., Vienna, Austria

References: [Fouts (1998)]

- 2G6: Binds to JRFL oligomer with an affinity comparable to IgG_1b12, but does not neutralize the virus, so binding of oligomer is not always predictive of neutralization – conclusions of this paper contrast with –Parren98 – authors propose a model where 205-46-9 and 2G6 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts the neutralizing effect –Fouts98

609	588-D	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human($IgG_1\kappa$)

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

References: [Karwowska (1992a), Buchbinder (1992), Moore & Ho(1993), Jeffs (1996), Nyambi (1998)]

- 588-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay –Karwowska92
- 588-D: 4-fold increase in neutralization potency for 588-D when combined 1:1 with human MAb 447-D – Buchbinder92
- 588-D: Weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 –Moore93a
- 588-D: Called 588 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 –Jeffs96
- 588-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – CD4-BS Abs tended to bind weakly without clade specificity to virions, but bound well to soluble gp120 – 588-D did not bind to any B clade viruses, and weakly bound a clade A, C, and G clade isolate – 559/64-D, 558-D and 1202-D reacted had similar reactivities –Nyambi98

610	10/46c	Env(dis)	gp120(CD4BS dis)			rgp120	rat( )

References: [Cordell (1991), Jeffs (1996), Peet (1998)]

- 10/46c: Increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 –Jeffs96
- 10/46c: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – 10/46c was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to WT, and no enhanced immunogenicity of conserved regions –Peet98

611	BM12	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(unk)

References: [Kessler (1995)]

- BM12: Broad cross-clade neutralization of primary isolates – additive effect in combination with MAb 2F5 –Kessler95

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
612	654-D	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgGκ)

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

References: [Karwowska (1993), Laal (1994), Gorny (1994), Stamatatos & Cheng-Mayer(1995), Li (1997), Stamatatos (1997), Gorny (1997), Gorny (1998), Schonning (1998), Nyambi (1998), Hioe (1999)]

- 654-D: Dissociation constant gp120 IIIB 0.008 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D – reported to be human(IgG 1lambda) –Laal94
- 654-D: Mild oxidation of carbohydrate moieties inhibits binding –Gorny94
- 654-D: Binds to HIV-1 SF128A and SF162 –Stamatatos95
- 654-D: Called 654-30D – One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env –Li97
- 654-D: Anti-CD4 BS MAb 654-30D and IgG_1b12 have comparable binding affinities, neither mediates gp120-virion dissociation, but IgG1b12 can neutralize SF128A and SF162 and 654-D cannot – 654-D actually enhances infection by both viruses in primary macrophages –Stamatatos97
- 654-D: Called 654-D100 – 654-D100 and IgG_1b12 neutralized viruses HIV-BRU and a mutated virus that lacks the V3 loop glycan equally effectively – in contrast, sera from guinea pigs immunized with BRU gp120 neutralize viruses more effectively that lack the V3 glycan– Schonning98
- 654-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – CD4-BS Abs tended to bind very weakly without clade specificity to virions, but bound well to soluble gp120 – 654-D bound only to JRFL –Nyambi98
- 654-D: The presence of leukocyte function-associated molecule 1 (LFA-1) promotes virus infectivity and hinders neutralization, and anti-LFA-1 MAbs can enhance the neutralizing effect of anti-HIV V3 MAb 447-52D and anti-HIV CD4BS MAb IgG_1b12 – non-neutralizing anti-HIV CD4BS MAb 654-D did not become neutralizing in the presence of anti-LFA-1 MAbs –Hioe99

613	S1-1	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human($IgG_1\lambda$)

References: [Lake (1992), Moran (1993), Wisnewski (1996)]

- S1-1: Neutralizes IIIB and MN without complement, and neutralizes RF and a clinical isolate with complement – binds to native but not denatured gp120 – inhibits sCD4-gp120 binding –Lake92
- S1-1: Heavy (V HI) and light (V lambdaIII) chain sequenced – no enhancing activity – similar germline sequence to MAb 86, but very different activity –Moran93
- S1-1: S1-1 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
614	559/64-D	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human($IgG_1\kappa$)

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

References: [Karwowska (1992a), McKeating (1992c), Spear (1993), Forthal (1995), Jeffs (1996), Hioe (1997), Nyambi (1998)]

- 559/64-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay –Karwowska92
- 559/64-D: Did not mediate deposition of complement component C3 on HIV infected cells –Spear93
- 559/64-D: Neutralizing activity, no ADCC activity, and no viral enhancing activity –Forthal95
- 559/64-D: Called 559 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 –Jeffs96
- 559/64-D: Used in the development of resting cell neutralization assay –Hioe97
- 559/64-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – CD4BS Abs tended to bind weakly without clade specificity to virions, but bound well to soluble gp120 – 559/64-D did not bind to any B clade viruses, and weakly bound clade A, C, and G isolates – 559/64-D, 558-D and 1202-D had similar reactivities –Nyambi98

615	428	Env(dis)	gp120(CD4BS dis)			HIV-1 infection	human(unk)

References: [Karwowska (1992a), Jeffs (1996)]

- 428: Slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 –Jeffs96

616	558-D	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(unk)

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

References: [McKeating (1992c), Nyambi (1998)]

- 558-D: Blocks gp120-CD4 binding – binds a panel of mutants all except for 256 S/Y and 262 N/T, which are probably conformationally disruptive –McKeating92b
- 558-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – CD4BS Abs tended to bind weakly without clade specificity to virions, but bound well to soluble gp120 – 558-D did not bind to any B clade viruses, and weakly bound to clade A, C, and G isolates – 559/64-D, 558-D and 1202-D had similar reactivities –Nyambi98

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
617	448-D	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human($IgG_1\lambda$)

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

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

- 448-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay –Karwowska92
- 448-D: Called 448D – blocks gp120-CD4 binding – substitutions at gp120 residues 88, 113, 117, 257, 368 and 370 reduce binding – epitope similar to rat MAbs 39.13g and 39.3b –McKeating92b
- 448-D: Did not mediate deposition of complement component C3 on HIV infected cells –Spear93
- 448-D: Dissociation constant gp120 IIIB 0.029 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D –Laal94
- 448-D: Neutralizing activity, positive ADCC activity, and no viral enhancing activity –Forthal95
- 448-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells –Manca95
- 448-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env –Li97
- 448-D: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding –Wyatt98

618	729-D	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human($IgG_1\kappa$)

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

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

- 729-D: Dissociation constant gp120 IIIB 0.025 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D –Laal94
- 729-D: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – reported here to have a λ light chain, but originally reported in –Laal94 to be $IgG_1\kappa$ –D'Souza97
- 729-D: Called 720-30D – one of 14 human MAbs tested for ability to neutralize chimeric SHIV-vpu+, which expressed HIV-1 IIIB env –Li97
- 729-D: Neutralizes TCLA strains, but not primary isolates –Parren97

619	HF1.7	Env(dis)	gp120(CD4BS dis)		L	purified anti-Leu-3a MAb	murine(IgM)

References: [Chanh (1987)]

- HF1.7: An anti-Id antibody, stimulated by anti-CD4 MAb Leu-3a, binds a recombinant gp160, suggesting HF1.7 mimics CD4 –Chanh87

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
620	D60	gp160(dis)	gp120(CD4BS dis)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Richardson Jr (1996), Sugiura (1999)]

- D60: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D60 is part of a group of MAbs labeled B-I, that had limited cross-reactivity – of seven clade B isolates BH8 and B-al were they only two that consistently reacted with B-I MAbs – B-I MAbs fully blocked CD4 binding –Sugiura99

621	50-61A	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgGκ)

References: [Fevrier (1995)]

- 50-61A: Neutralizes lab strains LAI and SF2 – competes with sera from 45 seropositive subjects – binding affinity 2.4×10^{-10} M –Fevrier95

622	48-16	Env(dis)	gp120(CD4BS dis)		no	HIV-1 infection	human(IgGκ)

References: [Fevrier (1995)]

- 48-16: Broadly cross-reactive, reacts outside the CD4 binding site and V3 region – competes with sera from 45 seropositive subjects – binding affinity $2 - 5 \times 10^{-9}$ –Fevrier95

623	L41	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgG$_1\kappa$)

References: [Ditzel (1995)]

- L41: Substitutions at 133 D/R, 256 S/Y, 257 T/R, 368 D/R or D/T, 370 E/Q or E/R, 384 Y/E, and 421 K/L reduce binding – paradoxically, this Fab was retrieved from the library after masking with known anti-CD4BS MAbs – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available –Ditzel95

624	L28	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgG$_1\kappa$)

References: [Ditzel (1995)]

- L28: Substitutions at 257 T/R, 368 D/R, 370 E/R and 370 E/Q, 475 M/S 102 E/L and 463 N/D reduce binding – binding was enhanced by removal of the V3 loop and by substitutions 45 W/S, 298 R/G, 381 E/P, 382 F/L, 420 I/R, 435 Y/H or Y/R – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available –Ditzel95

625	L33	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgG$_1\kappa$)

References: [Ditzel (1995)]

- L33: binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available –Ditzel95

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
626	L42	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human($IgG_1\kappa$)

References: [Ditzel (1995)]

- L42: Substitutions at 257 T/R, 368 D/R, 370 E/R, 266 A/E and 477 D/V reduce binding – binding was significantly enhanced by 381 E/P and 382 F/L – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available –Ditzel95

627	L52	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human($IgG_1\kappa$)

References: [Ditzel (1995)]

- L52: Binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available –Ditzel95

628	GP13	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgG_1)

References: [Schutten (1993), Back (1993), Bagley (1994), Schutten (1995a), Schutten (1995b), Bolmstedt (1996), Wisnewski (1996), Schutten (1996), Schutten (1997)]

- GP13: Neutralized a broad range of HIV-1 strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D), 384(Y/E) –Schutten93
- GP13: Mutations in a neutralization resistant isolate obtained by passage of the IIIB isolate in chimpanzees reduced neutralization, but the escape was not as clear as seen with anti-V3 MAbs –Back93
- GP13: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor –Schutten95
- GP13: Neutralizes T-cell adapted viruses but not the SI strain 16.2, despite high binding affinity –Schutten95a
- GP13: Sera were obtained from guinea pigs vaccinated either with gp160, or with gp160 lacking N-linked glycans at N406, N448, and N463 – these sera could block equally well both the CD4 BS MAb GP13 and the V3 MAb F58/H3 –Bolmstedt96
- GP13: GP13 is V H5 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96
- GP13: IIIB neutralizing MAbs *in vitro* fail to neutralize in a mouse model it *in vivo* –Schutten96
- GP13: Neutralized (50%) an SI-env chimeric virus and enhanced (>5 fold) an NSI-env chimeric virus –Schutten97
- GP13: UK Medical Research council AIDS reagent: ARP3054

629	GP44	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgG_1)

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

- GP44: Exhibited a more restricted pattern of neutralizing activity than GP13 and GP68 – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D) –Schutten93
- GP44: GP44 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
630	L72	Env(dis)	gp120(CD4BS dis)				murine( )

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

References: [Ditzel (1997)]

- L72: Used to bind gp120 to solid phase to select MAbs from a phage selection library –Ditzel97

631	GP68	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgG_1)

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

- GP68: Neutralized a broad range of HIV-1 lab strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 117(K/W), 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q), 384(Y/E), 435(Y/H) –Schutten93
- GP68: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – GP68 required markedly higher concentrations to neutralize the mutant than wild type –Klasse93b
- GP68: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor –Schutten95
- GP68: GP68 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96
- GP68: UK Medical Research Council AIDS reagent: ARP3055

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
632	ICR 39.13g	Env(dis)	gp120(CD4BS dis)		L	rgp120 BH10	rat(IgG_{2b})

Donor: Jackie Cordell and C. Dean

References: [Cordell (1991), McKeating (1992a), McKeating (1992c), McKeating (1993b), Moore & Ho(1993), Thali (1993), Klasse (1993a), McLain & Dimmock(1994), Beretta & Dalgleish(1994), McKeating (1996), Armstrong & Dimmock(1996), Klasse & Sattentau(1996), Peet (1998)]

- ICR 39.13g: Cross-competes with MAbs ICR 39.3b and 15e –Cordell91
- ICR 39.13g: Binds to a conformational epitope involved in CD4 binding – exerts a synergistic effect in combination with V3 directed MAbs –McKeating92a
- ICR 39.13g: Neutralization activity against HXB10, RF, SF-2 and MN strains of HIV-1 –McKeating93a
- ICR 39.13g: Conformational, does not bind denatured gp120 – weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 –Moore93a
- ICR 39.13g: Strongly inhibits CD4 inducible MAb 48d –Thali93
- ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG –McLain94
- ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type –Klasse93b
- ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background –McKeating96b
- ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b –Armstrong96
- ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g –Klasse96
- ICR 39.13g: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – ICR 39.13g was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to WT, and no enhanced immunogenicity of conserved regions –Peet98
- ICR 39.13g: UK Medical Research Council AIDS reagent: ARP390

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
633	ICR 39.3b	Env(dis)	gp120(CD4BS dis)		L	rgp120 BH10	rat(IgG_{2b})

Donor: J. Cordell and C. Dean

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

- ICR 39.3b: also known as 39.3, 39.3b and ICR39.3b
- ICR 39.3b: Cross-competes with MAbs ICR 39.13g and 15e –Cordell91
- ICR 39.3b: Conformational, does not bind to denatured IIIB –Moore93a
- ICR 39.3b: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively –McLain94
- ICR 39.3b: Neutralizes only if the antibody is added prior to the attachment of the virus to the cell, in contrast to 39.13g –Armstrong96
- ICR 39.3b: Called 39.3b – increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 –Jeffs96
- ICR 39.3b: Called 39.3 – summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding –Wyatt98
- ICR 39.3b: UK Medical Research Council AIDS reagent: ARP391

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
634	15e	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human($IgG_1\kappa$)

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

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

- 15e: Broadly neutralizing, binds multiple strains, competes with CD4 for gp120 binding, DTT reduction of env abrogates binding – more potent blocking of gp120-sCD4 binding than MAbs G3-536 and G3-537 –Ho91a
- 15e: Cross-competes with MAbs ICR 39.13g and ICR 39.3b –Cordell91
- 15e: Binds to gp120 of HIV-1 IIIB, but not RF – mediates ADCC – deletion of the V3 loop from gp120 does not alter ADCC activity –Koup91
- 15e: gp120 mutants that affect 15e epitope binding: 113, 257, 368, 370, 421, 427, 475 – four of these coincide with amino acids important for the CD4 binding domain –Ho92
- 15e: Precipitation of Delta 297–329 env glycoprotein, with a deleted V3 loop, is much more efficient that precipitation of wild type –Wyatt92
- 15e: Amino acid substitutions in HXB2 that strongly inhibit binding, similar to –Ho92, some additional, 88, 102, 117, 113, 257, 368, 370, 421, 427, 457, 470, 480 –Thali92a
- 15e: Called N70-1.5e – does not enhance infection of HIV-1 IIIB and MN –Thali92a
- 15e: Conformational, does not bind denatured gp120 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 –Moore93a
- 15e: Binding to Delta V1/2 and Delta V1/2/3 mutant glycoproteins is greater than binding to wildtype gp120 –Wyatt93
- 15e: Called 15E – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – 15E neutralization was not affected by this mutation –Watkins93
- 15e: Heavy chain is V HIV, V2-1 – light chain is V_kappaI, Hum01/012. Compared to 21h and F105 –Bagley94
- 15e: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 21h and 17b) –Thali94
- 15e: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – anti-CD4 MAbs moderately inhibit gp120 binding to GalCer, possibly through steric hindrance – binding of GalCer to gp120 inhibited but did not completely block 15e binding –Cook94
- 15e: Cross-reactive with gp120 proteins from clades B and D, less so with A and C, and not reactive with clade E and F –Moore94b
- 15e: Binds with higher affinity to monomer than to oligomer, moderate association rate –Sattentau95a
- 15e: The V4 and V5 domains are essential for 1.5e binding, in contrast to the V1, V2, and V3 loops –Lee95
- 15e: Called 1.5e – Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background –McKeating96b

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

15e cont.

- 15e: gp120 binding enhanced by anti-V3 MAb 5G11 and anti-V2 MAb G3-136 – binding inhibited by other CD4 binding site MAbs, antibodies that bind to gp120 only when CD4 is bound, and CD4-IgG –Moore96
- 15e: Anti-CD4BS MAbs 15e, 21h, and IgG_1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs –Poignard96b
- 15e: Inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b
- 15e: Neutralizes HIV-1 LAI less potently than V3 specific MAbs –McDougal96
- 15e: 15e is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96
- 15e: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 15e bound monomer, did not bind oligomer or neutralize JRFL –Fouts97
- 15e: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 15e could only achieve 50% neutralization, but could act synergistically with anti-V3 MAb 694/98-D to achieve 90% –Li97
- 15e: Does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-93, are deleted –Wyatt97
- 15e: Called 1.5E – Binds to 7/7 isolates from breakthrough cases from a MN gp120 vaccine trial –Berman97
- 15e: Neutralizes TCLA strains, but not primary isolates –Parren97
- 15e: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding –Wyatt98
- 15e: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98
- 15e: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MAbs 15e, F91 and IgG_1b12 bound better to the deleted protein than to wild type –Binley98
- 15e: Competes with CG-10 binding, a MAb raised against a gp120 CD4 complex, this was probably due to the disruption of CD4-gp120 by 15e –Sullivan98
- 15e: No detectable neutralizing activity among primary isolates with different co-receptor usage – some neutralization of TCLA strains –Trkola98
- 15e: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer –Fouts98
- 15e: Called 1.5e – the HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – 1.5e enhances and does not neutralize YU2 env even at 50 ug/ml – Sullivan98b
- 15e: UK Medical Research Council AIDS reagent: ARP3016

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
635	1125H	Env(dis)	gp120(CD4BS dis)		L (MN)	HIV-1 infection	human($IgG_1\kappa$)

Donor: Shermaine Tilley, Public Health Research Institute, USA

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

- 1125H: Binding to gp120 inhibited by CD4 – epitope is destroyed by reduction, but not by removal of N-linked sugars – potent neutralization of MN, RF, SF-2 and IIIB – neutralization synergy with anti-V3 MAb 4117C –Tilley91a
- 1125H: Amino acid substitutions in HXB2 that strongly inhibit binding: 88, 102, 117, 113, 257, 368, 370, 421, 427, 457, 470, 480 –Thali92a
- 1125H: Binding to soluble gp120 enhanced by the presence of an anti-V3 HuMAb, 41148D –Pinter93a
- 1125H: Precipitation of Delta 297–329 env glycoprotein, with has a deleted V3 loop, is much more efficient that precipitation of wild type –Wyatt92
- 1125H: Neutralization was MN specific – failed to neutralize JRCSF, and 2 B subtype and 1 D subtype primary isolates in a multi-laboratory study involving 11 labs –D'Souza95
- 1125H: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G –Warrier96
- 1125H: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding –Pincus96
- 1125H: Called 1125h – summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding –Wyatt98
- 1125H: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against all four strains –Alsmadi98
- 1125H: A neutralization assay was developed based on hemi-nested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates –Yang98

636	5145A	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgG_1)

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

- 5145A: Potent and broadly cross-reactive neutralization of lab strains –Pinter93
- 5145A: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G –Warrier96
- 5145A: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding –Pincus96
- 5145A: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against all four strains –Alsmadi98

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
637	21h	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human(IgG_1)

Donor: J. Robinson, Tulane University, LA

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

- 21h: Amino acid substitutions in HXB2 that inhibit binding, some shared with CD4 binding inhibition, 88, 113, 257, 368, 370, 421, 470, 480 –Thali92a
- 21h: Binding to Delta V1/2 and Delta V1/2/3 mutant glycoproteins is greater than binding to wildtype gp120 –Wyatt93
- 21h: Conformational, does not bind denatured gp120 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 –Moore93a
- 21h: Has strong cross-reactivity with gp120 monomers from most subtypes, A-F, with the least reactivity to clade E –Moore94b
- 21h: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies –Moore94d
- 21h: Heavy chain is V HIII, VDP-35 – light chain is V_lambdaIIIa, Hum318. Compared to 15e and F105 –Bagley94
- 21h: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 15e and 17b) –Thali94
- 21h: Binds with higher affinity to monomer than to oligomer, moderate association rate –Sattentau95a
- 21h: Anti-CD4 binding site MAb – reciprocal inhibition by anti-C1, -C4 and other anti-CD4 binding site antibodies – enhanced by some anti-V2 MAbs and anti-V3 MAb 5G11 – enhances binding of some anti-V3 and -V2 MAbs –Moore96
- 21h: Anti-CD4BS MAbs 15e, 21h, and IgG_1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs –Poignard96b

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

21h cont.

- 21h: 21h is V H3 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96
- 21h: Called 2.1H – Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background –McKeating96b
- 21h: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 21h bound monomer, did not bind oligomer or neutralize JRFL –Fouts97
- 21h: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 67 μg/ml –Li97
- 21h: Viral binding inhibition by 21h strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) –Ugolini97
- 21h: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding – major deletions in C1 and C5 and deletions of the V1V2 and V3 loops do not diminish binding –Wyatt97
- 21h: Neutralizes TCLA strains, but not primary isolates –Parren97
- 21h: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding –Wyatt98
- 21h: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98
- 21h: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer – conclusions of this paper contrast with – Parren98 –Fouts98
- 21h: UK Medical Research Council AIDS reagent: ARP3017

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
638	F105	Env(dis)	gp120(CD4BS dis)		L	HIV-1 infection	human($IgG_1\kappa$)

Donor: Marshall Posner, Boston MA

References: [Posner (1991), Thali (1991), Thali (1992a), Marasco (1992), Wyatt (1992), Posner (1992b), Posner (1992a), Moore & Ho(1993), Posner (1993), Cavacini (1993a), Cavacini (1993b), Wyatt (1993), Montefiori (1993), Potts (1993), Klasse (1993a), Pincus (1993), Watkins (1993), Bagley (1994), Thali (1994), Cook (1994), Cavacini (1994b), Cavacini (1994a), Earl (1994), Chen (1994a), Turbica (1995), Posner (1995), Cavacini (1995), Sullivan (1995), Khouri (1995), Jagodzinski (1996), Wolfe (1996), McDougal (1996), Wisnewski (1996), Pincus (1996), Litwin (1996), Chen (1996), Parren (1997b), D'Souza (1997), Li (1997), Cao (1997), Wyatt (1997), Wyatt (1998), Cavacini (1998b), Li (1998), Cavacini (1998a), Brand (1998), Sullivan (1998a), Kropelin (1998), Sugiura (1999), Giraud (1999)]

- F105: First description of F105, binds topographically near the CD4-binding site – inhibits binding of free, infectious virions to uninfected HT-H9 cells, but does not react with virus adsorbed to uninfected HT-H9 cells – soluble rCD4 pre-bound to infected cells inhibits F105 binding – F105 inhibits infection of HT-H9 cells in standard neutralization assays with HIV-1 and MN strains –Posner91
- F105: Neutralization escape mutants result from changes in amino acids in four discontinuous regions: C2, 256-262; C3, 386,370
- F105: Amino acid substitutions that impair F105 neutralization inhibit gp120-CD4 interaction –Thali92a
- F105: MAb cDNA sequence – V H4 V71-4 rearranged with a D H D-D fusion product of dlr4 and da4, and with J H5 – V kappa is from the Humvk325 germline gene joined with Jkappa 2 –Marasco92
- F105: Precipitation of Delta 297–329 env glycoprotein, which has a deleted V3 loop, is much more efficient than precipitation of wild type –Wyatt92
- F105: F105 mediates ADCC against SF2 through the CD16+ population of PBMC – does not mediate complement-dependent cytotoxicity –Posner92
- F105: Significant enhancement of F105 binding to RF infected cells preincubated with V3-specific MAbs V3-2 and V3-1 –Posner92a
- F105: Called F-105 – neutralizes IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 –Moore93a
- F105: F105 binds to and neutralizes selected lab strains and 3/9 HIV-1 primary isolates – synergistic enhancement of neutralization by seropositive sera –Posner93
- F105: No neutralization of primary isolates observed (John Moore, pers comm)
- F105: Additive MN or SF2 neutralization when combined with anti-V3 MAbs 447-52D and 257-D –Cavacini93
- F105: Serum from all asymptomatic HIV-1 positive people tested block F105 binding, but only from 27% of symptomatic individuals –Cavacini93a
- F105: Binding to Delta V1/2 and Delta V1/2/3 mutant glycoproteins is 2.4- and 13-fold greater, respectively, than binding to wildtype gp120 –Wyatt93
- F105: Study of synergism between F105 and sera from vaccinated volunteers with V3-loop specific neutralization activity – 2/3 sera demonstrated neutralization synergy, and 3/3 binding/fusion-inhibition synergy –Montefiori93

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

F105 cont.

- F105: Study of synergism of neutralization and binding comparing F105 and sCD4 with the V3 MAbs: 50.1, 59.1, 83.1, and 58.2 – synergy was observed, and the data suggest that binding of one ligand (F105) can increase the binding of the second (e. g. V3 loop MAbs) due to conformational changes –Potts93
- F105: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – required >81 fold higher concentrations to neutralize the mutant than wild type –Klasse93b
- F105: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – F105 was used as a control – infected lab workers and some of the gp160 vaccinees had a MAb response that could inhibit gp120-CD4 binding, at lower titers than the infected lab workers –Pincus93a
- F105: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – F105 neutralization was not affected by this mutation –Watkins93
- F105: Comparison of MAb F105 sequences with those of MAbs 21h and 15e –Bagley94
- F105: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MABs 48d, 21h, 15e and 17b) –Thali94
- F105: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – anti-CD4 MAbs moderately inhibit gp120 binding to GalCer, possibly through steric hindrance – binding of GalCer to gp120 inhibited but did not completely block F105 binding–Cook94
- F105: Administered intravenously to four cynomologus monkeys, plasma pharmacokinetics and biological activity tested –Cavacini94
- F105: Fab fragments show reduced capacity to neutralize IIIB, MN, and RF compared to intact IgG 1, suggesting bivalent interaction may be important in binding and neutralization –Cavacini94a
- F105: Used as a positive control for CD4 BS antibodies in a study of the influence of oligomeric structure of Env in determining the repertoire of the Ab response –Earl94
- F105: A human CD4+ T lymphocyte line was transduced to express Fab fragments of F105 – heavy and light chains are joined by an inter-chain linker – in the transduced cells infected with HIV-1, the Fab binds intracellularly to the envelope protein and inhibits HIV-1 production – secreted Fab fragments neutralize cell-free HIV-1 – combined intra- and extracellular binding activities of the expressed Fab make transduced cells resistant to HIV-1 infection and also can protect surrounding lymphocytes by secreting neutralizing antibodies –Marasco93,Chen94a
- F105: An immunoassay for titrating CD4BS serum antibody was developed using a gp120-coated solid phase and competition with MAb F105 – 109/110 French HIV-1+ sera and 51/56 HIV-1+ African sera had detectable CD4BS Abs using this assay, demonstrating CD4 binding site conservation among diverse subtypes – CD4BS Abs were detected soon after seroconversion and persisted – 0/21 HIV-2+ sera reacted, indicating that the HIV-1 and HIV-2 CD4BS Abs are not cross-reactive –Turbica95
- F105: Eight patient phase Ia trial for use as an immunotherapeutic – no clinical or biochemical side effects observed, plasma levels geq of 10 μg/ml maintained for 21 days –Posner95

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

F105 cont.

- F105: Efficient neutralization of T-cell adapted lines HXBc2 and MN, no neutralization of primary isolates 89.6, ADA and YU2 – even some enhancement of infection of ADA and YU2 was observed –Sullivan95
- F105: Biotinylated F105 was used for competition studies with Ab derived from pregnant HIV-1+ women – a correlation between maternal anti-CD4 BS Abs overlapping the F105 binding site and lack of HIV-1 transmission to infants was noted –Khouri95
- F105: Changing heavy chain from IgG 1 to IgG 3 increased neutralization efficiency –Cavacini95
- F105: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop results in less potent inhibition of F105 binding by CRDS – binding site of F105 described as 256–257 ST, 368–370 DPE, 421 K, and 470–484 PGGGDMRDNWRSELY –Jagodzinski96
- F105: Phase I study – MAb clearance in plasma has a 13 day half-life –Wolfe96
- F105: Neutralizes HIV-1 LAI less potently than V3 specific MAbs –McDougal96
- F105: F105 is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96
- F105: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding –Pincus96
- F105: Binding of F105 to oligomeric gp120 occurs despite the fact it cannot neutralize primary isolates –Litwin96
- F105: Intracellular co-expression of heavy and light chains of the Fab105 fragment MAb F105 was enhanced by inclusion of an internal ribosome entry site (IRES) sequence – the Fab105 IRES expression cassette was cloned into an adeno-associated virus (AAV) shuttle vector, and transduced into human lymphocytes which were able to produce and secrete the Fab105 fragments while maintaining normal growth – several primary HIV-1 patient isolates were effectively blocked –Chen96
- F105: Neutralizes TCLA strains, but not primary isolates –Parren97
- F105: In a multilaboratory blinded study, failed to neutralize any of nine B clade primary isolates –D'Souza97
- F105: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – F105 could only achieve 50% neutralization alone – all Ab combinations tested showed synergistic neutralization – F105 has synergistic response with MAbs 694/98-D (anti-V3), 48d, 2F5, and 2G12, and also with HIVIG –Li97
- F105: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to a CD4BS MAb, F105 or sCD4 –Cao97
- F105: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-93, are deleted –Wyatt97

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

F105 cont.

- F105: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding –Wyatt98
- F105: Phase I dose escalation study, single dose of 100 or 500 mg/m2 was given to 4 HIV+ patients – sustained levels, no immune response against F105, no toxicity, infused Ab retained function – there was no evidence of anti-HIV-1 activity and virus was not diminished at day 1 or 7, by culture or plasma RNA –Cavacini98
- F105: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) –Li98
- F105: The MAb F240 binds to the immunodominant region of gp41 and enhances infection in the presence of complement – reactivity of F240 is enhanced by preincubation of cells with sCD4 or anti-CD4BS MAb F105 –Cavacini98a
- F105: Immunoprecipitation of gp120 and gp160 expressed from a rec Semliki Forest virus by F105 and IgG_1b12 indicated that the SFV expressed HIV-1 Env was folded appropriately – and SVF-HIV-1 Env vaccine gave the strongest anti-HIV-1 Env response in mice, when compared to an HIV-1 Env DNA vaccine and a rgp160 protein –Brand98
- F105: A of comparison of 25 gp120 specific, conformation dependent MAbs was done and F105 was used for competition studies – F105 did cross-compete with multiple CD4BS specific MAbs, however most could not neutralize even the autologous NL4-3 strains –Sugiura99
- F105: F105 enhances viral entry of viruses carrying the YU2 envelope glycoproteins, but neutralizes HXBc2 – Sullivan98b
- F105: Anti-C1 region MAb 87-135/9 blocks gp120 interaction with CD4+ cells – blocking activity is additive when combined with antibodies which bind in the C4 region of gp120 (F105, 388/389, and b12) –Kropelin98
- F105: NIH AIDS Research and Reference Reagent Program: 857

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
639	IgG_1b12	Env(dis)	gp120(CD4BS dis)		L P	HIV-1 infection	human($IgG_1\kappa$)

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

References: [Burton (1991), Barbas III (1992), Roben (1994), Burton (1994), Moore (1994b), Sattentau(1995), Moore (1995a), Moore & Ho(1995), Parren (1995), Trkola (1995), Ditzel (1995), Sullivan (1995), Yang (1997), Moore & Sodroski(1996), Gauduin (1996), Poignard (1996b), Poignard (1996a), Trkola (1996a), Sattentau(1996), McKeating(1996), D'Souza (1997), Schutten (1997), Mo (1997), Fouts (1997), Li (1997), Kessler II (1997), Moore & Trkola(1997), Stamatatos (1997), Valenzuela (1998), Ditzel (1997), Ugolini (1997), Wyatt (1997), Wyatt (1998), Burton & Montefiori(1997), Boots (1997), Parren (1997b), Parren (1997a), Parren & Burton(1997), Mondor (1998), Parren (1998a), Connor (1998), Binley (1998), Fouts (1998), Takefman (1998), Parren (1998b), Brand (1998), Schonning (1998), Sullivan (1998a), Frankel (1998), Kropelin (1998), Poignard (1999), Jackson (1999), Hioe (1999), Montefiori & Evans(1999), Giraud (1999), Beddows (1999), Binley (1999)]

- IgG_1b12: Fab b12 was derived from IgG1b12, Fab 3B3 was derived from Fab b12 by random mutagenesis and selected for increased affinity to sgp120
- IgG_1b12: The original Fab fragment was derived from a combinatorial phage library from bone marrow of an HIV-1 positive individual –Burton91
- IgG_1b12: Anti-CD4 binding site Fab, potent neutralizing activity, greater affinity for a subpopulation of gp120 molecules suggested to be in a mature confirmation – mutations in gp120 that abrogate binding: 368 D/R or D/T, 370 E/R, and 477 D/V, of clone HXBc2 of LAI – sensitive to V1 and V2 substitutions –Roben94
- IgG_1b12: Very potent neutralization, of primary and lab strains, at concentrations that could be achieved by passive immunization – reduced binding with A,C, and D clade viruses relative to B clade, poor reactivity with E clade – isolates that were refractive to neutralization by sera from HIV-1+ donors could be neutralized by IgG1 b12 –Burton94
- IgG_1b12: Cross-reactive with some gp120s, (but not all), from clades A-D – not reactive with gp120 from clades E or F –Moore94b
- IgG_1b12: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity –Sattentau95
- IgG_1b12: Anti-CD4 binding site MAb – very potent neutralization of a number of primary isolates –Moore95b
- IgG_1b12: Complete protection against HIV-1 infection was achieved in hu-PBL-SCID mice by passive immunization with physiologically relevant doses – pharmacokinetics showed serum half-life of 30.2 +/- 1.3 hours for Fab b12 and 7.4 +/- 0.7 days for IgG1 b12 in mice, but IgG1 half-lives in human are generally between 21–23 days – Parren95,Parren97c
- IgG_1b12: Called BM12 – broad cross-clade neutralization of primary isolates – additive neutralization in combination with MAb 2F5 –Kessler95
- IgG_1b12: Review: unusual properties for anti-CD4 BS MAb: sensitive to V2 substitutions, preferential recognition of the oligomer on the cell surface –Moore95c
- IgG_1b12: Could potently neutralize primary isolates from within clade B, but showed a slight reduction in efficacy outside of clade B –Trkola95a

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

IgG_1b12 cont.

- IgG_1b12: Because of Fab b12's reduction in binding when the V2 loop is deleted and when aa 183/184 PI/SG substitutions are made –Roben94, competition studies were done with Fab L78 anti-V2 MAbs SC258 and 684-238
- IgG_1b12: Fab b12 showed potent neutralization of T-cell-line-adapted strains, but much reduced neutralization of 3 primary isolates – 2 of the 3 primary isolates also had reduced binding affinity, but the third was as efficiently immunoprecipitated as HXBc2 –Sullivan95
- IgG_1b12: Saturation mutagenesis of the complementarity-determining region and optimization strategies were used to create very high affinity versions of this Fab – increased affinity was dominated by a slowing of the off rate –Yang95
- IgG_1b12: Potent neutralizing *ex vivo* of virus taken directly from plasma of HIV-1 infected individuals – little correlation between neutralization sensitivity of passaged virus and plasma derived virus – more effective than MAb 19b –Gauduin96
- IgG_1b12: Review: Unique among anti-CD4BS MAbs in terms of being potent against both lab adapted virus and primary isolates – one of three MAbs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates –Poignard96
- IgG_1b12: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs –Poignard96b
- IgG_1b12: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study – Trkola96b
- IgG_1b12: Review: Only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 –Sattentau96
- IgG_1b12: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 mug per ml for 90% viral inhibition – IgG1b12 failed to neutralize only 1/9 primary isolates, although there was some variation between test sites –D'Souza97
- IgG_1b12: Inhibited some SI- and NSI-env chimeric viruses but enhanced one NSI-env chimeric virus 3 fold – Schutten97
- IgG_1b12: JRCSF was cultured in the presence of IgG1b12 until a 100-fold resistance to neutralization was selected – resistance was due to three changes: V2 substitution D182N and C3 substitution P365L conferred resistance, and V2 D164N was also required for a viable virus – IgG1b12 resistant virus remained sensitive to MAbs 2G12 and 2F5 –Mo97
- IgG_1b12: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – IgG1b12 bound monomer, oligomer, and neutralized JRFL –Fouts97
- IgG_1b12: b12 was used in its IgG 1 form – of 14 human MAbs, the most potent neutralizer of SHIV-vpu+, which expressed HIV-1 IIIB env – all Ab combinations tested showed synergistic neutralization – b12 has a synergistic response with MAbs 694/98-D (anti-V3), 2F5, and 2G12 –Li97
- IgG_1b12: 35 primary isolates were tested and all were neutralized by IgG1b12 (including 4, UG270, RW92/026, ZB20, and 301727 which been had reported as not neutralized by IgG1b12 –Trkola95a) – IgG1b12 could neutralize even when added after the virus to the culture – selection for 400-fold increased affinity did not enhance neutralization by antibody – IgG1b12 was more potent with greater breadth than MAb 2F5 –Kessler97

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

IgG_1b12 cont.

- IgG_1b12: Review: MABs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MABs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs' epitopes –Moore97
- IgG_1b12: MAb was slightly more efficient at neutralization than Fab – inhibits viral binding to cells and viral entry – doesn't affect CD4-independent binding to T-cells –Valenzuela97
- IgG_1b12: Viral binding inhibition by IgG1b12 strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) –Ugolini97
- IgG_1b12: Major deletions in C1 and C5 and deletions of the V1V2 and V3 loops do not diminish binding –Wyatt97
- IgG_1b12: This is a review that includes a description of IgG1b12, noting approximately equivalent affinities for sgp120 and unprocessed gp160, and somewhat enhanced affinity for the native oligomer on TCLA viruses – primary viruses have reduced affinity, but still in the useful range for neutralization – there can be complete protection in hu-PBL-SCID mice with Ab even when administered several hours after viral challenge – competes with sCD4, but unlike other CD4BS antibodies, it is sensitive to mutations in V2 –Burton97
- IgG_1b12: In this review, the technique and potential application of Fab expression and selection in phage display libraries, and subsequent production of IgG molecules is discussed – b12 is exceptionally potent at neutralization and can successfully neutralize most B clade primary isolates, and many isolates from other subtypes as well – 3B3 was derived from b12 by selection for higher affinity using the CDR walking strategy – 3B3 has 8-fold enhancement of binding, a linear correlation was found between neutralization and affinity, and 3B3 can neutralize strains b12 cannot –Parren97c
- IgG_1b12: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – IgG1b12 blocks CD4 binding and is the most potent neutralizing Ab – many 15 and 21-mer phage inserts were recognized, but it was not possible to derive a consensus – common features were a W and at least one acidic residue, and one sequence was found multiple times: NWPRWWEEFVDKHSS, and this peptide could compete with gp120 – two short stretches found in the phage peptides might mimic gp120 components of the epitope: positions 382-384, FFY(I), and 423–426 I(FV)I(V)NM –Boots97
- IgG_1b12: Fab b12 is unusual in that it binds to gp140 and monomeric gp120 with similar affinities, and with a higher affinity to the native oligomer – authors propose this antibody may be exceptional because it binds the virus rather than viral debris – IgG1b12 can protect against infection prior to or shortly after challenge of hu-PBL-SCID mice with TCLA strains and primary strains, but the serum concentrations required were higher than for *in vitro* neutralization –Parren97,Parren97a
- IgG_1b12: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding – IgG1b12 is an unusual CD4BS antibody because it is particularly potent as a neutralizing antibody and it is susceptible to changes in the V1-V2 stem loop structure, and so it may disrupt an interaction between CD4 and conserved amino acids on the V1-V2 stem –Wyatt98
- IgG_1b12: Enhances binding of Hx10 to CD4 positive or negative HeLa cells, inhibits binding to CD4+ T-cell line A3.01 – neutralizes HeLa and A3.01 cell Hx10 infection –Mondor98

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

IgG_1b12 cont.

- IgG_1b12: IgG1b12, FAb b12 and 3B3 derived from b12 were all included in this study – the rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope – binding affinity of divalent IgG1b12 is 17-fold greater than monovalent Fab b12 –Parren98
- IgG_1b12: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D –Connor98
- IgG_1b12: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MAbs 15e, F91 and IgG1b12 bound better to the deleted protein than to wild type –Binley98
- IgG_1b12: Binds JRSF oligomer with high affinity, as do 205-46-9 and 2G6, but IgG1b12 is neutralizing, the other two are not – conclusions of this paper contrast with Parren98 – authors propose a model where 205-46-9 and 2G6 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts the neutralizing effect – rank order of CD4BS antibodies oligomer binding is IgG1b12 = 2G6 = 205-46-9 > 205-43-1 = 205-42-15 > 15e = 21h = F91, and the only thing notably distinguishing about neutralizing IgG1b12 is that it depends on residues in V2 –Fouts98
- IgG_1b12: Induces Complement-mediated lysis in MN but not primary isolates – primary isolates are refractive to CML –Takefman98
- IgG_1b12: MAbs 2G12, 2F5 and b12 are broadly neutralizing, as are some human polyconal sera, but this paper describes a set of primary isolates that are resistant to all three MAbs and 2 broadly neutralizing sera – results indicate that resistance levels of pediatric isolates might be higher than adult isolates – resistance in general did not seem to be conferred by a loss of binding affinity for gp120 or gp41, rather by a more global perturbation of oligomeric Envelope –Parren98a
- IgG_1b12: Immunoprecipitation of gp120 and gp160 expressed from a rec Semliki Forest virus by F105 and IgG1b12 indicated that the SFV expressed HIV-1 Env was folded appropriately – and SVF-HIV-1 Env vaccine gave the strongest anti-HIV-1 Env response in mice, when compared to an HIV-1 Env DNA vaccine and a rgp160 protein –Brand98
- IgG_1b12: MABs 654-D100 and IgG1b12 neutralized viruses HIV-BRU and a mutated virus that lacks the V3 loop glycan equally effectively – in contrast, sera from guinea pigs immunized with BRU gp120 neutralize viruses more effectively that lack the V3 glycan– Schonning98
- IgG_1b12: FAb b12 – the HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – FAb fragment b12 also enhances YU2 entry, ruling out Fc interactions or Env cross-linking as a mechanism, while neutralizing HXBc2 –Sullivan98b

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

IgG_1b12 cont.

- IgG_1b12: Prevention of the initial infection of mucosal dendritic cells and disruption of DC to T cell transmission are desirable attributes of anti-HIV-1 vaccine stimulated Abs – IgG1b12 and a combination of 2F5 and 2G12 could neutralize viral entry into DCs – IgG1b12 could block transmission from infected DC to T cells –Frankel98
- IgG_1b12: anti-C1 region MAb 87-135/9 blocks gp120 interaction with CD4+ cells – blocking activity is additive when combined with antibodies which bind in the C4 region of gp120 (F105, 388/389, and b12) –Kropelin98
- IgG_1b12: The presence of leukocyte function-associated molecule 1 (LFA-1) promotes virus infectivity and hinders neutralization, and anti-LFA-1 MAbs can enhance the neutralizing effect of anti-HIV V3 MAb 447-52D and anti-HIV CD4BS MAb IgG1b12 – non-neutralizing anti-HIV CD4BS MAb 654-D did not become neutralizing in the presence of anti-LFA-1 MAbs –Hioe99
- IgG_1b12: rgp120 derived from a R5X4 subtype B virus was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – TCLA strains showed enhanced IgG1b12 neutralization sensitivity relative to PBMC-adapted lines – IgG1b12 was able to bind, with low affinity, to the rgp120 monomer HIV-1 W61D –Beddows99
- IgG_1b12: A meeting summary presented results regarding neutralization – D. Burton and J. Mascola presented results concerning passive immunization and protection of hu-PBL-SCID mice and macaques, respectively, and both found combinations of MAbs that were able to achieve 99% neutralization *in vitro* corresponded to efficacy *in vivo* –Montefiori99
- IgG_1b12: does not inhibit attachment of virus to cells and was used as a control of a study of neutralization by a MAb F58 based micro antibody –Jackson99
- IgG_1b12: Hu-PBL-SCID mice were infected with HIV-1s JRCSF and SF162 to study the effect of NAbs on an established infection – at day 6 post infection, mice were given 50 mg/kg of b12, an amount that would have been protective if given up to 8 hours post-infection, and 100-fold higher than the amount required for 90% neutralization *in vitro* – no significant differences in the initial rate of decrease in viral load or the plateau levels of viral RNA between the b12 treated and control mice were seen – in most of the Ab treated mice escape mutants were observed with varying patterns of mutations – a combination of b12, 2G12 and 2F5 protected 1/3 mice, and an isolate from one of the other two was resistant to neutralization by all three MAbs –Poignard99
- IgG_1b12: The MAbs with the broadest neutralizing activity, IgG1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519; nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes –Binley00
- IgG_1b12: UK Medical Research Council AIDS reagent: ARP3065
- IgG_1b12: NIH AIDS Research and Reference Reagent Program: 2640

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
640	b3	Env(dis)	gp120(CD4BS dis)				Fab, human(unk)

References: [Parren (1997b), Parren (1998a)]

- b3: Neutralizes TCLA strains, but not primary isolates –Parren97
- b3: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98

641	b11	Env(dis)	gp120(CD4BS dis)				Fab, human(unk)

References: [Parren (1998a)]

- b11: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98

642	b6	Env(dis)	gp120(CD4BS dis)		L		Fab, human(unk)

References: [Parren (1997b), Parren (1998a)]

- b6: Neutralizes TCLA strains, but not primary isolates –Parren97
- b6: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98

643	b13	Env(dis)	gp120(CD4BS dis)				Fab, human(unk)

References: [Parren (1995), Parren (1998a)]

- b13: Fab b13 was used as a control in a hu-PBL SCID mouse study – animals were protected from HIV-1 SF2 infection by IgG_1b12, somewhat by Fab b12, but not by b13 –Parren95,Parren97c
- b13: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
644	b14	Env(dis)	gp120(CD4BS dis)				Fab, human(unk)

References: [Parren (1998a)]

- b14: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98

645 F91 — Env(dis) — gp120(CD4BS dis) — ( )

Donor: J. Robinson, University of Connecticut, Storrs

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

- F91: Called F-91 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 –Moore93a
- F91: Has strong cross-reactivity with gp120 monomers from most subtypes, A-F –Moore94b
- F91: Unusual pattern of reciprocal enhancement with several anti-V2 and V3 directed MAbs – reciprocal inhibition of other CD4BS MAbs –Moore96
- F91: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – F91 bound monomer, did not bind oligomer or neutralize JRFL –Fouts97
- F91: Weak inhibition of binding of Hx10 to CD4 positive or negative cells, weakly neutralizing –Mondor98
- F91: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98
- F91: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MAbs 15e, F91 and IgG_1b12 bound better to the deleted protein than to wild type –Binley98
- F91: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer – conclusions of this paper contrast with –Parren98 –Fouts98

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
646	HT6	Env(dis)	gp120(CD4BS dis)		L (weak)	HIV-1 infection	human(unk)

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

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

- HT6: HT5, HT6, and HT7 are also known as 205-43-1 , 205-42-15, and 205-46-9, respectively –Fouts98
- HT6: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN –Moore95b
- HT6: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was not quite as extensively cross-reactive –Moore94b
- HT6: MAbs IgG_1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL –Fouts97
- HT6: HT5 and HT6 bind JRSF oligomer but with low affinity, and are not neutralizing – conclusions of this paper contrast with –Parren98 –Fouts98

647	HT5	Env(dis)	gp120(CD4BS dis)		L (weak)	HIV-1 infection	human(unk)

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

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

- HT5: HT5, HT6, and HT7 are also known as 205-43-1, 205-42-15, and 205-46-9, respectively –Fouts98
- HT5: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN –Moore95b
- HT5: 205-46-9 was cross-reactive across clades A-F, 205-43-1 very cross-reactive but not quite as extensive 205-46-9 –Moore94b
- HT5: MAbs IgG_1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL –Fouts97
- HT5: HT5 and HT6 bind JRSF oligomer but with low affinity, and are not neutralizing – conclusions of this paper contrast with –Parren98 –Fouts98

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
648	HT7	Env(dis)	gp120(CD4BS dis)		L (IIIB)	HIV-1 infection	human(unk)

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

- HT7: HT5, HT6, and HT7 are also known as 205-43-1 , 205-42-15, and 205-46-9, respectively –Fouts98
- HT7: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only neutralizes IIIB well, with sporadic weak neutralization of other isolates –Moore95b
- HT7: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was cross-reactive, but not quite as extensive –Moore94b
- HT7: MAbs IgG_1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL –Fouts97
- HT7: Binds JRSF oligomer with high affinity, at least as high as IgG_1b12, but IgG1b12 is neutralizing, H7 is not – conclusions of this paper contrast with –Parren98 – authors propose a model where H7 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts the neutralizing effect –Fouts98

649	MAG 55	Env(dis)	gp120(CD4BS dis)		L	sCD4-(rHXB2 gp120)-complex	murine( )

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

- MAG 55: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 470 P/L, 475 M/S, 477 D/V – neutralizes MN, IIIB and RF –Kang94
- MAG 55: Called #55 – binding reciprocally inhibited by other anti-CD4 binding site MAbs, and by some C1-C5 MAbs – binding enhanced by anti-V3 MAb 110.5 and anti-V2 MAbs G3-136 and G3-4 – enhances binding of many anti-V3 and -V2 MAbs. –Moore96

650	MAG 72	Env(dis)	gp120(CD4BS dis)		L	sCD4-(rHXB2 gp120)-complex	murine( )

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

- MAG 72: Amino acid substitutions that reduce binding 10 fold: 257 T/R or A or G, 262 N/T, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 477 D/V – neutralizes MN, IIIB and RF –Kang94
- MAG 72: Called L72 – used to bind gp120 to solid phase to select MAbs from a phage selection library –Ditzel97

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
651	MAG 86	Env(dis)	gp120(CD4BS dis)		L	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc
References: [Kang (1994)]
- MAG 86: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 470 P/L, 477 D/V – neutralizes MN, IIIB and RF –Kang94

652	MAG 96	Env(dis)	gp120(CD4BS dis)		L	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc
References: [Kang (1994)]
- MAG 96: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R – weak neutralization of IIIB –Kang94

653	MAG 116	Env(dis)	gp120(CD4BS dis)		L	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc
References: [Kang (1994)]
- MAG 116: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L – neutralizes MN, IIIB and RF –Kang94

654	MAG 3B	Env(dis)	gp120(CD4BS dis)		no	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc
References: [Kang (1994)]
- MAG 3B: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R or A or G, 262 N/T, 368 D/R or T, 370 E/R or Q, 381 E/P, 384 Y/E, 421 K/L, 475 M/S, 477 D/V –Kang94

655	MAG 12B	Env(dis)	gp120(CD4BS dis)		L	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc
References: [Kang (1994)]
- MAG 12B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 477 D/V – weak neutralization of IIIB –Kang94

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
656	830D	Env(dis)	gp120(CD4BS dis)		L		( )

References: [Wyatt (1998)]

- 830D: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding –Wyatt98

657	MAG 29B	Env(dis)	gp120(CD4BS dis)		L	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc

References: [Kang (1994)]

- MAG 29B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 386 N/Q, 421 K/L – weak neutralization of IIIB –Kang94

658	120-1B1	Env(dis)	gp120(CD4BS dis)		L		human(unk)

Donor: Virus Testing Systems Corp., Houston, TX

References: [Watkins (1993)]

- 120-1B1: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – 120-1B1 was not affected by this mutation –Watkins93

659	DO8i	Env(dis)	gp120(CD4BS dis BRU)			HIV-1 infection	Fab, human(unk)

References: [Parren (1998a)]

- DO8i: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98
- DO8i – the HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – FAb fragment DO8i also enhances YU2 entry, ruling out Fc interactions or Env cross-linking as a mechanism, while neutralizing HXBc2 – DO8i was obtained by panning libraries derived from bone marrow from a long term asymptomatic donor against BRU gp120 –Sullivan98b

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
660	DA48	Env(dis)	gp120(CD4BS dis BRU)			HIV-1 infection	human( )

References: [Parren (1998a), Sullivan (1998a)]

- DA48: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98
- DA48: The HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – FAb Ab fragment DA48 also enhances YU2 entry, ruling out Fc interactions or Env cross-linking as a mechanism – while DA48 enhances YU2, it neutralizes HXBc2 – DA48 was obtained by panning libraries derived from bone marrow from a >15 year long term non-progressor against BRU gp120 –Sullivan98b

661	M6	Env(dis)	gp120(CD4BS dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- M6: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – M6 is part of a group of MAbs labeled A1 – all A1 MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, blocked CD4 binding, were sensitive to mutations in gp120 positions 368 and 370 that directly contact CD4 –Sugiura99

662	M12	Env(dis)	gp120(CD4BS dis IIIB)		L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- M12: There is a p15 gag specific MAb also named M12
- M12: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – M12 is part of a group of MAbs labeled A1 – all A1 MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, blocked CD4 binding, were sensitive to mutations in gp120 positions 368 and 370 that directly contact CD4 – 50% neutralization of NL4-3 was achieved with 21 ug/ml of M12 –Sugiura99

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
663	M13	Env(dis)	gp120(CD4BS dis IIIB)		L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- M13: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – M13 is part of a group of MAbs labeled A1 – all A1 MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, blocked CD4 binding, were sensitive to mutations in gp120 positions 368 and 370 that directly contact CD4 – 50% neutralization of NL4-3 was achieved with 35 ug/ml of M13 –Sugiura99

664	D21	Env(dis)	gp120(CD4BS dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D21: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D21 is part of a group of MAbs labeled A1 – all A1 MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, blocked CD4 binding, were sensitive to mutations in gp120 positions 368 and 370 that directly contact CD4 –Sugiura99

665	D25	Env(dis)	gp120(CD4BS dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D25: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D25 is part of a group of MAbs labeled A1 – all A1 MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, blocked CD4 binding, were sensitive to mutations in gp120 positions 368 and 370 that directly contact CD4 –Sugiura99

666	D39	Env(dis)	gp120(CD4BS dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D39: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D39 is part of a group of MAbs labeled A1 – all A1 MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, blocked CD4 binding, were sensitive to mutations in gp120 positions 368 and 370 that directly contact CD4 –Sugiura99

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
667	D33	gp160(dis)	gp120(CD4BS dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D33: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D33 is part of a group of MAbs labeled A1 – all A1 MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, blocked CD4 binding, were sensitive to mutations in gp120 positions 368 and 370 that directly contact CD4 – D33 was unusual for the group of A1 MAbs, because while it blocked CD4 binding completely, but competed with MAbs that did not in a BIAcore assay – both the N- and C-terminal ends of gp120 are involved in D33 binding –Sugiura99

668	D24	gp160(dis)	gp120(CD4BS dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D24: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D24 is part of a group of MAbs labeled B-I, that had limited cross-reactivity – of seven clade B isolates BH8 and B-al were they only two that consistently reacted with B-I MAbs – B-I MAbs fully blocked CD4 binding –Sugiura99

669	D28	gp160(dis)	gp120(CD4BS dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D28: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D28 is part of a group of MAbs labeled B-I, that had limited cross-reactivity – of seven clade B isolates BH8 and B-al were they only two that consistently reacted with B-I MAbs – B-I MAbs fully blocked CD4 binding –Sugiura99

670	D35	gp160(dis)	gp120(CD4BS dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D35: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D35 is part of a group of MAbs labeled B-I, that had limited cross-reactivity – of seven clade B isolates BH8 and B-al were they only two that consistently reacted with B-I MAbs – B-I MAbs fully blocked CD4 binding –Sugiura99

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
671	D42	gp160(dis)	gp120(CD4BS dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D42: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D42 is part of a group of MAbs labeled B-I, that had limited cross-reactivity – of seven clade B isolates BH8 and B-al were they only two that consistently reacted with B-I MAbs – B-I MAbs fully blocked CD4 binding –Sugiura99

672	D52	gp160(dis)	gp120(CD4BS dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D52: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D52 is part of a group of MAbs labeled B-I, that had limited cross-reactivity – of seven clade B isolates BH8 and B-al were they only two that consistently reacted with B-I MAbs – B-I MAbs fully blocked CD4 binding –Sugiura99

673	D53	gp160(dis)	gp120(CD4BS dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- D53: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D53 is part of a group of MAbs labeled B-I, that had limited cross-reactivity – of seven clade B isolates BH8 and B-al were they only two that consistently reacted with B-I MAbs – B-I MAbs fully blocked CD4 binding –Sugiura99

674	T13	Env(dis)	gp120(CD4BS dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- T13: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – T13 is one of three MAbs labeled group Cb, that was type-specific for BH8 – T13 fully blocked CD4 binding, and the deletion of the V3 loop enhanced binding 10-fold –Sugiura99

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
675	T49	Env(dis)	gp120(CD4BS dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- T49: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – T49 is one of three MAbs labeled group Cb, that was type-specific for BH8 – T49 fully blocked CD4 binding, and the deletion of the V3 loop enhanced binding 10-fold –Sugiura99

676	T56	Env(dis)	gp120(CD4BS dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- T56: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – T56 is one of three MAbs labeled group Cb, that was type-specific for BH8 – T56 fully blocked CD4 binding, and the deletion of the V3 loop enhanced binding 10-fold –Sugiura99

677	MTW61D	Env(dis)	gp120(CD4BS dis W61D)		L	HIV-1 infection	human( )

References: [Sullivan (1998a)]

- MTW61D – the HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – FAb fragment MTW61D also enhances YU2 entry, ruling out Fc interactions or Env cross-linking as a mechanism, while neutralizing HXBc2 – MTW61D was obtained by panning libraries derived from bone marrow from a long term asymptomatic donor against gp120 from primary isolate W61D –Sullivan98b

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
678	A32	Env(dis)	gp120(CD4i C1-C4 dis)		no	HIV-1 infection	human(IgG_1)

Donor: J. Robinson, Tulane University, LA

References: [Moore (1994b), Wyatt (1995), Moore & Ho(1995), Moore & Sodroski(1996), Wu (1996), Trkola (1996a), Binley (1997a), Fouts (1997), Burton & Montefiori(1997), Wyatt (1997), Boots (1997), Parren (1997b), Sullivan (1998b), Binley (1998), Binley (1999)]

- A32: Reacted with virtually every gp120 monomer of every clade tested, most conserved gp120 monomer epitope known –Moore94b
- A32: Epitope is better exposed upon CD4 binding to gp120 – binding of A32 enhances binding of 48d and 17b – studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 –Wyatt95
- A32: Review: epitope is distinct from CD4BS MAbs, 48d and 17b, and 2G12 –Moore95c
- A32: Reciprocal inhibition of binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of some anti-V2 and sCD4 inducible MAbs (48d and 17b) – very similar competition pattern to 2/11c, A32 and 211/c are unique among known human and rodent MAbs –Moore96
- A32: Not neutralizing – binds domains that interact with gp41 – MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 and binding of A32 does not block this inhibition –Wu96
- A32: Does not neutralize JR-FL, or any strain strongly – partial inhibition of gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b
- A32: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – A32 bound monomer, did not bind oligomer or neutralize JRFL –Fouts97
- A32: Review –Burton97
- A32: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding –Wyatt97
- A32: Does not neutralize TCLA strains or primary isolates –Parren97
- A32: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – A32 has a unique epitope involving mostly C2 but C1 and C4 contribute – six quite variable phage inserts were recognized, with a consensus of LPWYN – a central Trp was the most conserved element, consistent with W427 being an important residue for binding gp120 –Boots97
- A32: Enhances binding of CD4i MAbs 17b and 48d, and a MAb generated in response to gp120-CD4 complex CG10 –Sullivan98
- A32: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer –Binley98

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

A32 cont.

- A32: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519; nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes –Binley00

679	17b	Env(dis)	gp120(CD4i dis)	L P (weak)	HIV-1 infection	human(unk)

References: [Thali (1993), Moore (1993c), Thali (1994), Beretta & Dalgleish(1994), Wyatt (1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Wu (1996), Trkola (1996a), Binley (1997a), Fouts (1997), Li (1997), Weinberg (1997), Ditzel (1997), Cao (1997), Wyatt (1997), Parren (1997b), Kwong (1998), Wyatt (1998), Moore & Binley(1998), Rizzuto (1998), Sullivan (1998b), Sullivan (1998a), Binley (1998), Binley (1999)]

- 17b: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs
- 17b: Epitope is better exposed upon CD4 binding to gp120 – competes with 15e and 21h, anti-CD4 binding site MAbs – 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 433A/L, 438 P/R and 475 M/S confer decreased sensitivity to neutralization –Thali93
- 17b: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding of 17b –Moore93d
- 17b: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 21h and 15e) –Thali94
- 17b: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 17b in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 – similar effect observed for 48d and A32 –Wyatt95
- 17b: Binds with higher affinity to monomer and oligomer, slow association rate, poor neutralization of lab strain – this is in contrast to 48d, which has very different kinetics –Sattentau95a
- 17b: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) – anti-V3 MAb 5G11 enhances binding, as do C1-C4 discontinuous epitopes A32 and 2/11c – enhances binding of some anti-V2 MAbs –Moore96
- 17b: Binding did not result in significant gp120 dissociation from virion, in contrast to 48d, although the the gp41 epitope of MAb 50–69 was exposed –Poignard96b
- 17b: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of 17b blocks this inhibition –Wu96
- 17b: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b

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

17b cont.

- 17b: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 17b bound monomer, oligomer, and neutralized JRFL in the presence of sCD4, but if sCD4 was not present, 17b only bound monomer –Fouts97
- 17b: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 17b has synergistic response in combination with anti-V3 MAb 694/98-D –Li97
- 17b: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the Can0A V3 peptide, suggesting Can0A V3 is a conformer that mimics the 48d – it does not bind to 17b, distinguishing the epitopes –Weinberg97
- 17b: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to a CD4BS MAb, F105, or sCD4 –Cao97
- 17b: Binds to sgp120 efficiently, but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – partial reexposure if sCD4 was bound – could not bind to HXBc2 gp120 if the 19 C-term amino acids were deleted in conjunction with amino acids 31–93 in C1, but binding was restored in the presence of sCD4 –Wyatt97
- 17b: Neutralizes TCLA strains, but not primary isolates –Parren97
- 17b: 17b FAb was co-crystallized with a gp120 core and CD4, and it's binding site can be directly visualized – 17b binds to the "bridging sheet" of gp120, an antiparallel β sheet region, contacting residues from the C4 region and the V1/V2 stem – the contact area is small for an Ab-antigen interactive surface, and dominated in the Ab by the heavy chain – the center of the binding region has hydrophobic interactions, and the periphery charge interactions, acidic on 17b and basic on gp120 –Kwong98
- 17b: Summary of the implications of the crystal structure of a gp120 core bound to CD4 and 17b, combined with what is known about mutations that reduce NAb binding to gp120 – probable mechanism of neutralization is interference with chemokine receptor binding – mutations in 88N, 117K, 121K, 256S, 257T, N262, Delta V3, E370, E381, F 382, R 419, I 420, K 421, Q 422, I 423, W 427, Y 435, P 438, M 475 of HXBc2 (IIIB) reduce binding – the only variable residues in gp120 that contact 17b are 202T and 434M – the contact points for 17b with the crystallized incomplete gp120 are mostly in the heavy chain of the Ab, and there is a gap between 17b's light chain and the partial gp120 which may be occupied by the V3 loop in a complete gp120 molecule – the authors propose that the V2 and V3 loops may mask the CD4i Ab binding site, and that the V2 loop may be repositioned upon CD4 binding –Wyatt98
- 17b: Moore and Binley provide a commentary on the papers by –Rizzuto98, –Wyatt98 and –Kwong98 – they point out 17b shares binding elements in gp120 with chemokine receptor molecules, and that CD4 needs to bind to gp120 first to make the 17b epitope accessible and it may be stericly blocked in the CD4 bound virus, thus making it a poor NAb for primary isolates –Moore98
- 17b: Site directed mutagenesis of a WU2 protein with the V1-V2 loops deleted revealed key residues for 17b-gp120 interaction and interaction of gp120 and CCR5 – mutations in residues that reduced 17b by geq 70% binding were R/D 419, I/R 420, Q/L 422, Y/S 435, I/S 423, K/D 121 and K/D 421– 17b can neutralize HIV-1 strains that use different chemokine receptors, supporting a common region in gp120 in chemokine-receptor interaction –Rizzuto98

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

17b cont.

- 17b: sCD4 induces 17b binding in primary isolates and TCLA strains – amino acids that reduce the efficiency of binding were determined and found also to compromise syncytia formation and viral entry – V1V2 deletion or sCD4 binding can expose the 17b epitope for both HXBc2 and macrophage tropic YU2 – neutralizing potency of 17b is probably weak due to poor exposure of the epitope – 17b epitope exposure upon sCD4 binding can occur over a wide range of temperatures, consistent with the energy of CD4 binding being sufficient to drive the V1/V2 loop into a new conformation –Sullivan98
- 17b: The HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops, and the presence of V1/V2 increased the enhancement – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – 17b enhances YU2 enhanced viral entry 10-fold, whereas HXBc2 was neutralized – Sullivan98b
- 17b: A panel of MAbs was shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4i MAbs 17b and 48d bound better to the deleted protein than to wild type –Binley98
- 17b: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519; nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes –Binley00

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
680	48d	Env(dis)	gp120(CD4i dis)		L P (weak)	HIV-1 infection	human(IgG$_1\kappa$)

Donor: J. Robinson, University of Connecticut, Storrs

References: [Thali (1993), Moore & Ho(1993), Moore (1993c), Thali (1994), Moore (1994b), D'Souza (1995), Sattentau(1995), Wyatt (1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Trkola (1996a), Binley (1997a), Li (1997), Weinberg (1997), Lee (1997), Ugolini (1997), Wyatt (1997), Parren (1997b), Wyatt (1998), Mondor (1998), Parren (1998a), Sullivan (1998b), Yang (1998), Binley (1998)]

- 48d: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs
- 48d: Epitope is better exposed upon CD4 binding to gp120 – competes with ICR 39.13, 15e and 21h, anti-CD4 binding site MAbs – inhibited by anti-CD4BS MAb ICR 39.13g and linear anti-C4 MAbs G3-42 and G3-508 – 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 421 K/L, 433A/L, 438 P/R and 475 M/S confer decreased sensitivity to neutralization –Thali93
- 48d: Called 4.8d – Neutralizes IIIB – reactive with SF-2 gp120 – does not inhibit HIV-1 sera from binding to IIIB gp120 –Moore93a
- 48d: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding of 17b –Moore93d
- 48d: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 21h, 15e and 17b) –Thali94
- 48d: Poor cross-reactivity with gp120 from most clades –Moore94b
- 48d: Called 4.8D – Found to neutralize MN, but not JRCSF, two B subtype primary isolates, or a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs–D'Souza95
- 48d: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence of sCD4 involves the V1/V2 loops, with more significant involvement of V2 – similar effect observed for 17b and A32 –Wyatt95
- 48d: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity –Sattentau95
- 48d: Binds with similar affinity to monomer and oligomer, moderate association rate, potent neutralization – this is in contrast to 17b, which has very different kinetics –Sattentau95a
- 48d: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) – anti-C1-C4 discontinuous epitope MAbs A32 and 2/11c enhance binding – reciprocal enhanced binding with some anti-V2 MAbs –Moore96
- 48d: Binding resulted in gp120 dissociation from virion, mimicking sCD4, and exposure of the gp41 epitope of MAb 50-69, in contrast to CD4BS MAbs –Poignard96b
- 48d: Neutralizes JR-FL – slightly inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b

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

48d cont.

- 48d: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – all Ab combinations tested showed synergistic neutralization – 48d has synergistic response with MAbs 694/98-D (anti-V3) and F105 –Li97
- 48d: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the Can0A V3 peptide, suggesting Can0A V3 is a conformer that mimics the 48d, (but not 17b), epitope –Weinberg97
- 48d: Prefers CD4-gp120 complex to gp120 alone, but does not enhance fusion, in contrast to MAb CG10, in fact it inhibits syncytium formation –Lee97
- 48d: Viral binding inhibition by 48d was strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) –Ugolini97
- 48d: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding –Wyatt97
- 48d: Neutralizes TCLA strains, but not primary isolates –Parren97
- 48d: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization of 48d is interference with chemokine receptor binding – CD4 binding increases exposure of epitope due to V2 loop movement – 88N, 117K, 121K, 256S, 257T, N262, δ V3, E370, E381, F 382, R 419, I 420, K 421, Q 422, I 423, W 427, Y 435, P 438, M 475 mutations in HXBc2 (IIIB) decrease binding –Wyatt98
- 48d: Inhibits binding of Hx10 to both CD4 positive and CD4 negative HeLa cells –Mondor98
- 48d: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98
- 48d: CD4i MAbs 17b and 48d compete with MAb CG10, and the binding sites may overlap – MAb A32 enhances binding of 17b, 48d and CG10 –Sullivan98
- 48d: A neutralization assay was developed based on hemi-nested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates –Yang98
- 48d: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4i MAbs 17b and 48d bound better to the deleted protein than to wild type –Binley98
- 48d: NIH AIDS Research and Reference Reagent Program: 1756

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
681	522-149	Env(dis)	gp120(C1 dis)		no	Env glycopro	( )

Donor: G. Robey, Abbott Inc.
References: [Moore & Sodroski(1996), Trkola (1996a), Binley (1998)]

- 522-149: Binding is enhanced by C5 antibodies M91 and 1C1 – mutual binding-inhibition with anti-C1 antibody 133/290 – binding is destroyed by a W/L (position 61, LAI) gp120 amino acid substitution – other C1 antibodies enhance binding to gp120 –Moore96
- 522-149: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b
- 522-149: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer –Binley98

682	MAG 45	Env(dis)	gp120(C1 dis)		no	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc
References: [Kang (1994), Moore & Sodroski(1996), Wyatt (1997)]

- MAG 45: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb –Kang94
- MAG 45: Reciprocal binding inhibition with anti-C1-C5 and anti-C1-C4 discontinuous MAbs – binding enhanced by anti-V3 5G11 – inhibits binding of anti-CD4 binding site MAbs –Moore96
- MAG 45: Called #45 – binds to efficiently sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-50, are deleted –Wyatt97

683	MAG 95	Env(dis)	gp120(C1 dis)		no	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc
References: [Kang (1994)]

- MAG 95: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb –Kang94

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
684	MAG 97	Env(dis)	gp120(C1 dis)		no	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc
References: [Kang (1994)]

- MAG 97: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb –Kang94

685	MAG 104	Env(dis)	gp120(C1 dis)		no	sCD4-(rHXB2 gp120)-complex	murine( )

Donor: C. Y. Kang, IDEC Inc
References: [Kang (1994)]

- MAG 104: Only observed amino acid substitution that reduces binding: 88 N/P and 106 E/A – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb –Kang94

686	M90	Env(dis)	gp120(C1 dis)		no	451 Env	(IgG_1)

Donor: Fulvia di Marzo Veronese
References: [di Marzo Veronese (1992), Devico (1995), Moore & Sodroski(1996), Ditzel (1997), Wyatt (1997), Binley (1998), Binley (1999)]

- M90: Reactive only with native gp120, so binds to a discontinuous epitope – reacts with multiple strains –Veronese92
- M90: Reacted with both non-reduced (but not denatured) covalently cross-linked gp120-CD4 complex –Devico95
- M90: Reciprocal inhibition of binding of other anti-C1 MAbs – inhibits CD4 binding site MAbs – enhances binding of V2 MAbs G3-4 and SC258 –Moore96
- M90: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-82, are deleted –Wyatt97
- M90: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer –Binley98
- M90: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519; nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes –Binley00

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
687	212A	Env(dis)	gp120(C1 dis)		no	HIV-1 infection	human(unk)

Donor: J. Robinson, Tulane University, LA

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

- 212A: Mutations that inhibit binding: C1 (45 W/S) and V5 (463 N/D) – and enhance binding: V2 (179/180 LD/DL) and C5 (495 G/K) –Moore94c
- 212A: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs –Moore96
- 212A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 212A bound monomer, did not bind oligomer or neutralize JRFL –Fouts97
- 212A: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids are deleted –Wyatt97
- 212A: Does not neutralize TCLA strains or primary isolates –Parren97
- 212A: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 –Sullivan98
- 212A: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer –Binley98

688	p7	Env(dis)	gp120(C1 dis HXBc2)			HIV infection	human Fab(IgG_1)

References: [Ditzel (1997), Parren (1997b)]

- p7: gp120 immobilized on solid phase by capture with sCD4 was used for selection of Fabs – three novel N-term Fabs were obtained that bind to similar epitopes, p7, p20, and p35 – a C1 W/S substitution at position 45 abolished binding, a Y/D at position 45 reduced binding, and C5 region substitutions 475 M/S and 493 P/K enhanced binding – compete with MAbs M85, M90 and 212A, but not M91 and G3-299 –Ditzel97
- p7: Does not neutralize TCLA strains or primary isolates –Parren97

689	L19	Env(dis)	gp120(C1 dis HXBc2)			HIV infection	human Fab(IgG_1)

References: [Ditzel (1997)]

- L19: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for the selection of Fabs – six N-term Fabs, L19 L34, L35, L52, L59, and L69, were obtained that have a similar epitope to Fab p7 –Ditzel97

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
690	L100	Env(dis)	gp120(C1-C2 dis HXBc2)			HIV infection	human Fab(IgG_1)

References: [Ditzel (1997), Parren (1997b), Parren & Burton(1997)]

- L100: Does not neutralize TCLA strains or primary isolates –Parren97
- L100: gp120 immobilized on solid phase by capture with sCD4 and then masked with Fab p7 allowed selection of a new Fab, L100, with a novel specificity for C1 and C2 – gp120 C1 substitutions 69 W/L and 76 P/Y abolish L100 binding, and C2 substitutions 252 R/W, 256 S/Y, 262 N/T and 267 E/L abolish or strongly inhibit L100 binding – inhibits binding of MAbs M90 and G3-299, but not M85, 212A, and M91 –Ditzel97,Parren97c

691	2/11c	Env(dis)	gp120(C1-C4 dis)		L (weak)	HIV-1 infection	human(unk)

Donor: J. Robinson, Tulane University, LA

References: [Moore & Sodroski(1996), Trkola (1996a), Binley (1997a), Fouts (1997), Li (1997), Wyatt (1997), Binley (1998)]

- 2/11c: Inhibits binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of some anti-V2 and CD4i MAbs (48d and 17b) – similar reactivity pattern to A32, but less cross-reactive and lower affinity – A32 and 211/c are unique among known human and rodent MAbs –Moore96
- 2/11c: Called 211c – does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b
- 2/11c: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – 2/11c bound monomer, did not bind oligomer or neutralize JRFL –Fouts97
- 2/11c: Called 2.11c – One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 67 μg/ml –Li97
- 2/11c: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31-74, are deleted –Wyatt97
- 2/11c: Called 211/c – a panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer –Binley98

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
692	C11	Env(dis)	gp120(C1-C5 dis)		no	HIV-1 infection	human(unk)

Donor: J. Robinson, Tulane University, LA

References: [Robinson (1992), Moore (1994d), Moore & Sodroski(1996), Trkola (1996a), Wu (1996), Binley (1997a), Fouts (1997), Wyatt (1997), Parren (1997b), Sullivan (1998b), Binley (1999)]

- C11: Mutations that inhibit binding: C1 (45 W/S, 88 N/P) – V5 (463 N/D) – and C5 (491 I/F,493 P/K and 495 G/K) and enhance binding: C1 (36 V/L) – V1-V2 (152/153 GE/SM) – andDeltaV1/V2/V3 –Moore94c
- C11: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs –Moore96
- C11: Did not block ability of gp120-sCD4 complexes to inhibit MIP-1α binding – binds to gp41-binding domain –Wu96
- C11: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b
- C11: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – C11 bound monomer, did not bind oligomer or neutralize JRFL –Fouts97
- C11: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – partial reexposure if sCD4 was bound – does not bind to HXBc2 gp120 if the 19 C-term amino acids are deleted –Wyatt97
- C11: Does not neutralize TCLA strains or primary isolates –Parren97
- C11: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 –Sullivan98
- C11: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519; nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes –Binley00

693	L81	Env(dis)	gp120(C1-C5 dis)		no	HIV infection	human(IgG_1)

References: [Ditzel (1997), Parren (1997b)]

- L81: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – L81 binding is abolished by C1 substitution 45 W/S, C5 substitution 491 I/F, and C3 substitution L/A –Ditzel97
- L81: Does not neutralize TCLA strains or primary isolates –Parren97

694	B2C	Env( )	gp120(C3 HIV2ROD)	HYQ(core)	L	Peptide	murine( )

References: [Matsushita (1995)]

- B2C: Viral neutralization was type-specific for HIV-2 ROD –Matsushita95

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
695	2F19C	Env( )	gp120(C3 HIV2ROD)	APGK(core)	no	Peptide	murine( )
	References: [Matsushita (1995)]						
	• 2F19C: Binds in WB, but binds poorly to Env on the cell surface –Matsushita95						
696	1024	Env( )	gp120(C4)				( )
	References: [Berman (1997)]						
	• 1024: Binds to 1/7 isolates from breakthrough cases from a MN gp120 vaccine trial –Berman97						
697	1331A	Env( )	gp120(C5)			HIV-1 infection	human( )
	Donor: Susan Zolla-Pazner (NYU Med. Center)						
	References: [Nyambi (1998)]						
	• 1331A: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-C5 Abs 670-D and 1331A bound to 3/4 B clade viruses (they don't bind to IIIB), and to subtype D MAL –Nyambi98						
698	CRA-6	Env(dis)	gp120(V1V2 dis)		no	?	murine( )
	References: [Shotton (1995)]						
	• CRA-6: Called CRA6 – same competition group as CRA-3 –Shotton95						
699	11/68b	Env(dis)	gp120(V1V2 dis)		L (HXB2)	rBH10 gp120	rat(IgG_1)
	Donor: Shotton and Dean						
	References: [McKeating (1993b), Shotton (1995), Peet (1998)]						
	• 11/68b: Changes at residues 183/184 (PI/SG) within V2, 435 (Y/H) in C4, abrogate binding –McKeating93a						
	• 11/68b: 435 (Y/H) in C4 does not abrogate binding (John Moore, per comm, 1996)						
	• 11/68b: Cross-competes with MAbs 62c, 66c, 66a, and CRA-4 – similar to MAb 62c – HXB2 neutralization escape mutant had a D/N substitution at residue 185 – non-reciprocal inhibition of binding of CRA-3 and CRA-6 –Shotton95						
	• 11/68b: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – 11/68b was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to WT, and no enhanced immunogenicity of conserved regions –Peet98						
	• 11/68b: UK Medical Research Council AIDS reagent: ARP3041						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
700	62c	Env(dis)	gp120(V1V2 dis)		no	rBH10 gp120	rat(IgG_1)

References: [Shotton (1995)]

- 62c: Cross-competes with MAbs 11/68b, 66c, 66a, and CRA-4 – same cross-competition group as MAb 11/68b – non-reciprocal inhibition of binding of CRA-3 and CRA-6 – substitutions 176–177 FY/AT, 179–180 LD/DL, 183–184 PI/SG, and 191–193 YSL/GSS abrogate binding – binds but does not neutralize Hx10 –Shotton95
- 62c: UK Medical Research Council AIDS reagent: ARP3075

701	L15	Env(dis)	gp120(V1V2 dis)		P (weak)	HIV infection	human(IgG_1)

References: [Ditzel (1997), Parren (1997b)]

- L15: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – 2 anti-V2 Fabs were obtained with very similar epitopes, L15 and L17 – deletions in V1 and V2 abolished binding, and rodent anti-V2 MAbs SC258, CRA3, G3-G4,G3-136, BAT-085, and 52–684 all compete with L15 –Ditzel97
- L15: Does not neutralize TCLA strains but neutralizes some primary isolates weakly –Parren97

702	T54	Env(dis)	gp120(V1V2 dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD

References: [Earl (1994), Sugiura (1999)]

- T54: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – T54 is one of two MAbs labeled B-II, that had limited cross-reactivity with seven clade B isolates and did not fully blocked CD4 binding – deletion of V1/V2 loops abrogated binding –Sugiura99

703	1088	Env( )	gp120(V2)				( )

References: [Berman (1997)]

- 1088: Binds weakly to 2/7 isolates from breakthrough cases from a MN gp120 vaccine trial –Berman97

704	1361	Env( )	gp120(V2)			gp120	human( )

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

References: [Nyambi (1998)]

- 1361: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – V2 Abs 697-D, 1361, and 1357 tended to bind weakly with a similar pattern of specificity to virions, but bound well to soluble gp120: weak binding to 1/4 B clade viruses (CA5), and also weak binding to a subtype D virus, MAL –Nyambi98

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
705	1357	Env( )	gp120(V2)				human( )

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Nyambi (1998)]

- 1357: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – V2 Abs 697-D, 1361, and 1357 tended to bind very weakly with a similar pattern of specificity to virions, but bound well to soluble gp120: weak binding only to subtype D MAL –Nyambi98

706 L17 | Env(dis) | gp120(V2 dis) | | | human Fab( )

References: [Ditzel (1997), Parren (1998a)]

- L17: The rank order of FAb binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than FAb binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98

707 684-238 | Env(dis) | gp120(V2 dis) | | L | IIIB gp120 from infected cells | murine( )

Donor: Gerry Robey, Abbott Laboratories
References: [Moore (1993a), Thali (1993), Gorny (1994), Ditzel (1995), Moore & Sodroski(1996), Ditzel (1997)]

- 684-238: Specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – neutralizes BH10 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177FY/AT, 179/180LD/DL, 183/184PI/SG, and 192-194YSL/GSS –Moore93b
- 684-238: Weakly neutralizing, IC 50 = 84 μg/ml –Gorny94
- 684-238: Does not compete with IgG_1b12, reciprocal inhibition with MAbs L39, L40, and L78 –Ditzel95
- 684-238: Limited reciprocal enhancement of binding with anti-V3 and C4 region antibodies – reciprocal inhibition with V2 region antibodies –Moore96

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
708	CRA-3	Env(dis)	gp120(V2 dis)		no	rBH10 gp120	murine(IgG_{2a})

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

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

- CRA-3: Conformational, does not bind well to denatured gp120 –Moore93a
- CRA-3: specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192–194 YSL/GSS – epitope probably involves stem of V1/V2 loop structure –Moore93b
- CRA-3: Many MAbs enhance binding, including some anti-C5, C1, V4, and C4 MAbs – enhances binding of only a small number of anti-V3 loop MAbs –Moore96
- CRA-3: Called CRA3 – Same competition group as CRA6 –Shotton95
- CRA-3: UK Medical Research Council AIDS reagent: ARP324

709	CRA-4	Env(dis)	gp120(V2 dis)		L (HXB2)	rBH10 gp120	murine(IgG_1)

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

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

- CRA-4: Changes at residues 191/192/193 (YSL/GSS) within V2, 435 (Y/H) in C4, abrogate binding – type-specific neutralization –McKeating93a
- CRA-4: Conformational, does not bind well to denatured gp120 –Moore93a
- CRA-4: Specific for BH10 and HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192–194 YSL/GSS –Moore93b
- CRA-4: Cross-competes with MAbs 11/68b, 62c, 66c, 66a – similar to 66c and 66a – non-reciprocal inhibition by MAbs 12b, 60b and CRA-6 –Shotton95
- CRA-4: The only MAbs that enhanced binding were anti-V3 MAb 5G11 and anti-C1 MAb 135/9 binding – reciprocal inhibition of anti-V2 MAbs –Moore96
- CRA-4: UK Medical Research Council AIDS reagent: ARP325

710	66a	Env(dis)	gp120(V2 dis)		L (HXB2)	rBH10 gp120	murine(IgG_1)

References: [Shotton (1995)]

- 66a: Substitutions 176–177 FY/AT, 179–180 LD/DL, 183–184 PI/SG, and 191–193 YSL/GSS abrogate binding – same competition group as CRA4 –Shotton95
- 66a: UK Medical Research Council AIDS reagent: ARP3074

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
711	66c	Env(dis)	gp120(V2 dis)		L (HXB2)	rBH10 gp120	murine(IgG_1)

References: [Shotton (1995)]

- 66c: Substitutions 176–177 FY/AT, 179–180 LD/DL, 183–184 PI/SG, and 191–193 YSL/GSS abrogate binding – same competition group as CRA4 –Shotton95

712	SC258	Env(dis)	gp120(V2 dis)		L	IIIB gp120 from infected cells	murine( )

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

- SC258: Called 52-581-SC258 – binds to BH10, MN, and RF gp120 – neutralizes BH10 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192–194 YSL/GSS –Moore93b
- SC258: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity – 177 Y/H inhibits SC258 neutralization –Yoshiyama94
- SC258: Very poor reactivity with gp120 molecules outside of clade B –Moore94b
- SC258: Does not compete with IgG_1b12 – reciprocal inhibition with MAbs L39, L40, and L78 –Ditzel95
- SC258: Several MAbs binding to various gp120 epitopes enhance binding, but the only MAb that SC258 enhanced binding of was anti-CD4 binding site MAb F91 – reciprocal inhibition with V2 region antibodies –Moore96
- SC258: Does not inhibit gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study – listed as not neutralizing –Trkola96b

713	110-B	Env(dis)	gp120(V2 dis)		no	BRU infected cell lysates	murine( )

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

- 110-B: specific for BH10, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 168 K/L, 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192–194 YSL/GSS –Moore93b

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
714	L39	Env(dis)	gp120(V2-CD4BS dis)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Ditzel (1995)]

- L39: This Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L39 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684–238 – heavy and light chain variable region sequence is available –Ditzel95

715	L40	Env(dis)	gp120(V2-CD4BS dis)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Ditzel (1995)]

- L40: This Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L40 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding only partially affected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684–238 – heavy and light chain variable region sequence is available –Ditzel95

716	L78	Env(dis)	gp120(V2-CD4BS dis)		L	HIV-1 infection	human($IgG_1\kappa$)

References: [Ditzel (1995)]

- L78: Substitutions at V2: (152/153 GE/SM, 183/184 PI/SG, 191/193 YL/GS), 262 N/T, V3 (314 G/W), CD4BS (257 T/R, 368 D/R, 370 E/R) inhibit binding, and some C4 and C5 substitutions enhance binding – this Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – Fab neutralizes MN and LAI – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684–238 – heavy and light chain variable region sequence is available –Ditzel95

717	L25	Env(dis)	gp120(V2-CD4BS dis)		L (weak)	HIV-1 infection	human(IgG_1)

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

- L25: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – a single anti-V2-CD4 BS Fab was obtained with with sensitivity to substitutions in the V2 and CD4 BS regions – rodent anti-V2 MAb SC258 competes with L25 –Ditzel97
- L25: Neutralizes TCLA strains weakly, but not primary isolates –Parren97

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
718	D27	Env(dis)	gp120(V3-CD4BS dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)
	Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD **References:** [Earl (1994), Otteken (1996), Sugiura (1999)] • D27: Pulse label experiments of 4 MAbs (D20, D27, T20, and T22) binding to noncleavable gp160 revealed that these anti-CD4 MAbs bound with a delay, and that the epitope formed with a $t_{1/2}$ of about 10 minutes –Otteken96 • D27: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D27 is one of two MAbs labeled group Ca, that was type-specific for BH8 – D27 fully blocked CD4 binding, and the deletion of the V3 loop abrogated binding –Sugiura99					
719	D56	Env( )	gp120(V3-CD4BS dis IIIB)		L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)
	Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD **References:** [Earl (1994), Sugiura (1999)] • D56: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D56 is one of two MAbs labeled group Ca, that was type-specific for BH8 – D56 fully blocked CD4 binding, and the deletion of the V3 loop abrogated binding – 12.5 ug/ml of D56 was required to achieve 50% neutralization of HIV-1 NL4-3 –Sugiura99					
720	M096/V3	Env(dis)	gp120(V3 309–318 + 329–338)	IQRGPGRAFV + AHCN-ISRAKW		rIIIB Env 286-467	human(IgM)
	References: [Ohlin (1992)] • M096: Generated through *in vitro* "immunization" of uninfected-donor lymphocytes –Ohlin92						
721	MO101/V3,C4	Env(dis)	gp120(V3 314–323 + 494–503)	GRAFVTIGKI + LGVA-PTKAKR		rIIIB Env 286-467	human(IgM)
	References: [Ohlin (1992)] • MO101: generated through *in vitro* "immunization" of uninfected-donor lymphocytes – reacts with peptides from the V3 and C4 regions –Ohlin92						
722	11/75a/21/41	Env(dis)	gp120(V3 dis)				( )
	References: [McKeating (1992a), Peet (1998)] • 11/75a/21/41: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, but anti-V3 MAb 11/75a/21/41 binding was dramatically diminished by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to WT, and no enhanced immunogenicity of conserved regions –Peet98						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
723	41.1	Env(dis)	gp120(V3 dis HXB10)		L (HXB2)	rgp120 BH10	rat(IgG_{2a})

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

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

- 41.1: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 41.1 is not affected – Reitz88,Klasse93b
- 41.1: Called ICR41.1i – Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – neutralization mediated by 3 molecules of IgG per virion – most efficient at neutralization of the three MAbs studied – acts with multi-hit kinetics –McLain94
- 41.1: Called ICR41.1i – IgG2c? – Neutralization was affected if the Ab was added after the virus bound to the host cells at 24 degrees C or below –Armstrong96
- 41.1: Called ICR41.1i – Neutralization occurs by blocking a post-fusion internalization event, in contrast to MAb F58 –Armstrong96a
- 41.1: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 –Jeffs96
- 41.1: Viral binding inhibition by 41.1 was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) –Ugolini97

724	1334-D	Env( )	gp120(V3 HIV451)	TRTSV		HIV-1 infection	human($IgG_1\kappa$)

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

References: [Zolla-Pazner (1999a), Zolla-Pazner (1999b)]

- 1334-D: This MAb was selected on oligomeric gp160 from HIV451 –Zolla-Pazner99
- 1334-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group –Zolla-Pazner99a

725	K24	gp160( )	gp120(V3 IIIB)				murine( )

Donor: Hybridolab, Institute Pasteur

References: [Altmeyer (1999)]

- K24: A Semliki Forest virus (SFV) expression system carrying BX08 env was used to study the conformation of gp120 env – intracytoplasmic gp120 was recognized by the anti-V3 MABs K24 and F5.5, while gp120 at the plasma membrane was detected only by conformation dependent MAbs 2G12, 670-D and 694/98D and not V3 MAbs – expression in rat brain also showed that surface-expressed Env was recognized only by the conformation-dependent antibodies and not by anti-V3 antibodies –Altmeyer99

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
726	F5.5	gp160( )	gp120(V3 IIIB)				murine( )

Donor: Hybridolab, Institute Pasteur

References: [Altmeyer (1999)]

- F5.5: A Semliki Forest virus (SFV) expression system carrying BX08 env was used to study the conformation of gp120 Env – intracytoplasmic gp120 was recognized by the anti-V3 MAbs K24 and F5.5, while gp120 at the plasma membrane was detected only by conformation dependent MAbs 2G12, 670-D and 694/98D and not V3 MAbs – expression in rat brain also showed that surface-expressed Env was recognized only by the conformation-dependent antibodies and not by anti-V3 antibodies –Altmeyer99

727	D47	Env( )	gp120(V3 IIIB)	unk		IIIB vaccinia expressed Env	murine( )

References: [Richardson Jr (1996), Wyatt (1997), Earl (1997), Otteken (1996)]

- D47: Used for capture of oligomeric Env for antigen capture ELISA – binding of this antibody to oligomeric Env IIIB was not blocked by human sera from the US, consistent with a low prevalence of IIIB-like V3 strains –Richardson96
- D47: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding –Wyatt97
- D47: Used for comparison in a study of gp41 antibodies – D47 binds to a greater extent to cell surface expressed Env than any of 38 conformation dependent anti-gp41 MAbs –Earl97
- D47: Pulse label experiments of MAb binding to noncleavable gp160 revealed that this anti-V3 MAb bound immediately and binding stayed constant through chase period –Otteken96

728	5G11	Env( )	gp120(V3 loop)			?	( )

Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA

References: [Moore & Sodroski(1996)]

- 5G11: Binds to conformation sensitive epitope in the V3 loop – reciprocal inhibition of other V3 loop MAbs – reciprocal enhancement of some C1-C5 MAbs (unusual for an anti-V3 MAb) and CD4 binding site MAbs – and enhances binding of V2 MAbs –Moore96

729	110.J	Env( )	gp120(V3 loop)			?	( )

Donor: F. Traincard, Pasteur Institute, France

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

- 110.J: Inhibits sCD4-inducible anti-CD4 binding site MAb 48d –Thali93
- 110.J: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs –Moore96

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
730	G3-1472	Env( )	gp120(V3 loop)			?	( )

Donor: M. Fung
References: [Moore & Sodroski(1996)]

- G3-1472: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs – binding inhibited by anti-C4 MAbs –Moore96

731 1108 Env( ) Env(V3 mimotope) HIV-1 infection human(IgG$_1\lambda$)

References: [Zolla-Pazner (1999a), Zolla-Pazner (1999b)]

- 1108: Selected with peptide 987, a mimotope of anti-V3 MAb 447-D – MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group –Zolla-Pazner99a
- 1108: The sequence of peptide 987, used to select MAb 1108, is ADGAWRSVHLGPGRGSGSGMGK –Zolla-Pazner99

732 TH1 Env( ) gp120(V3) unk L (MN,JRCSF) human(IgG$_1\lambda$)

Donor: Michael Fung, Tanox Biosystem, USA
References: [D'Souza (1995), Yang (1998)]

- TH1: Found to neutralize MN and JRCSF, but not two B subtype primary isolates, nor a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs –D'Souza95
- TH1: A neutralization assay was developed based on hemi-nested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates –Yang98

733 AG1121 Env( ) gp120(V3) L ? ( )

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

- AG1121: Recognizes monomeric gp120 from T-cell adapted line HXBc2 and primary isolate 89.6 equally well, but 89.6 was three-fold less sensitive to neutralization by AG1121 than HXBc2 –Sullivan95
- AG1121: Called 1121 – Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 –Cao97

734 9305 Env( ) gp120(V3) L murine( )

Donor: Du Pont, Wilmington DE
References: [McDougal (1996)]

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
735	55/1l	gp160( )	gp120(V3)				( )
	References: [Peet (1998)]						
	• 55/1l: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 55/1l binding was abrogated by V3 serine substitutions in the V3 loop – mice injected with serine substituted gp120 had a reduced response relative to WT, and no enhanced immunogenicity of conserved regions –Peet98						
736	55/45a/1l	gp160(dis)	gp120(V3)				( )
	References: [Peet (1998)]						
	• 55/45a/11: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 55/45a/11 binding was only marginally diminished by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to WT, and no enhanced immunogenicity of conserved regions –Peet98						
737	55/68b	gp160( )	gp120(V3)				( )
	References: [Peet (1998)]						
	• 55/68b: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 55/68b binding was abrogated by V3 serine substitutions in the V3 loop – mice injected with serine substituted gp120 had a reduced response relative to WT, and no enhanced immunogenicity of conserved regions –Peet98						
738	MO101/ V3,C4	Env(dis)	gp120(V3+C5 314–323 + 494–503)	GRAFVTIGKI + LGVA-PTKAKR		pB1 (IIIB Env 286-467)	human(IgM)
	References: [Ohlin (1992)]						
	• MO101: generated through *in vitro* "immunization" of uninfected-donor lymphocytes: reacts with peptides from the V3 and C4 regions –Ohlin92						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
739	2G12	Env(dis)	gp120(V3V4 dis)		L P	HIV-1 infection	human($IgG_1\kappa$)

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

References: [Buchacher (1994), Trkola (1995), Moore & Ho(1995), McKeating (1996), McKeating(1996), Trkola (1996b), Moore & Sodroski(1996), Poignard (1996b), Trkola (1996a), Sattentau(1996), D'Souza (1997), Mo (1997), Binley (1997a), Fouts (1997), Li (1997), Moore & Trkola(1997), Mascola (1997), Ugolini (1997), Burton & Montefiori(1997), Parren (1997b), Andrus (1998), Wyatt (1998), Mondor (1998), Parren (1998a), Sullivan (1998b), Connor (1998), Binley (1998), Trkola (1998), Fouts (1998), Takefman (1998), Parren (1998b), Li (1998), Wyatt & Sodroski(1998), Frankel (1998), Kunert (1998), Schonning (1998), Montefiori & Evans(1999), Beddows (1999), Altmeyer (1999), Poignard (1999), Binley (1999)]

- 2G12: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells –Buchacher94
- 2G12: Highly potent Cross-clade neutralizing activity –Trkola95a
- 2G12: Conformationally sensitive epitope destroyed by mutations altering the N-linked glycosylation sites near the base of the V3 loop and the amino-terminal flank of the V4 loop –Trkola96
- 2G12: Binding weakly enhanced by some anti-C1, -C4, -V3, and CD4 binding site MAbs – unusual in that 2G12 binding neither enhanced or inhibited the binding of other MAbs included in the study –Moore96
- 2G12: Review: binding site is distinct from CD4BS MAbs epitope and is unique among known gp120 MAbs, human or rodent –Moore95c
- 2G12: Review: exceptional capacity to neutralize primary isolates in terms of both breadth and potency – one of three MAbs (IgG_1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates –Poignard96
- 2G12: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b
- 2G12: Neutralizes primary isolates, HXB2, and chimeric virus with gp120 from primary isolates in an HXB2 background –McKeating96b
- 2G12: Review: Only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 –Sattentau96
- 2G12: In a multilab evaluation of monoclonal antibodies, only IgG_1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 mug per ml for 90% viral inhibition – neutralized 6 of 9 primary isolates –D'Souza97
- 2G12: A JRCSF variant that was selected for IgG_1b12 resistance remained sensitive to MAbs 2G12 and 2F5, for combination therapy –Mo97
- 2G12: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 2G12 bound monomer, and weakly bound oligomer and neutralized JRFL –Fouts97
- 2G12: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 2G12 was a strong neutralizer of SHIV-vpu+ – all Ab combinations tested showed synergistic neutralization – 2G12 has synergistic response with MAbs 694/98-D (anti-V3), 2F5, F105, and b12 –Li97

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

2G12 cont.

- 2G12: Review: MABs 2F5, 2G12 and IgG_1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MAbs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs' epitopes –Moore97
- 2G12: Using concentrations of Abs achievable *in vivo*, the triple combination of 2F5, 2G12 and HIVIG was found to be synergistic to have the greatest breadth and magnitude of response against 15 clade B primary isolates –Mascola97
- 2G12: Viral binding inhibition by 2G12 was strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) –Ugolini97
- 2G12: Review that discusses this MAb – reacts with residues at the base of the V3 loop and V4, and most of the changes that reduce binding are glycosylation sites – it is not clear whether the binding site is peptidic or direct carbohydrate –Burton97
- 2G12: Neutralizes TCLA strains and primary isolates –Parren97
- 2G12: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIV-1 LAI in hu-PBL-SCID mice, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection –Andrus98
- 2G12: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope –Parren98
- 2G12: Summary of the implications of the crystal structure of gp120 combined with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by 2G12 is unknown, but dependent on proper glycosylation and 2G12 is predicted to be oriented towards the target cell when bound, so neutralization may be due to steric hindrance – mutations in positions N 295, T 297, S 334, N 386, N 392 and N 397 HXBc2 (IIIB) decrease 2G12 binding, and the binding region is 25 angstroms from the CD4 binding site – probably the Ab binds in part to carbohydrates, which may account for both its broad reactivity and the scarcity of Abs in the same competition group –Wyatt98
- 2G12: Enhances Hx10 binding to CD4 positive or negative HeLa cells, but inhibited binding to CD4+ T-cell line A3.01 – neutralizes Hx10 infection of the HeLa cells –Mondor98
- 2G12: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG_1b12, 2F5 and 447-52D –Connor98
- 2G12: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 –Sullivan98
- 2G12: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Delta V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – MAb 2G12 was the only exception to this, showing reduced binding efficiency –Binley98
- 2G12: A wide range of neutralizing titers was observed that was independent of co-receptor usage –Trkola98

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

2G12 cont.

- 2G12: Notes that 2G12 and 2F5, potent neutralizing antibodies, were identified by screening for cell surface (oligomeric envelope) reactivity –Fouts98
- 2G12: Induces Complement-mediated lysis in MN but not primary isolates – primary isolates are refractive to CML –Takefman98
- 2G12: MAbs 2G12, 2F5 and b12 are broadly neutralizing, as are some human polyconal sera, but this paper describes a set of primary isolates that are resistant to all three MAbs and 2 broadly neutralizing sera – results indicate that resistance levels of pediatric isolates might be higher than adult isolates – resistance in general did not seem to be conferred by a loss of binding affinity for gp120 or gp41, rather by a more global perturbation of oligomeric Envelope –Parren98a
- 2G12: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) –Li98
- 2G12: Discussed in a review of the antigenic and receptor binding-domains of gp120 in relation to the structure of the molecule – antibodies are discussed by category (anti-V2, anti-V3, CD4i, CD4BS...), however as 2G12 binds to a rarely immunogenic region, and it is dependent on glycosylation, it was discussed individually –Wyatt98a
- 2G12: The complete V, J and D(H) domain was sequenced – unlike non-neutralizing anti-gp41 MAb 3D6, five neutralizing MAbs (2F5, 2G12, 1B1, 1F7, and 3D5) showed extensive somatic mutations giving evidence of persistent antigenic pressure over long periods – 2G12 D(H) has the best homology to a D(H) segment between D3-22 and D4-23, a region not usually considered for heavy-chain rearrangement because it lacks associated recombination signals in the flanking regions, Kunert *et al*. suggest this may be why Abs that compete with 2G12 are rare –Kunert98
- 2G12: In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, 2G12 was found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan and has a mutation at the tip of the loop more efficiently than it neutralizes HIV-BRU – Schonning98
- 2G12: Prevention of the initial infection of mucosal dendritic cells is a desirable attributes of anti-HIV-1 vaccine stimulated Abs – IgG_1b12 and a combination of 2F5 and 2G12 could neutralize viral entry into DCs –Frankel98
- 2G12: A meeting summary presented results regarding neutralization –MAbs 2G12 and 2F5 tested for their ability to neutralize primary isolate infection of genetically engineered cell lines (cMAGI and others, presented by T. Matthews, A. Trkola, J. Bradac) – an advantage of such cells lines over PBMCs is that markers (X-Gal) can be added for staining to simplify the assay – the consensus of the meeting was that these engineered cell lines did not improve the sensitivity of detection of primary isolate neutralization – D. Burton and J. Mascola presented results concerning passive immunization and protection of hu-PBL-SCID mice and macaques, respectively, and both found combinations of MAbs that were able to achieve 99% neutralization *in vitro* corresponded to efficacy *in vivo* –Montefiori99
- 2G12: rgp120 derived from a R5X4 subtype B virus was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – 2G12 was able to bind with low affinity to the rgp120 monomer HIV-1 W61D –Beddows99

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

2G12 cont.

- 2G12: A Semliki Forest virus (SFV) expression system carrying BX08 env was used to study the conformation of gp120 env – intracytoplasmic gp120 was recognized by the anti-V3 MAbs K24 and F5.5, while gp120 at the plasma membrane was detected only by conformation dependent MAbs 2G12, 670-D and 694/98D and not V3 MAbs – expression in rat brain also showed that surface expressed env was recognized only by the conformation-dependent antibodies and not by anti-V3 antibodies –Altmeyer99
- 2G12: Hu-PBL-SCID mice were infected with HIV-1s JRCSF and SF162 to study the effect of NAbs on an established infection – no significant differences in the initial rate of decrease in viral load or the plateau levels of viral RNA between the b12 treated and control mice were seen – in most of the Ab treated mice b12 escape mutants were observed with varying patterns of mutations – a combination of b12, 2G12 and 2F5 protected 1/3 mice, and an isolate from one of the other two was resistant to neutralization by all three MAbs –Poignard99
- 2G12: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519; nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes –Binley00
- 2G12: UK Medical Research council AIDS reagent: ARP3030
- 2G12: NIH AIDS Research and Reference Reagent Program: 1476

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
740	D7324	Env( )	gp120(C-term)			Peptide from the C-term	sheep( )

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

- D7324: Binding unaltered by gp120 binding to sCD4, in contrast to 110.5, 9284, 50–69 and 98–6 –Sattentau91
- D7324: Binds to the last 15 amino acids in gp120 – used for antigen capture ELISA –Wyatt95
- D7324: Epitope in C5 – Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b
- D7324: Used to capture gp120 onto solid phase for epitope mapping –Moore93b,Moore93c,Ditzel97,Binley98

741	23A	Env(dis)	gp120(C-term)		no	?	( )

Donor: J. Robinson, Tulane University, LA
References: [Thali (1992a), Thali (1993), Wu (1996), Trkola (1996a), Fouts (1997), Binley (1999)]

- 23A: Called 2.3A – Did not block ability of gp120-sCD4 complexes to inhibit MIP-1α binding – binds to gp41-binding domain –Wu96
- 23A: C5 binding MAb – does not inhibit gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study –Trkola96b
- 23A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – 23A bound monomer, did not bind oligomer or neutralize JRFL –Fouts97
- 23A: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519; nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes –Binley00

742	120-1	gp160( )	gp120(C-term 503-532)		no	Peptide	murine(IgMκ)

References: [Chanh (1986), Dalgleish (1988)]

743	C31	Env( )	gp120(gp120)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Boyer (1991)]

- C31: Broadly-reactive group specific MAb – high yield cultivation of human MAb –Boyer91

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
744	P5-3	Env( )	gp120(gp120)			HIV-1 infection	human($IgG_1\lambda$)
	Donor: Evan Hersh and Yoh-Ichi Matsumoto **References:** [Robinson (1990b), Pincus (1991)] • P5-3: No enhancing activity for HIV-1 IIIB –Robinson90a • P5-3: Poor immunotoxin activity when coupled to RAC – isotype specified as: IgG 3λ –Pincus91 • P5-3: NIH AIDS Research and Reference Reagent Program: 378						
745	BAT401	Env( )	gp120(gp120)		L	Inact IIIB	murine(IgG_1)
	References: [Fung (1987)]						
746	BAT267	Env( )	gp120(gp120)		L	Inact IIIB	murine(IgG_1)
	References: [Fung (1987)]						
747	BAT509	Env( )	gp120(gp120)		L	Inact IIIB	murine(IgG_1)
	References: [Fung (1987)]						
748	13.10	Env( )	gp120(gp120)		no	HIV-1 infection	human($IgG_1\lambda$)
	Donor: Evan Hersh and Yoh-Ichi Matsumoto **References:** [Lake (1989), Moran (1993), Wisnewski (1996)] • 13.10: First HIV-1 specific human-mouse hybridoma that produces a MAb that binds to gp120 and gp160 –Lake89 • 13.10: Heavy (V HI) and light (V lambdaII) chain sequenced – no enhancing or neutralizing activity – called No. 13 –Moran93 • 13.10: 13.10 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96 • 13.10: NIH AIDS Research and Reference Reagent Program: 377						
749	F285	Env( )	Env(gp120)			HIV-1 infection	human(IgG_1)
	References: [Wisnewski (1995), Wisnewski (1996)] • F285: F285 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96						
750	multiple Fabs	Env( )	gp120(gp120)			HIV-1 infection	human(unk)
	References: [Burton (1991)] • A panel of anti-gp120 Fabs was generated by antigen selection from a random combinatorial library prepared from bone marrow from an asymptomatic individual –Burton91						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
751	multiple MAbs	Env( )	gp120(gp120)			gp120 complexed with MAb M77	murine( )

References: [Denisova (1996)]

- When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes, as well as an array of MAbs to discontinuous epitope – 10 of 36 MAbs were mapped to linear epitopes and are mentioned elsewhere in this database, the others are: GV5H1, GV4D5, GV4G10, GV1A8, GV10H5, GV8E11, GV2H4, GV6E6, GV1F7, GV1G9, GV4G5, GV6B12, GV1E8, GV2B7, GV1B11, GV6H5, GV6G2, GV6B5, GV1E10, GV5E3, GV5B9, GV5F4, GV6G4, GV1A12, GV5C11, GV6B6, GV3C10 –Denisova96

752	human sera	Env( )	gp120(gp120)			HIV-1 infection	human(IgG)

References: [Binley (1997b)]

- Retention of anti-Env antibodies and loss of anti-Gag antibodies during progression was studied, and suggested to be the result of the loss of T-cell help and the unique ability of Env to stimulate B cells even in a backdrop of declining CD4 cells, because of the ability of Env to bind to the CD4 molecule –Binley97a

753	polyclonal	Env( )	gp120(gp120)		L	HIV-1 Pr55gag VLP with anchored gp120 or V3+CD4 linear domains	Macaca mulatta( )

References: [Wagner (1998)]

- A VLP is a non-infectious virus-like particle self-assembled from HIV Pr55 gag – macaques were immunized with VLPs bound to either gp120 or V3+CD4 linear domains – gag and env CTL specific CTL were stimulated in each case, and Ab response to gag and gp120 and was elicited, but the gp120 neutralizing response occurred only with whole gp120, not V3+CD4 – despite the CTL and Ab response, immunized macaques were infected by intervenous challenge with SHIV chimeric challenge stock –Wagner98

754	polyclonal	Env( )	gp120(gp120)		L	DNA gag/pol, vif, and CMN160 vaccine	murine( )

References: [Kim (1997)]

- A gag/pol, vif or CMN160 DNA vaccine, when delivered in conjunction with the plasmid encoding the co-stimulatory molecules B7 and IL-12, gave a dramatic increase in both the cytotoxic and proliferative responses in mice
- The Ab response was detected by ELISA, but the CMN160 DNA vaccinated mice showed a neutralizing Ab response

755	polyclonal	Env( )	gp120(gp120)		P	HIV-1 infection	human( )

References: [Bradney (1999)]

- Sera were taken from long term non-progressors and evidence for viral escape was noted – serum could neutralize earlier autologous isolates, but not contemporary isolates –Bradney99

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
756	polyclonal	Env( )	gp120(gp120)		L P	HIV-1 gag/env in canary pox, boost with SF-2 rgp120	human( )

References: [Belshe (1998)]

- NAbs were obtained by a HIV-1 gag/env in canary pox vaccination of eight volunteers after boosting with rgp120 against lab strains – 1/8 primary isolates was neutralized, BZ167–Belshe98

757	HBW4	Env( )	gp120(gp120 IIIB)			HIV-1 infection	human($IgG_1\lambda$)

References: [Moran (1993), Wisnewski (1995), Wisnewski (1996)]

- HBW4: Heavy (V HII) and light (V lambdaII) chain sequenced –Moran93
- HBW4: HBW4 is V H2 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals –Wisnewski96

758	polyclonal	Env( )	gp120(gp120 IIIB)			gp120 or gp160 DNA vaccine	murine( )

References: [Shiver (1997)]

- DNA vaccinations of BALBc mice with a gp120 or gp160 DNA vaccine elicited a strong T cell proliferative response with Th1-like secretion of γ interferon and IL-2, with little or no IL-4, as well as antigen specific gp120 Abs –Shiver97

759	T20	gp160(dis)	gp120(gp120 dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Otteken (1996), Sugiura (1999)]

- T20: Pulse label experiments of 4 MAbs (D20, D27, T20, and T22) binding to noncleavable gp160 revealed that these anti-CD4BS MAbs bound with a delay, and that the epitope formed with a $t_{1/2}$ of about 10 minutes –Otteken96
- T20: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – T20 is part of a group of MAbs labeled AII – all AII MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, and could only partially blocked CD4 binding –Sugiura99

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
760	T22	gp160(dis)	gp120(gp120 dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Otteken (1996), Sugiura (1999)]

- T22: Pulse label experiments of 4 MAbs (D20, D27, T20, and T22) binding to noncleavable gp160 revealed that these anti-CD4BS MAbs bound with a delay, and that the epitope formed with a $t_{1/2}$ of about 10 minutes –Otteken96
- T22: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – T22 is part of a group of MAbs labeled AII – all AII MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, and could only partially blocked CD4 binding –Sugiura99

761	T27	gp160(dis)	gp120(gp120 dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- T27: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – T27 is part of a group of MAbs labeled AII – all AII MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, and could only partially blocked CD4 binding –Sugiura99

762	T52	Env(dis)	gp120(gp120 dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Sugiura (1999)]

- T52: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – T52 is one of two MAbs labeled B-II, that had limited cross-reactivity with seven clade B isolates and did not fully blocked CD4 binding – deletion of V1/V2 loops abrogated binding –Sugiura99

763	polyclonal	Env( )	gp120(gp120 W61D)		L	rgp120 HIV-1 W61D	human( )

References: [Beddows (1999)]

- rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with HIV-1 positive subjects – vaccinee sera had more potent responses to linear V1/V2 and V3 epitopes than did the sera from HIV-1+ individuals, but could only neutralize homologous or heterologous virus only after adaptation to T-cell lines – neutralization activity was lost after re-adaptation to growth in PBMCs – in contrast, sera from infected individuals could neutralize both PBMC and T-cell line adapted viruses –Beddows99

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
764	ID6	Env( )	gp120(gp120 N-term 1–193 BH10)	UNDEFINED AMINO TERMINUS		?	murine(IgG_1)

References: [Ugen (1993), Cook (1994)]

- ID6: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding –Cook94
- ID6: NIH AIDS Research and Reference Reagent Program: 2343

765	AD3	Env( )	gp120(gp120 N-term 1–193 BH10)	UNDEFINED AMINO TERMINUS		?	murine(IgG_1)

References: [Ugen (1993), Cook (1994)]

- AD3: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding –Cook94
- AD3: NIH AIDS Research and Reference Reagent Program: 2342

766	8F101	Env(dis)	gp120(gp120-CD4 dis)			sCD4-(rHXB2 gp120)-complex	murine(IgG)

References: [Devico (1995)]

- 8F101: MAbs specifically reactive to crosslinked gp120 and CD4 were derived (8F101, 8F102) – conformation dependent – competition studies indicate the epitope is immunogenic in infected humans –Devico95

767	8F102	Env(dis)	gp120(gp120-CD4 dis)			sCD4-(rHXB2 gp120)-complex	murine(IgG)

References: [Devico (1995)]

- 8F102: MAbs specifically reactive to crosslinked gp120 and CD4 were derived (8F101, 8F102) – conformation dependent – competition studies indicate the epitope is immunogenic in infected humans –Devico95

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
768	CG-10	Env(dis)	gp120(gp120-CD4 dis)		L	CD4/gp120 IIIB complex	murine(IgG_1)

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

- CG-10: Reacts exclusively with sCD4-gp120 complex, not with sCD4 or gp120 alone –Gershoni93
- CG-10: Called CG10 – MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4, and MAb CG10 does not block this inhibition –Wu96
- CG-10: Called CG10 – Promotes envelope mediated cell fusion between CD4+ cells and cells infected with either T-cell and macrophage tropic viruses – infection of HeLa CD4+ (MAGI) cells by HIV-1 LAI, ELI1, and ELI2 strains was increased two-to four-fold in the presence of CG10 –Lee97
- CG-10: Called CG10 – disrupts gp120-CCR5 interaction and competes with MAb 17b –binds near the conserved bridging sheet of gp120 – mutations in positions K/D 121, T/D 123, K/D 207, K/D 421, Q/L 422, Y/S 435, M/A 434, K/A 432 and I/S 423 result in a geq 70% reduction in CG10 binding –Rizzuto98
- CG-10: Called CG10 – CD4BS MAb 15e competes with CG-10 binding, probably due to the disruption of CD4-gp120 by 15e – CD4i MAbs 17b and 48d compete and the binding sites may overlap – MAb A32 enhances binding of 17b, 48d and CG10 – MAbs C11, 2G12 and 212A do not affect CG10 binding – CG-10 can bind gp120 with V1/V2 and V3 deleted – HXBc2 mutations Delta 119-205, 314 G/W, 432 K/A, 183,184 PI/SG decrease CG-10 recognition, HXBc2 mutations Delta 298–327 (V3), 384 Y/E, 298 R/G, 435 Y/S enhance recognition – the CD4 contribution to the CG10 epitope maps to the CD4 CDR2-like loop – CG10 can neutralize HIV-1 in the presence of sCD4 even though it does not do so in the context of cell surface CD4 binding to gp120 –Sullivan98

769	CG-4	Env(dis)	gp120(gp120-CD4 dis)		no	CD4/gp120 complex	murine(IgG_1)

Donor: Jonathan Gershoni, Tel Aviv University, Isreal
References: [Gershoni (1993)]

- CG-4: Reacts with gp120 and sCD4-gp120 complex, not with sCD4 –Gershoni93

770	CG-9	Env(dis)	gp120(gp120-CD4 dis)		L	CD4/gp120 complex	murine(IgG_1)

References: [Gershoni (1993)]

- CG-9: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 –Gershoni93

771	CG-25	Env(dis)	gp120(gp120-CD4 dis)		L	CD4/gp120 complex	murine(IgG_1)

References: [Gershoni (1993)]

- CG-25: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 –Gershoni93

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
772	CG-76	Env(dis)	gp120(gp120-CD4 dis)		L	CD4/gp120 complex	murine(IgG_1)

References: [Gershoni (1993)]
- CG-76: Reacts equally well with sCD4-gp120 and sCD4, but not with purified gp120 –Gershoni93

773	12H2	Env(dis)	gp41(ectodomain 530–677 HXB2)		no	Env in a Semliki Forest Virus vector	murine(IgM_κ)

References: [Giraud (1999)]
- 12H2: Env in a Semliki Forest Virus (SFV) vector was used to vaccinate mice intramuscularly as naked RNA, and an Ab response was induced to Env from which 12H2 was derived – and advantage of this method is that the protein properly expressed –Giraud99

774	N2-4	Env( )	gp41(gp41)		no	HIV-1 infection	human($IgG_1\kappa$)

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Robinson (1990b)]
- N2-4: No enhancing activity for HIV-1 IIIB –Robinson90a
- N2-4: NIH AIDS Research and Reference Reagent Program: 528

775	M25	Env( )	gp41(gp41)			purified HTLV-III	murine($IgG\kappa$)

References: [di Marzo Veronese (1985), Watkins (1996)]
- M25: heavy and light chains cloned and sequenced – binding requires heavy and light chain in combination, in contrast to M77 –Watkins96

776	10E9	Env( )	gp41(gp41)			HIV-1 infection	murine(IgG_1)

References: [Papsidero (1988)]
- 10E9: 100/100 HIV+ human sera could inhibit 10E9 binding –Papsidero88

777	3H6	Env( )	gp41(gp41)				murine( )

References: [Pinter (1995)]
- 3H6: There is another MAb with this ID that recognizes Rev –Orsini95
- 3H6: Generated in response to virus grown in protein-free medium –Pinter95b

778	31710B	Env( )	gp41(gp41)				human(IgG_1)

References: [Alsmadi & Tilley(1998)]
- 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 –Alsmadi98

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing Immunogen	Species(Isotype)
779	1342	Env( )	gp41(gp41)		HIV-1 infection	human( )

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Nyambi (1998)]

- 1342: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-gp41 Abs 98-6, 1367 and 1342 were not able to bind detectably with any of the viruses from any clade –Nyambi98

780	1367	Env( )	gp41(gp41)		HIV-1 infection	human( )

Donor: Susan Zolla-Pazner (NYU Med. Center)
References: [Nyambi (1998)]

- 1367: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-gp41 Abs 98-6, 1367 and 1342 were not able to bind detectably with any of the viruses from any clade –Nyambi98

781	NC-1	Env(dis)	gp41(gp41 core IIIB)		N36(L6)C34	murine(IgG_{2a})

Donor: S. Jiang, New York Blood Center, NY, NY
References: [Jiang (1998)]

- NC-1: Ab elicited in response to immunization with N36(L6)C34, a peptide that folds into a six helix bundle like gp41 – NC-1 binds to the surface of HIV-1 infected cells only in the presence of sCD4, recognizing the fusogenic core structure – binding affinity was decreased by point mutations that disrupt core formation and abolish membrane fusion activity, (I573P and I573A) – NC-1 can recognize discontinuous epitopes from B clade isolate SC, but not E clade strain N243, O group strain GAB, or HIV-2 ROD [Jiang (1998)]

782	Chessie 8	Env( )	gp41(gp41 cytoplasmic domain)			murine(IgG)

Donor: G. Lewis
References: [Lewis (1991), Poumbourios (1995), Rovinski (1995)]

- Chessie 8: Used to precipitate gp160 in immunoblots in a study examining the feasibility of using unprocessed gp160 glycoprotein as an immunogen –Rovinski95

783	K14	Env(dis)	gp41(gp41 dis)		no	human(IgG_1)

References: [Teeuwsen (1990), Schutten (1995a), Schutten (1995b), Schutten (1996), Schutten (1997)]

- 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 –Teeuwsen90
- K14: Reduced affinity for both SI and NSI viruses relative to MAb MN215, failed to neutralize SI strain –Schutten95a
- K14: In a study of NSI and SI virus neutralization, K14 did not influence viral entry –Schutten97

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
784	T30	Env(dis)	gp41(gp41 dis)		no	tetrameric Env	murine( )
	References: [Earl (1994), Earl (1997)]						
	• T30: binds to the region 580 to 640, but does not bind to peptides spanning this region – binding depends on N-linked glycosylation of Asn 616 – no other antibody tested inhibited binding, but binding could be inhibited by sera from HIV+ individuals –Earl97						
785	Md-1	Env(dis)	gp41(gp41 dis)		no	?	human($IgG_1\lambda$)
	Donor: R. A. Myers State of Maryland Dept. of Health						
	References: [Myers (1993), Chen (1995), Binley (1996)]						
	• Md-1: Called MD-1 – discontinuous epitope that binds in the N-terminal region – reacts exclusively with oligomer –Myers93						
	• Md-1: Called MD-1 – 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 –Chen95						
	• 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 –Binley96						
	• Md-1: NIH AIDS Research and Reference Reagent Program: 1223						
786	H2	Env(dis)	gp41(gp41 dis)			?	human($IgM\kappa$)
	Donor: BioInvent, Lund, Sweden, commercial						
	References: [Muller (1991)]						
	• 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 –Muller91						
787	MO43	Env(dis)	gp41(gp41 dis)		no	*in vitro* r Env penv9	human(IgM)
	References: [Ohlin (1989)]						
	• 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 –Ohlin89						
788	MO30	Env(dis)	gp41(gp41 dis)		no	*in vitro* r Env penv9	human(IgM)
	References: [Ohlin (1989)]						
	• 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 –Ohlin89						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
789	MO28	Env(dis)	gp41(gp41 dis)		no	*in vitro* r Env penv9	human(IgM)

References: [Ohlin (1989)]

- 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 –Ohlin89

790	126-50	Env(dis)	gp41(gp41 dis HXB2)		no	HIV-1 infection	human($IgG_{2\kappa}$)

References: [Robinson (1990b), Tyler (1990), Robinson (1991), Xu (1991)]

- 126-50: No enhancing activity for HIV-1 IIIB –Robinson90a
- 126-50: Serves as target for antibody-dependent cellular cytotoxicity ADCC –Tyler90
- 126-50: No enhancing or neutralizing activity –Robinson91
- 126-50: Specific for a conformational epitope –Xu91

791	126-6	Env(dis)	gp41(gp41 dis HXB2)		no	HIV-1 infection	human($IgG_{2\kappa}$)

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

References: [Robinson (1990b), Robinson (1991), Xu (1991), Eddleston (1993), Chen (1995), Binley (1996), Earl (1997)]

- 126-6: No enhancing activity for HIV-1 IIIB –Robinson90a
- 126-6: No enhancing or neutralizing activity –Robinson91
- 126-6: Specific for a conformational epitope –Xu91
- 126-6: Called SZ-126.6 –Eddleston93
- 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 –Chen95
- 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 –Binley96
- 126-6: NIH AIDS Research and Reference Reagent Program: 1243

792	D43	Env(dis)	gp41(gp41 dis HXB2)			dimeric Env	murine(IgG)

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

- D43: This is a linear gp41 epitope, mapping in the region 635–678 – human sera blocked binding in oligomeric ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 –Richardson96
- D43: Partially conformation dependent – doesn't bind to short peptides, but does bind to the region spanning 641–683 – binding can be blocked by MAbs T3, D38 and D45 – MAbs in this competition group reacted with 9/10 HIV-1 strains, not binding to JRFL –Earl97

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
793	T3	Env(dis)	gp41(gp41 dis HXB2)			tetrameric Env	murine(IgG)

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

- T3: Partially conformation dependent – doesn't bind to short peptides, but does bind to the region spanning 641–683 – binding can be blocked by MAbs D43, D38 and D45 – MAbs in this competition group reacted with 9/10 HIV-1 strains, not binding to JRFL –Earl97

794	T4	Env(dis)	gp41(gp41 dis IIIB)		L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

References: [Earl (1994), Broder (1994), Richardson Jr (1996), Weissenhorn (1996), Earl (1997), Otteken (1996), Binley (1999)]

- 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 –Broder94
- 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 –Weissenhorn96
- 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 –Earl97
- 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 –Otteken96
- T4: The MAbs with the broadest neutralizing activity, IgG_1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519; nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes –Binley00

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
795	D12	Env(dis)	gp41(gp41 dis IIIB)		L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

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

- 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 –Broder94
- D12: This antibody was blocked more strongly by human sera than other anti-gp41 MAbs (D20, D43, D61, and T4) in a oligomeric ELISA assay –Richardson96
- D12: MAbs D10 and D12 are very easily blocked by human sera from HIV+ individuals –Earl97
- 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 –Otteken96

796	D1	Env(dis)	gp41(gp41 dis IIIB)			vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

References: [Otteken (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 –Otteken96

797	D16	Env(dis)	gp41(gp41 dis IIIB)		L	dimeric Env	murine(IgG)

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

- 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 –Weissenhorn96
- 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) –Earl97

798	Fab D5	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab D5: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96

799	Fab D11	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab D11: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
800	Fab G1	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab G1: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96

801	Fab T3	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab T3: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96

802	Fab M10	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996), Parren (1997b)]

- Fab M10: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96
- Fab M10: Does not bind to MN native oligomer, but does bind to both LAI and MN rgp120 and rgp140 –Parren97

803	Fab M12	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab M12: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96

804	Fab M15	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab M15: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96

805	Fab S6	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab S6: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96

806	Fab S8	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)

References: [Binley (1996)]

- Fab S8: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
807	Fab S9	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
	References: [Binley (1996)]						
	• Fab S9: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96						
808	Fab S10	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
	References: [Binley (1996)]						
	• Fab S10: Binds to Cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced –Binley96						
809	Fab L2	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
	Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California						
	References: [Binley (1996), Earl (1997)]						
	• Fab L2: Binds to Cluster III region – competes with MAb Md-1, but not MAbs 126–6 and D50 – conformation sensitive – variable regions sequenced –Binley96						
810	Fab L11	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
	References: [Binley (1996)]						
	• Fab L11: Binds to Cluster III region – competes with MAb Md-1, but not MAbs 126–6 and D50 – conformation sensitive – variable regions sequenced –Binley96						
811	Fab L1	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
	References: [Binley (1996)]						
	• Fab L1: Binds to Cluster III region – competes with MAb Md-1, but not MAbs 126–6 and D50 – conformation sensitive – variable regions sequenced –Binley96						
812	Fab G5	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
	References: [Binley (1996)]						
	• Fab G5: Binds to Cluster III region – competes with MAb Md-1, but not MAbs 126–6 and D50 – conformation sensitive – variable regions sequenced –Binley96						
813	Fab G15	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
	References: [Binley (1996)]						
	• Fab G15: Binds to Cluster III region – competes with MAb Md-1, but not MAbs 126–6 and D50 – conformation sensitive – variable regions sequenced –Binley96						

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
814	Fab A9	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
		References: [Binley (1996)]					
		• Fab A9: Binds to Cluster III region – competes with MAb Md-1, but not MAbs 126–6 and D50 – conformation sensitive – variable regions sequenced –Binley96					
815	Fab A12	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
		References: [Binley (1996)]					
		• Fab A12: Uncharacterized epitope – variable regions sequenced –Binley96					
816	Fab L9	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\kappa$)
		References: [Binley (1996)]					
		• Fab L9: Uncharacterized epitope – variable regions sequenced –Binley96					
817	Fab A2	Env(dis)	gp41(gp41 dis LAI)		no	HIV-1 infection	human($IgG_1\lambda$)
		References: [Binley (1996)]					
		• Fab A2: Uncharacterized epitope – variable regions sequenced –Binley96					
818	2A2	Env( )	gp41(gp41 N-term)		no	HIV-1 infection	human($IgG_1\kappa$)
		References: [Weissenhorn (1996)]					
		• 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 –Weissenhorn96					
819	31A1	Env( )	gp41(p24+gp41)		no	*in vitro* immunization, denatured HIV-1	human($IgM\kappa/\lambda$)
		References: [Pollock (1989)]					
		• 31A1: Reacts with both p24 and gp41 –Pollock89					
820	39A64	Env( )	gp41(p24+gp41)		no	*in vitro* immunization, denatured HIV-1	human($IgM\kappa/\lambda$)
		References: [Pollock (1989)]					
		• 39A64: Reacts with both p24 and gp41 –Pollock89					
821	39B86	Env( )	gp41(p24+gp41)		no	*in vitro* immunization, denatured HIV-1	human($IgM\kappa/\lambda$)
		References: [Pollock (1989)]					
		• 39B86: Reacts with both p24 and gp41 –Pollock89					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
822	9303	Env( )	gp41(p24+gp41)		no		murine( )
		Donor: Du Pont **References:** [McDougal (1996)]					
823	N70-2.3a	Env(dis)	gp120(272-509 dis)		no	HIV-1 infection	human(IgG_1)
		Donor: J. Robinson, Tulane University, LA **References:** [Robinson (1990a), Takeda (1992)] • N70-2.3a: Broad reactivity –Robinson90c • N70-2.3a: Fc receptor mediated enhancement of HIV-1 infection – binds a conformational site in the carboxyl half of gp120, distinct from 1.5e –Takeda92					
824	1B1	Env( )	Env(Env)		L	HIV-1 infection	human( )
		Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, Vienna, Austria **References:** [Buchacher (1994), Purtscher (1994), Kunert (1998)] • 1B1: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells –Buchacher94 • 1B1: The complete V, J and D(H) domain was sequenced – unlike non-neutralizing anti-gp41 MAb 3D6, five neutralizing MAbs (2F5, 2G12, 1B1, 1F7, and 3D5) showed extensive somatic mutations giving evidence of persistent antigenic pressure over long periods –Kunert98					
825	1F7	Env( )	Env(Env)		L	HIV-1 infection	human( )
		Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, Vienna, Austria **References:** [Buchacher (1994), Purtscher (1994), Kunert (1998)] • 1F7: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells –Buchacher94 • 1F7: The complete V, J and D(H) domain was sequenced – unlike non-neutralizing anti-gp41 MAb 3D6, five neutralizing MAbs (2F5, 2G12, 1B1, 1F7, and 3D5) showed extensive somatic mutations giving evidence of persistent antigenic pressure over long periods –Kunert98					
826	3D5	Env( )	Env(Env)		L	HIV-1 infection	human( )
		Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, Vienna, Austria **References:** [Buchacher (1994), Purtscher (1994), Kunert (1998)] • 3D5: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells –Buchacher94 • 3D5: The complete V, J and D(H) domain was sequenced – unlike non-neutralizing anti-gp41 MAb 3D6, five neutralizing MAbs (2F5, 2G12, 1B1, 1F7, and 3D5) showed extensive somatic mutations giving evidence of persistent antigenic pressure over long periods –Kunert98					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
827	MAG 6B	Env(dis)	gp120(Env dis)		no	sCD4-(rHXB2 gp120)-complex	murine( )
		Donor: C. Y. Kang, IDEC Inc **References:** [Kang (1994)] • MAG 6B: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R or G or A, 262 N/T, 368 D/R or T, 370 E/R or Q, 381 E/P, 384 Y/E, 421 K/L, 475 M/S, 477 D/V –Kang94					
828	P43110	Env(dis)	gp120(Env dis)			unk	( )
		Donor: Advanced Biosciences (Kensington, MD) **References:** [di Marzo Veronese (1992), VanCott (1995)] • P43110: Does not recognized denatured form of the gp120 protein –VanCott95					
829	6E10	Env(dis)	gp120(Env dis)		L	rsgp160	( )
		Donor: Phil Berman **References:** [Berman (1991)]					
830	multiple MAbs	Env(dis)	gp120(Env dis)			gp120	murine( )
		References: [Denisova (1996)] • When gp120 was used as an immunogen, in contrast to gp120 bound to an anti-V3 MAb, few MAbs were generated and all bound better to the native than to the denatured protein – MAbs generated were: G1B12, G2F7, G9G8, G12F12, G1B8, G11F11, G9E8, G1B11, G1B6, G6F2, G2E7 –Denisova96					
831	multiple MAbs	Env(dis)	gp120(Env dis)			gp120-CD4 complex	murine( )
		References: [Denisova (1996)] • When gp120-CD4 was used as an immunogen, in contrast to gp120 bound to an anti-V3 MAb, few MAbs were generated and all bound better to the native than to the denatured protein – MAbs generated were: CG43, CG41, CG49, CG53, CG42, CG4, CG46, CG40, CG52, CG51, CG48, CG50, CG125, CG124, CG121 –Denisova96					
832	1025	Env(dis)	gp120(Env dis)				( )
		References: [Berman (1997)] • 1025: Binds to 1/7 isolates from breakthrough cases from a MN gp120 vaccine trial –Berman97					

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species(Isotype)
833	D20	Env(dis)	gp120(Env dis IIIB)		no	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)

Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD
References: [Earl (1994), Broder (1994), Richardson Jr (1996), Otteken (1996), Earl (1997), Sugiura (1999)]

- D20: Binding completely blocked by pooled human sera –Broder94
- D20: Human sera blocked binding in oligomeric ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 –Richardson96
- D20: Pulse label experiments of 4 MAbs (D20, D27, T20, and T22) binding to noncleavable gp160 revealed that these anti-CD4 MAbs bound with a delay, and that the epitope formed with a $t_{1/2}$ of about 10 minutes –Otteken96
- D20: Used for comparison in a study of gp41 antibodies – D20 binds to a greater extent to cell surface expressed Env than any of 38 conformation dependent anti-gp41 MAbs –Earl97
- D20: A of comparison of 25 gp120 specific, conformation dependent MAbs was done – D20 is part of a group of MAbs labeled A1 – all A1 MAbs were broadly cross-reactive with gp160 from B-clade R5, X4, and R5X4 viruses, blocked CD4 binding, were sensitive to mutations in gp120 positions 368 and 370 that directly contact CD4 –Sugiura99

834	polyclonal	Env( )	gp120( )		L	HIV-1 infection	chimpanzee(IgG)

References: [Shibata (1999)]

- polyclonal: Purified IgG from chimpanzee sera infected with several HIV-1 strains was used for passive administration to macaques which were subsequently challenged with the virulent SHIV bearing the HIV-1 env DH12 – *in vitro* neutralization correlated with protection *in vivo* –Shibata99