

PART IV: ANTIBODY BINDING SITES

How to Use Part IV: Monoclonal Antibody Index (MAB), Anti-HIV Antibody Tables, and Maps

This section summarizes HIV-specific antibodies arranged sequentially according to the location of their binding domain, organized by protein. We attempted to make this section as comprehensive as possible. For the MABs capable of binding to linear peptides, we require that the binding site be contained within a region of 30 or so amino acids, but not that the precise boundaries be defined. MABs that cannot bind to linear peptides are grouped by category at the end of each protein. Antibody categories, for example CD4BS antibodies, are noted in the index at the beginning of this section. Studies of polyclonal Ab responses are also included. Responses that are just characterized by binding to a protein, with no known specific binding site, are listed at the end of each protein. For more recent updates, epitope sequence alignments, and search capabilities, please see our web site: <http://hiv-web.lanl.gov/immunology>.

A. INDICES

Two indices are provided. The first lists the MAB's IDs in alphabetical order so one can find their location in the table. The second provides a concise list of the MABs in order of their appearance in the antibody table, *i.e.*, ordered by the protein coding regions spanning HIV-1.

B. TABLES:

Each MAB has a nine-part basic entry:

- **Number:** Order of appearance in this table.
 - **MAB ID:** The name of the monoclonal antibody with synonyms in parentheses. MABs often have several names. For example, punctuation can be lost and names are often shortened (M-70 in one paper can be M70 in another). Polyclonal responses are listed as "polyclonal" in this field.
 - **HXB2 Location:** Position of the binding site on the viral strain HXB2, which is used as a reference strain throughout this publication. The numbering corresponds to the protein maps. Because of HIV-1 variation the epitope may not actually be present in HXB2, and the position in HXB2 indicates the position aligned to the epitope.
 - **Author Location:** The amino acid positions of the epitope boundaries and the reference sequence are listed as given in the primary publication.
- Frequently, these positions as published are imprecise, and do not truly correspond to the numbering of the sequence, but they provide a reasonable guide to the peptide's approximate location in the protein. Also, in many cases, reference sequence identification was not provided; because of HIV-1's variability, position numbers require a reference strain to be meaningful. Binding sites that cannot be defined through peptide binding or interference studies are labeled as discontinuous. The approximate location on the protein, sequence number, and reference sequence are listed. If you are interested in finding the precise positions of an antigen you are studying on the HXB2 reference strain, please try using the interactive position locator at our web site: <http://hiv-web.lanl.gov/NUM-HXB2/HXB2.MAIN.html>.
- **Sequence:** The amino acid sequence of the binding region of interest, based on the reference strain used in the study defining the binding site. On rare occasions, when only the position numbers and not the actual peptide sequence was specified in the original publication, if the sequences were numbered inaccurately by the primary authors, we may have misrepresented the binding site's amino acid sequence. Therefore, epitopes that were not explicitly written in the primary publication, that we determined by looking up the reference strain and the numbered location, are followed by a question mark in the table.
 - **Neutralizing:** **L:** neutralizes lab strains. **P:** neutralizes primary isolates. **no:** does not neutralize.
 - **Immunogen:** The antigenic stimulus of the original B cell response. If a vaccine was used as the original antigenic stimulation, not a natural infection, this is noted on a separate line, and additional information about the vaccine antigen is provided as available.
 - **Species(Isotype):** The host that the antibody was generated in, and the isotype of the antibody.
 - **References:** All publications that we could find that refer to the use of a specific monoclonal antibody. First is a list of all references. Second is a list of the donors, and is meant to serve as a potential guide to a source of information about an antibody or how to obtain it, as well as to provide credit. Then comes a list of notes describing the context of each study, and what was learned about the antibody in the study.

HIV Antibodies

C. MAPS

The names of MAbs and the location of well characterized linear binding sites of 21 amino acids or less are indicated on protein sequences of the HXB2 clone. This map is meant to provide the relative location of epitopes on a given protein, but the HXB2 sequence may not actually bind to the MAb of interest, as it may vary relative to the sequence for which the epitope was defined. HXB2 was selected as the reference strain because so many studies use HXB2, and because crystal structures for HXB2-related proteins are often available.

ALIGNMENTS

Alignments that correspond to the epitopes are only available from the web site, not in this book, because of space limitations. All epitopes are aligned to the HXB2 sequence, with the sequence used to define the epitope indicated directly above it. In consensus sequences an upper case letter indicates the amino acid was present in all sequences, a lower case letter indicates the amino acid was present in most sequences in a given position, and a question mark indicates two or more amino acids were represented with equal frequency.

The master alignment files from which the epitope alignments were created are available at our web site (http://hiv-web.lanl.gov/ALIGN_CURRENT/ALIGN-INDEX.html), and we restricted ourselves to full protein sequences for these alignments, excluding short fragments of sequences. The subtype designation and the country of isolation are indicated along with the common name of the sequence. The alignments were modified in some cases to optimize the alignment relative to the defined epitope and minimize insertions and deletions; epitope alignments are generated by anchoring on the C-terminal residue. A dash indicates identity to the consensus sequence, and a period indicates an insertion made to maintain the alignment. Stop codons are indicated with a \$, and frameshifts by a #, or ambiguous codons (nucleotide was r, y, or n) by an x; they are inserted to maintain the alignments.

D. REFERENCES AND NOTES

Part IV-A: Index of HIV-1 Antibodies

**Cross reference of MAb names and their
order of appearance in the MAb tables.
Alphanumeric sorting order is symbols,
numbers, capital letters, lowercase letters.**

HIV Antibodies

Name	No.	Name	No.	Name	No.	Name	No.	Name	No.
	783	10E3	478	11C10B10	103	1367	787	187.2.1	298
α	560	10E7	163	11D11F2	104	1379	791	1899	656
μ 5.5	448	10E9	885	11H9	25	1393A	842	19	206
0.5 β	471	10F10	396	12	215	13B5	113	1907	657
1-B-7	68	11	312	12-B-4	100	13E1	165	1908	658
1-E-4	57	11-C-5	61	120-1	681	13H8	512	1909	659
1-E-9	58	11/41e	326	120-16	629	14	217	19b	428
1.152 B3	170	11/4b	327	120-1B1	703	14D4E11	50	1A1	556
1.153 G10	178	11/4c	329	1202-D	704	15-21	32	1A7	89
1.158 E2	171	11/65	308	126-50	886	1570	706	1B1	889
1.160 B3	182	11/68b	832	126-6	788	1575	665	1B2C12	86
1.17.3	88	11/75a/21/41	870	1281	789	1576	652	1B8.env	615
1.2	241	11/85b	469	12G-A8g2	18	1577	672	1C1	528
10-E-7	59	110-B	839	12G-D7h11	19	1578	653	1C12B1	220
10-G-9	60	110.1	343	12G-H1c7	20	1579	654	1C4	189
10.1	252	110.1	541	12G10	313	1583	655	1D10	276
10/36e	467	110.3	458	12H-D3b3	17	1595	707	1D2F11	243
10/46c	700	110.4	459	12H2	887	1599	708	1D4A3	180
10/54	468	110.5	460	12I-D12g2	21	15F8C7	45	1D9	28
10/76b	325	110.6	465	12b	330	15e	709	1D9D5	242
1006-15D	417	110.C	366	13	216	16	218	1E8	169
1006-30-D	347	110.D	493	13-102-100	76	1662	515	1F11	564
101-342	679	110.E	365	13.10	888	1663	516	1F6	90
101-451	680	110.I	486	13/035	984	1664	517	1F7	890
102-135	882	110.J	855	13/042	983	167-7	631	1G10	258
1024	699	110/015	109	13/058	993	167-D	632	1G5C8	51
1025	883	1108	873	1324-E	388	1696	162	1G7	259
1026	474	1109/01	49	133/11	270	1697	518	1H5	565
1027-15D	420	111/052	46	133/192	278	17	219	2-19	207
1027-30-D	701	111/073	55	133/237	268	178.1	401	2-E-4	62
1034	475	111/182	36	133/290	269	1794	519	2-H-4	63
105-134	884	112/021	37	1331A	537	1795	503	2/11c	694
105-306	545	112/047	38	1331E	705	17b	784	205-43-1	710
105-518	820	1125H	702	1334-D	856	1804	520	205-46-9	711
105-732	624	113/038	56	1342	790	1807	521	21	221
106/01	114	113/072	74	135/9	314	1808	522	212A	684
108/03	108	1131-A	553	1357	840	181-D	594	213.1	352
1088	838	115.8	601	1361	841	183-H12-5C	133	21h	712

HIV Antibodies

Name	No.	Name	No.	Name	No.	Name	No.	Name	No.
23A	682	31/03	998	3E6	1004	4	618	559/64-D	720
23A5G4	92	311-11-D	403	3F10	226	4A7C6	275	58.2	461
23A5G5	93	31710B	891	3F2	989	4B3	567	588-D	721
240-D	595	31A1	827	3F5	529	4B4C4	245	59.1	476
241-D	134	31D6	172	3F9	193	4C11.D8	341	5B2	176
246-D	592	31G8	173	3G12	992	4C9	29	5B2	641
24G3	557	32	222	3G4	257	4D4	568	5B3	280
25.3	75	32/1.24.89	10	3H6	253	4D4#85	264	5C2E5	499
25/03	987	32/5.8.42	3	3H6	893	4D6	199	5D9	204
257-D	402	32/5.8.42	24	4	227	4D7/4	490	5E2.A3k	136
25C2	558	322-151	339	4-20	209	4E10	643	5F	186
26/028	994	32:32K	110	406/01	79	4F6	201	5F3	559
26/76	988	32E7	174	41-1	577	4G10	429	5F4/1	530
268-D	450	33	223	41-1	660	4G2	569	5F7	430
2A2	821	33D5	175	41-2	661	4G9	250	5F8	194
2A2/26	573	35	224	41-3	662	4H2B1	30	5G	187
2A3	1005	36.1	491	41-6	619	4H4	981	5G11	858
2A6	135	37.1.1	299	41-7	620	5-21-3	628	5G7D8	246
2C11	190	38/12b	336	41.1	871	50-61A	717	6-19	210
2C4	397	38/60b	337	41.4	578	50-69	574	6-D-12	70
2D9D5	249	386-D	451	41148D	404	50.1	413	6-E-7	71
2D9E7	244	38:9.6K	80	4117C	434	5020	463	6.1	1015
2E3	191	391/95-D	405	412-D	398	5021	455	60b	334
2E3	995	39A64	828	418-D	454	5023A	464	62c	833
2E4	1006	39B86	829	419-D	435	5023B	439	654-D	722
2F11	591	3A2	1007	41S-2	647	5025A	481	660-178	531
2F19C	697	3A6	35	428	714	5025B	456	66a	843
2F2	1002	3B10	11	42F	542	504-D	437	66c	844
2F5	634	3D10G6	94	43F	543	5042	457	670-D	549
2G12	878	3D12	225	447-52D	648	5042A	452	68.1	621
2G2	261	3D12	990	448-D	715	5042B	453	68.11	622
2G6	713	3D3	43	450-D	548	5145A	718	684-238	845
2H12	1008	3D3.B8	340	453-D	436	522-149	685	694/98-D	484
2H1B	321	3D5	892	47-2	52	537-D	462	697-D	322
3-B-7	69	3D6	625	48-16	716	55/11	375	6B9	185
3-H-7	26	3D9	566	489.1	279	55/45a/11	872	6B9	228
30:3E5	83	3E11	12	48d	785	55/68b	857	6C4/S	323
31-11	33	3E11	192	493-156	342	558-D	719	6C5	198

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Name	No.	Name	No.	Name	No.	Name	No.	Name	No.
6D5	318	88-158/02	666	A32	786	B32	494	CB-13/5	41
6D8	300	88-158/022	667	A47/B1	445	B33	319	CD-4/1	44
6E10	894	88-158/079	668	A9	896	B33	651	CD12B4	105
6G5	195	8B11	166	AC2	141	B34	496	CD9	145
7-1054	895	8C10	167	AC4	822	B35	291	CG-10	814
7-16	200	8C6/1	534	AD2	125	B36	364	CG-25	815
71-31	137	8E5	213	AD3	823	B4	897	CG-4	816
714/01	53	8E7	254	AD3	824	B4f8	15	CG-76	817
722-D	551	8F101	812	AE6	1012	B5	898	CG-9	818
729-D	723	8F102	813	AE6	1016	B6	899	CGP 47 439	400
74	335	8G4	202	AG11	1013	B8	671	CH9B2	146
75	623	8G5	168	AG1121	860	B9	290	CRA-3	847
750-D	547	8H10	13	AM5C6	985	BAT085	333	CRA-4	848
782-D	418	9-11	575	AM5C6	986	BAT123	415	CRA-6	834
7B6	196	902	483	Ab2	251	BAT267	900	CRA1	525
7C10	315	907	392	Ab3	260	BAT401	901	Chessie 8	810
7C3	211	908-D	419	Ab4	256	BAT509	902	Chim 1	539
7C4	188	91-5	48	Aw	406	BC1071	143	D/3G5	271
7C4	229	91-6	138	B10	281	BE10	144	D/4B5	292
7C6	197	9201	527	B12	353	BE3	106	D/5A11	293
7E2/4	263	9205	485	B13	354	BM12	732	D/5E12	273
7F11	212	924	393	B15	495	Bw	407	D/6A11	272
7F11	498	9284	380	B18	295	C β 1	472	D/6B2	294
8-22	208	9301	532	B2	282	C108G	324	D/6D1	489
8-6	205	9303	830	B20	296	C11	695	D1	904
8-D-2	64	9305	859	B21	357	C12	492	D12	905
8-D-5	72	98-4.3	139	B221	533	C13	355	D16	906
8-G-9	65	98-4.9	140	B23	358	C2003	184	D20	733
8-H-7	66	98-43	576	B24	359	C31	903	D21	734
8/38c	376	98-6	630	B242	277	C311E	391	D24	735
8/64b	377	989-D	555	B25	360	C4	316	D25	736
83.1	438	9A4C4	102	B26	362	C5122	101	D27	875
830A	846	9CL	725	B27	289	C5123	67	D28	737
830D	724	9G11	642	B29	363	C5126	27	D33	782
838-D	416	9G2	255	B2C	698	C5200	111	D35	738
847-D	348	9G5	31	B3	361	C6	283	D39	739
858-D	554	9G5A	593	B30	645	C8	649	D4	907
86	585		881	B31	650	CA5	126	D42	740

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Name	No.	Name	No.	Name	No.	Name	No.	Name	No.
D43	908	F240	596	G11G1	149	HyHIV-19	152	L28	759
D47	862	F285	910	G11H3	150	HyHIV-2	5	L33	760
D49	597	F5-2	40	G12	915	HyHIV-21	14	L39	852
D50	627	F5-4	95	G2	916	HyHIV-22	16	L40	853
D52	741	F5.5	863	G3-136	331	HyHIV-3	6	L41	761
D53	742	F58/D1	440	G3-1472	864	HyHIV-4	7	L42	762
D56	876	F7	911	G3-211	500	HyHIV-5	8	L5.1	274
D59/A2	446	F91	747	G3-299	504	HyHIV-6	9	L52	763
D60	743	FC12	129	G3-4	332	ICR 39.13g	755	L72	764
D61	598	FF1	73	G3-42	505	ICR 39.3b	756	L78	854
D7324	683	FH2	112	G3-508	506	ICR38.1a	509	L81	696
DA48	744	Fab A1	579	G3-519	507	ICR38.8f	510	LA9	664
DF3	127	Fab A12	912	G3-523	431	ID6	825	LH-104-A	154
DG8	122	Fab A2	913	G3-536	508	ID6	826	LH-104-B	115
DO142-10	408	Fab A4	580	G3-537	501	ID8F6	39	LH-104-C	155
DO8i	745	Fab A9	804	G44/H7	447	IE8G2	153	LH-104-E	85
DZ	674	Fab D11	792	G45-60	513	IIIB-13 V3	443	LH-104-G	118
Dv	409	Fab D5	793	GE4	130	IIIB-34 V3	444	LH-104-I	116
E7	1011	Fab G1	794	GP13	748	IIIB-V3-01	488	LH-104-K	87
E9	1003	Fab G15	805	GP44	749	IIIB-V3-21	368	M-1	602
EB1A9	81	Fab G5	806	GP68	750	IIIB-V3-26	367	M-11	603
EB5	123	Fab L1	807	GV1A8	311	IVI-4G6	675	M-13	604
EC3	128	Fab L11	808	GV1G2	546	IgG1b12	757	M-2	605
EC6	120	Fab L2	809	GV4D3	288	IgGCD4	758	M-22	606
ED6	663	Fab L9	914	GV4H3	344	J1	345	M-24	607
ED8	147	Fab M10	795	Gv	411	J3	346	M-25	608
EF7	84	Fab M12	796	H11	535	JB7	131	M-28	609
EH1	1014	Fab M12B	581	H2	917	JF11	132	M-29	610
EH12E1	148	Fab M15	797	H8	918	K14	920	M-36	611
F1	1001	Fab M26B	582	HBW4	919	K24	865	M-4	612
F105	746	Fab M8B	583	HF1.7	751	L-anti-Tat	248	M-6	613
F11.2.32	164	Fab S10	798	HH3	124	L100	693	M096/V3	866
F14.11	997	Fab S6	799	HT5	752	L14	107	M12	121
F172-D8	626	Fab S8	800	HT6	753	L14.17	1	M12	765
F19.26-4	421	Fab S9	801	HT7	754	L15	835	M13	766
F19.48-3	422	Fab T2	584	Hv	412	L17	849	M25	921
F19.57-11	423	Fab T3	802	HyHIV-1	4	L19	686	M38	538
F223	909	Fv	410	HyHIV-15	23	L25	851	M6	767

HIV Antibodies

Name	No.	Name	No.	Name	No.	Name	No.	Name	No.
M77	424	MO101/V3,C4	874	RTMAb8	179	anti-HIV-2 polyclonal	487	polyclonal	233
M85	262	MO101/V3,C4	879	RV110026	544	anti-K159	203	polyclonal	239
M86	266	MO101/V3,C4	880	S1-1	777	anti-gp120/V3	861	polyclonal	267
M89	356	MO28	923	SC258	850	anti-p24	142	polyclonal	320
M90	687	MO30	924	SP.BAL114	425	b11	727	polyclonal	338
M91	526	MO43	925	SP.SF2:104	426	b13	728	polyclonal	369
M92	265	MO86/C3	511	T1.1	285	b14	729	polyclonal	370
M96	301	MO9.42.2	96	T11	310	b3	730	polyclonal	371
MAG 104	688	MO9.50.2	97	T13	778	b6	731	polyclonal	372
MAG 109	384	MO97/V3	373	T2.1	307	clone 3	617	polyclonal	374
MAG 116	768	MO99/V3	390	T20	976	human sera	151	polyclonal	378
MAG 12B	769	MTW61D	776	T22	811	i5B11	119	polyclonal	379
MAG 29B	770	Md-1	803	T27	977	loop 2	449	polyclonal	381
MAG 3B	771	N11-20	480	T3	978	multiple Fabs	926	polyclonal	382
MAG 45	689	N2-4	930	T30	979	multiple MAbs	927	polyclonal	383
MAG 49	385	N70-1.9b	482	T32	599	multiple MAbs	928	polyclonal	389
MAG 53	386	N70-2.3a	931	T34	600	multiple MAbs	929	polyclonal	394
MAG 55	772	NC-1	819	T4	980	p7	692	polyclonal	395
MAG 56	387	ND-15G1	633	T49	779	polyclonal	2	polyclonal	399
MAG 6B	922	NF1A1	1009	T52	836	polyclonal α	562	polyclonal	414
MAG 72	773	NF2B2	1017	T54	837	polyclonal α	614	polyclonal	427
MAG 86	774	NF3A3	1018	T56	780	polyclonal	22	polyclonal	466
MAG 95	690	NF8B4	1019	T7.1	286	polyclonal	42	polyclonal	470
MAG 96	775	NM-01	473	T9	287	polyclonal	47	polyclonal	477
MAG 97	691	NT2/4D5.24	247	TG001	238	polyclonal	54	polyclonal	479
MAb 35	214	NT3/2D1.1	240	TG002	237	polyclonal	78	polyclonal	497
MF119.1	302	Nea 9301	433	TH-Ab1	640	polyclonal	82	polyclonal	502
MF169.1	349	P1/D12	441	TH1	869	polyclonal	91	polyclonal	514
MF170.1	350	P4/D10	442	TH9	781	polyclonal	117	polyclonal	523
MF39.1	297	P43110	932	V10	98	polyclonal	156	polyclonal	524
MF4.1	303	P5-3	933	V10-9	587	polyclonal	157	polyclonal	540
MF46.1	317	PC5009	561	V107	99	polyclonal	158	polyclonal	550
MF49.1	284	RL4.72.1	77	V7-8	161	polyclonal	159	polyclonal	552
MF53.1	304	RSD-33	328	W1	309	polyclonal	160	polyclonal	563
MF58.1	305	RT-4	234	W2	536	polyclonal	177	polyclonal	570
MF77.1	306	RT6H	181	Z13	644	polyclonal	183	polyclonal	571
MF87.1	351	RT7O	235	anti-CD4BS summary	726	polyclonal	231	polyclonal	572
MN215	432	RT7U	236	anti-HIV-1 RT	230	polyclonal	232	polyclonal	586

Name	No.	Name	No.
polyclonal	588	polyclonal	952
polyclonal	589	polyclonal	953
polyclonal	590	polyclonal	954
polyclonal	616	polyclonal	955
polyclonal	635	polyclonal	956
polyclonal	636	polyclonal	957
polyclonal	637	polyclonal	958
polyclonal	638	polyclonal	959
polyclonal	639	polyclonal	960
polyclonal	646	polyclonal	961
polyclonal	669	polyclonal	962
polyclonal	670	polyclonal	963
polyclonal	673	polyclonal	964
polyclonal	676	polyclonal	965
polyclonal	677	polyclonal	966
polyclonal	678	polyclonal	967
polyclonal	831	polyclonal	968
polyclonal	867	polyclonal	969
polyclonal	868	polyclonal	970
polyclonal	877	polyclonal	971
polyclonal	934	polyclonal	972
polyclonal	935	polyclonal	973
polyclonal	936	polyclonal	974
polyclonal	937	polyclonal	975
polyclonal	938	polyclonal	982
polyclonal	939	polyclonal	991
polyclonal	940	polyclonal	996
polyclonal	941	polyclonal	999
polyclonal	942	polyclonal	1000
polyclonal	943	polyclonal	1010
polyclonal	944	sc-FV p17	34
polyclonal	945		
polyclonal	946		
polyclonal	947		
polyclonal	948		
polyclonal	949		
polyclonal	950		
polyclonal	951		

HIV Antibodies

No.	Name	No.	Name	No.	Name	No.	Name	No.	Name
p17		p24		72	8-D-5	110	32:32K	146	CH9B2
1	L14.17	35	3A6	73	FF1	111	C5200	147	ED8
2	polyclonal	36	111/182	74	113/072	112	FH2	148	EH12E1
3	32/5.8.42	37	112/021	75	25.3	113	13B5	149	G11G1
4	HyHIV-1	38	112/047	76	13-102-100	114	106/01	150	G11H3
5	HyHIV-2	39	ID8F6	77	RL4.72.1	115	LH-104-B	151	human sera
6	HyHIV-3	40	F5-2	78	polyclonal	116	LH-104-I	152	HyHIV-19
7	HyHIV-4	41	CB-13/5	79	406/01	117	polyclonal	153	IE8G2
8	HyHIV-5	42	polyclonal	80	38:9.6K	p2p7p1p6		154	LH-104-A
9	HyHIV-6	43	3D3	81	EB1A9	118	LH-104-G	155	LH-104-C
10	32/1.24.89	44	CD-4/1	82	polyclonal	119	i5B11	156	polyclonal
11	3B10	45	15F8C7	83	30:3E5	120	EC6	157	polyclonal
12	3E11	46	111/052	84	EF7	121	M12	158	polyclonal
13	8H10	47	polyclonal	85	LH-104-E	122	DG8	159	polyclonal
14	HyHIV-21	48	91-5	86	1B2C12	123	EB5	160	polyclonal
15	B4f8	49	1109/01	87	LH-104-K	124	HH3	161	V7-8
16	HyHIV-22	50	14D4E11	88	1.17.3	125	AD2	Protease	
17	12H-D3b3	51	1G5C8	89	1A7	126	CA5	162	1696
18	12G-A8g2	52	47-2	90	1F6	127	DF3	163	10E7
19	12G-D7h11	53	714/01	91	polyclonal	128	EC3	164	F11.2.32
20	12G-H1c7	54	polyclonal	92	23A5G4	129	FC12	165	13E1
21	12I-D12g2	55	111/073	93	23A5G5	130	GE4	166	8B11
22	polyclonal	56	113/038	94	3D10G6	131	JB7	167	8C10
23	HyHIV-15	57	1-E-4	95	F5-4	132	JF11	168	8G5
24	32/5.8.42	58	1-E-9	96	MO9.42.2	Gag		RT	
25	11H9	59	10-E-7	97	MO9.50.2	133	183-H12-5C	169	1E8
26	3-H-7	60	10-G-9	98	V10	134	241-D	170	1.152 B3
27	C5126	61	11-C-5	99	V107	135	2A6	171	1.158 E2
28	1D9	62	2-E-4	100	12-B-4	136	5E2.A3k	172	31D6
29	4C9	63	2-H-4	101	C5122	137	71-31	173	31G8
30	4H2B1	64	8-D-2	102	9A4C4	138	91-6	174	32E7
31	9G5	65	8-G-9	103	11C10B10	139	98-4.3	175	33D5
32	15-21	66	8-H-7	104	11D11F2	140	98-4.9	176	5B2
33	31-11	67	C5123	105	CD12B4	141	AC2	177	polyclonal
34	sc-FV p17	68	1-B-7	106	BE3	142	anti-p24	178	1.153 G10
		69	3-B-7	107	L14	143	BC1071	179	RTMAb8
		70	6-D-12	108	108/03	144	BE10	180	1D4A3
		71	6-E-7	109	110/015	145	CD9	181	RT6H

HIV Antibodies

No.	Name	No.	Name	No.	Name	No.	Name	No.	Name
182	1.160 B3	219	17	254	8E7	291	B35	329	11/4c
183	polyclonal	220	1C12B1	255	9G2	292	D/4B5	330	12b
184	C2003	221	21	256	Ab4	293	D/5A11	331	G3-136
185	6B9	222	32	257	3G4	294	D/6B2	332	G3-4
186	5F	223	33	258	1G10	295	B18	333	BAT085
187	5G	224	35	259	1G7	296	B20	334	60b
188	7C4	225	3D12	260	Ab3	297	MF39.1	335	74
	Integrase	226	3F10	261	2G2	298	187.2.1	336	38/12b
189	1C4	227	4		gp160	299	37.1.1	337	38/60b
190	2C11	228	6B9	262	M85	300	6D8	338	polyclonal
191	2E3	229	7C4	263	7E2/4	301	M96	339	322-151
192	3E11	230	anti-HIV-1 RT	264	4D4#85	302	MF119.1	340	3D3.B8
193	3F9	231	polyclonal	265	M92	303	MF4.1	341	4C11.D8
194	5F8	232	polyclonal	266	M86	304	MF53.1	342	493-156
195	6G5	233	polyclonal	267	polyclonal	305	MF58.1	343	110.1
196	7B6	234	RT-4	268	133/237	306	MF77.1	344	GV4H3
197	7C6	235	RT7O	269	133/290	307	T2.1	345	J1
198	6C5	236	RT7U	270	133/11	308	11/65	346	J3
199	4D6		Vif	271	D/3G5	309	W1	347	1006-30-D
200	7-16	237	TG002	272	D/6A11	310	T11	348	847-D
201	4F6	238	TG001	273	D/5E12	311	GV1A8	349	MF169.1
202	8G4	239	polyclonal	274	L5.1	312	11	350	MF170.1
203	anti-K159		Tat	275	4A7C6	313	12G10	351	MF87.1
204	5D9	240	NT3/2D1.1	276	1D10	314	135/9	352	213.1
205	8-6	241	1.2	277	B242	315	7C10	353	B12
206	19	242	1D9D5	278	133/192	316	C4	354	B13
207	2-19	243	1D2F11	279	489.1	317	MF46.1	355	C13
208	8-22	244	2D9E7	280	5B3	318	6D5	356	M89
209	4-20	245	4B4C4	281	B10	319	B33	357	B21
210	6-19	246	5G7D8	282	B2	320	polyclonal	358	B23
211	7C3	247	NT2/4D5.24	283	C6	321	2H1B	359	B24
212	7F11	248	L-anti-Tat	284	MF49.1	322	697-D	360	B25
213	8E5	249	2D9D5	285	T1.1	323	6C4/S	361	B3
214	MAb 35		Rev	286	T7.1	324	C108G	362	B26
215	12	250	4G9	287	T9	325	10/76b	363	B29
216	13	251	Ab2	288	GV4D3	326	11/41e	364	B36
217	14	252	10.1	289	B27	327	11/4b	365	110.E
218	16	253	3H6	290	B9	328	RSD-33	366	110.C

HIV Antibodies

No.	Name	No.	Name	No.	Name	No.	Name	No.	Name
367	IIIB-V3-26	405	391/95-D	443	IIIB-13 V3	481	5025A	519	1794
368	IIIB-V3-21	406	Aw	444	IIIB-34 V3	482	N70-1.9b	520	1804
369	polyclonal	407	Bw	445	A47/B1	483	902	521	1807
370	polyclonal	408	DO142-10	446	D59/A2	484	694/98-D	522	1808
371	polyclonal	409	Dv	447	G44/H7	485	9205	523	polyclonal
372	polyclonal	410	Fv	448	μ 5.5	486	110.I	524	polyclonal
373	MO97/V3	411	Gv	449	loop 2	487	anti-HIV-2 polyclonal	525	CRA1
374	polyclonal	412	Hv	450	268-D	488	IIIB-V3-01	526	M91
375	55/11	413	50.1	451	386-D	489	D/6D1	527	9201
376	8/38c	414	polyclonal	452	5042A	490	4D7/4	528	1C1
377	8/64b	415	BAT123	453	5042B	491	36.1	529	3F5
378	polyclonal	416	838-D	454	418-D	492	C12	530	5F4/1
379	polyclonal	417	1006-15D	455	5021	493	110.D	531	660-178
380	9284	418	782-D	456	5025B	494	B32	532	9301
381	polyclonal	419	908-D	457	5042	495	B15	533	B221
382	polyclonal	420	1027-15D	458	110.3	496	B34	534	8C6/1
383	polyclonal	421	F19.26-4	459	110.4	497	polyclonal	535	H11
384	MAG 109	422	F19.48-3	460	110.5	498	7F11	536	W2
385	MAG 49	423	F19.57-11	461	58.2	499	5C2E5	537	1331A
386	MAG 53	424	M77	462	537-D	500	G3-211	538	M38
387	MAG 56	425	SP.BAL114	463	5020	501	G3-537	539	Chim 1
388	1324-E	426	SP.SF2:104	464	5023A	502	polyclonal	540	polyclonal
389	polyclonal	427	polyclonal	465	110.6	503	1795	541	110.1
390	MO99/V3	428	19b	466	polyclonal	504	G3-299	542	42F
391	C311E	429	4G10	467	10/36e	505	G3-42	543	43F
392	907	430	5F7	468	10/54	506	G3-508	544	RV110026
393	924	431	G3-523	469	11/85b	507	G3-519	545	105-306
394	polyclonal	432	MN215	470	polyclonal	508	G3-536	546	GV1G2
395	polyclonal	433	Nea 9301	471	0.5 β	509	ICR38.1a	547	750-D
396	10F10	434	4117C	472	C β 1	510	ICR38.8f	548	450-D
397	2C4	435	419-D	473	NM-01	511	MO86/C3	549	670-D
398	412-D	436	453-D	474	1026	512	13H8	550	polyclonal
399	polyclonal	437	504-D	475	1034	513	G45-60	551	722-D
400	CGP 47 439	438	83.1	476	59.1	514	polyclonal	552	polyclonal
401	178.1	439	5023B	477	polyclonal	515	1662	553	1131-A
402	257-D	440	F58/D1	478	10E3	516	1663	554	858-D
403	311-11-D	441	P1/D12	479	polyclonal	517	1664	555	989-D
404	41148D	442	P4/D10	480	N11-20	518	1697	556	1A1

No.	Name	No.	Name	No.	Name	No.	Name	No.	Name
557	24G3	595	240-D	633	ND-15G1	671	B8	709	15e
558	25C2	596	F240	634	2F5	672	1577	710	205-43-1
559	5F3	597	D49	635	polyclonal	673	polyclonal	711	205-46-9
560	α	598	D61	636	polyclonal	674	DZ	712	21h
561	PC5009	599	T32	637	polyclonal	675	IVI-4G6	713	2G6
562	polyclonal α	600	T34	638	polyclonal	676	polyclonal	714	428
563	polyclonal	601	115.8	639	polyclonal	677	polyclonal	715	448-D
564	1F11	602	M-1	640	TH-Ab1	678	polyclonal	716	48-16
565	1H5	603	M-11	641	5B2	679	101-342	717	50-61A
566	3D9	604	M-13	642	9G11	680	101-451	718	5145A
567	4B3	605	M-2	643	4E10	681	120-1	719	558-D
568	4D4	606	M-22	644	Z13	682	23A	720	559/64-D
569	4G2	607	M-24	645	B30	683	D7324	721	588-D
570	polyclonal	608	M-25	646	polyclonal	684	212A	722	654-D
571	polyclonal	609	M-28	647	41S-2	685	522-149	723	729-D
572	polyclonal	610	M-29	648	447-52D	686	L19	724	830D
573	2A2/26	611	M-36	649	C8	687	M90	725	9CL
574	50-69	612	M-4	650	B31	688	MAG 104	726	anti-CD4BS summary
575	9-11	613	M-6	651	B33	689	MAG 45	727	b11
576	98-43	614	polyclonal α	652	1576	690	MAG 95	728	b13
577	41-1	615	1B8.env	653	1578	691	MAG 97	729	b14
578	41.4	616	polyclonal	654	1579	692	p7	730	b3
579	Fab A1	617	clone 3	655	1583	693	L100	731	b6
580	Fab A4	618	4	656	1899	694	2/11c	732	BM12
581	Fab M12B	619	41-6	657	1907	695	C11	733	D20
582	Fab M26B	620	41-7	658	1908	696	L81	734	D21
583	Fab M8B	621	68.1	659	1909	697	2F19C	735	D24
584	Fab T2	622	68.11	660	41-1	698	B2C	736	D25
585	86	623	75	661	41-2	699	1024	737	D28
586	polyclonal	624	105-732	662	41-3	700	10/46c	738	D35
587	V10-9	625	3D6	663	ED6	701	1027-30-D	739	D39
588	polyclonal	626	F172-D8	664	LA9	702	1125H	740	D42
589	polyclonal	627	D50	665	1575	703	120-1B1	741	D52
590	polyclonal	628	5-21-3	666	88-158/02	704	1202-D	742	D53
591	2F11	629	120-16	667	88-158/022	705	1331E	743	D60
592	246-D	630	98-6	668	88-158/079	706	1570	744	DA48
593	9G5A	631	167-7	669	polyclonal	707	1595	745	DO8i
594	181-D	632	167-D	670	polyclonal	708	1599	746	F105

HIV Antibodies

No.	Name	No.	Name	No.	Name	No.	Name	No.	Name
747	F91	785	48d	823	AD3	861	anti-gp120/V3	899	B6
748	GP13	786	A32	824	AD3	862	D47	900	BAT267
749	GP44	787	1367	825	ID6	863	F5.5	901	BAT401
750	GP68	788	126-6	826	ID6	864	G3-1472	902	BAT509
751	HF1.7	789	1281	827	31A1	865	K24	903	C31
752	HT5	790	1342	828	39A64	866	M096/V3	904	D1
753	HT6	791	1379	829	39B86	867	polyclonal	905	D12
754	HT7	792	Fab D11	830	9303	868	polyclonal	906	D16
755	ICR 39.13g	793	Fab D5	831	polyclonal	869	TH1	907	D4
756	ICR 39.3b	794	Fab G1	832	11/68b	870	11/75a/21/41	908	D43
757	IgG1b12	795	Fab M10	833	62c	871	41.1	909	F223
758	IgGCD4	796	Fab M12	834	CRA-6	872	55/45a/11	910	F285
759	L28	797	Fab M15	835	L15	873	1108	911	F7
760	L33	798	Fab S10	836	T52	874	MO101/V3,C4	912	Fab A12
761	L41	799	Fab S6	837	T54	875	D27	913	Fab A2
762	L42	800	Fab S8	838	1088	876	D56	914	Fab L9
763	L52	801	Fab S9	839	110-B	877	polyclonal	915	G12
764	L72	802	Fab T3	840	1357	878	2G12	916	G2
765	M12	803	Md-1	841	1361	879	MO101/V3,C4	917	H2
766	M13	804	Fab A9	842	1393A	880	MO101/V3,C4	918	H8
767	M6	805	Fab G15	843	66a	881		919	HBW4
768	MAG 116	806	Fab G5	844	66c	882	102-135	920	K14
769	MAG 12B	807	Fab L1	845	684-238	883	1025	921	M25
770	MAG 29B	808	Fab L11	846	830A	884	105-134	922	MAG 6B
771	MAG 3B	809	Fab L2	847	CRA-3	885	10E9	923	MO28
772	MAG 55	810	Chessie 8	848	CRA-4	886	126-50	924	MO30
773	MAG 72	811	T22	849	L17	887	12H2	925	MO43
774	MAG 86	812	8F101	850	SC258	888	13.10	926	multiple Fabs
775	MAG 96	813	8F102	851	L25	889	1B1	927	multiple MAbs
776	MTW61D	814	CG-10	852	L39	890	1F7	928	multiple MAbs
777	S1-1	815	CG-25	853	L40	891	31710B	929	multiple MAbs
778	T13	816	CG-4	854	L78	892	3D5	930	N2-4
779	T49	817	CG-76	855	110.J	893	3H6	931	N70-2.3a
780	T56	818	CG-9	856	1334-D	894	6E10	932	P43110
781	TH9	819	NC-1	857	55/68b	895	7-1054	933	P5-3
782	D33	820	105-518	858	5G11	896	A9	934	polyclonal
783		821	2A2	859	9305	897	B4	935	polyclonal
784	17b	822	AC4	860	AG1121	898	B5	936	polyclonal

No.	Name	No.	Name	No.	Name
937	polyclonal	975	polyclonal	1012	AE6
938	polyclonal	976	T20	1013	AG11
939	polyclonal	977	T27	1014	EH1
940	polyclonal	978	T3	1015	6.1
941	polyclonal	979	T30	1016	AE6
942	polyclonal	980	T4	1017	NF2B2
943	polyclonal		Nef	1018	NF3A3
944	polyclonal	981	4H4	1019	NF8B4
945	polyclonal	982	polyclonal		
946	polyclonal	983	13/042		
947	polyclonal	984	13/035		
948	polyclonal	985	AM5C6		
949	polyclonal	986	AM5C6		
950	polyclonal	987	25/03		
951	polyclonal	988	26/76		
952	polyclonal	989	3F2		
953	polyclonal	990	3D12		
954	polyclonal	991	polyclonal		
955	polyclonal	992	3G12		
956	polyclonal	993	13/058		
957	polyclonal	994	26/028		
958	polyclonal	995	2E3		
959	polyclonal	996	polyclonal		
960	polyclonal	997	F14.11		
961	polyclonal	998	31/03		
962	polyclonal	999	polyclonal		
963	polyclonal	1000	polyclonal		
964	polyclonal	1001	F1		
965	polyclonal	1002	2F2		
966	polyclonal	1003	E9		
967	polyclonal	1004	3E6		
968	polyclonal	1005	2A3		
969	polyclonal	1006	2E4		
970	polyclonal	1007	3A2		
971	polyclonal	1008	2H12		
972	polyclonal	1009	NF1A1		
973	polyclonal	1010	polyclonal		
974	polyclonal	1011	E7		

Part IV-B: Table of HIV Monoclonal Antibodies and Polyclonal Antibody Responses

All HIV MAbs and polyclonal Abs that bind to linear epitopes 30 amino acids or less in length arranged by protein position. Abs that bind to conformational epitopes are listed at the end of each protein section. The table entries have been sorted in a nested way, first by protein, then by HXB2 start location, then by HXB2 end location, then by antibody type, and finally by antibody name. Any antibodies whose HXB2 location is unknown will appear at the end of the listing of the protein in which they are located.

Table of HIV MAbs

Table 1: p17

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
1 L14.17	p17(11–25)	p17(11–25 BRU)	GELDRWEKIRLRPGG	no	Vaccine	murine(IgG)
	<p>Vaccine: <i>Vector/type:</i> viral lysate <i>Strain:</i> BRU <i>HIV component:</i> virus References: [Tatsumi (1990), Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p>					
2 polyclonal	p17(11–25)	p17(11–25 LAI)	GELDRWEKIRLRPGG	no	Vaccine	mouse()
	<p>Vaccine: <i>Vector/type:</i> recombinant protein, virus-like particle <i>Strain:</i> LAI <i>HIV component:</i> p24, p17, p55 <i>Stimulatory Agents:</i> Freund's adjuvant References: [Truong (1997)]</p> <ul style="list-style-type: none"> • An ELISA assay was used to study a panel of Gag peptides – mature p24 CA epitopes mapped to residues 176–192, 201–218, 233–253, 285–304, and were recognized by antibodies elicited by rp24CA – one p17MA epitope, residues 11–25, and one p24CA epitope, residues 176–192, were recognized by antibodies raised against anti-p55 virus-like particles, suggesting different antigenic properties for p24CA and p17MA antibodies depending on whether they are produced against the mature soluble protein or the immature assembled form of the Gag protein [Truong (1997)] 					
3 32/5.8.42	p17(dis 12–19 + 100–105)	p17(dis 12–19 IIIB)	ELDRWEKI + ALDKIE		Vaccine	murine(IgG)
	<p>Vaccine: <i>Vector/type:</i> viral lysate References: [Papsidero (1989)]</p> <ul style="list-style-type: none"> • 32/5.8.42: Binds to two discontinuous regions, positions 12–19 and 100–105, peptides ELDRWEKI and ALDKIE – inhibited infectivity of cell free virus [Papsidero (1989)] 					
4 HyHIV-1	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY		Vaccine	murine(IgG1)
	<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> p17 References: [Liu (1995), Ota & Ueda(1998)]</p> <ul style="list-style-type: none"> • HyHIV-1: This paper compares the results of affinity constant (K_a) measurements of anti-p17 MAbs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MAbs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota & Ueda(1998)] 					
5 HyHIV-2	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY	no	Vaccine	murine(IgG1)
	<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> p17 References: [Liu (1995), Ota & Ueda(1998)]</p> <ul style="list-style-type: none"> • HyHIV-2: This paper compares the results of affinity constant (K_a) measurements of anti-p17 MAbs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MAbs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota & Ueda(1998)] 					

Table of HIV MABs

6	HyHIV-3	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY	no	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17			
		References: [Liu (1995), Ota & Ueda(1998)]					
		<ul style="list-style-type: none"> HyHIV-3: This paper compares the results of affinity constant (K_a) measurements of anti-p17 MABs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MABs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota & Ueda(1998)] 					
7	HyHIV-4	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY?	no	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17			
		References: [Liu (1995), Ota (1998), Ota & Ueda(1998)]					
		<ul style="list-style-type: none"> HyHIV-4: epitope uncertain, based on the best estimate from JMH1 sequence– K_a is $1.8 \times 10^7 \text{ M}^{-1}$ for rec p17 – stains the surface of infected cells indicating the antigen is exposed at the cell surface [Ota (1998)] HyHIV-4: This paper compares the results of affinity constant (K_a) measurements of anti-p17 MABs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MABs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota & Ueda(1998)] 					
8	HyHIV-5	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY	no	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17			
		References: [Liu (1995), Ota & Ueda(1998)]					
		<ul style="list-style-type: none"> HyHIV-5: This paper compares the results of affinity constant (K_a) measurements of anti-p17 MABs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MABs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota & Ueda(1998)] 					
9	HyHIV-6	p17(12–29)	p17(12–29 JMH1)	ELDKWEKIRLRPGGKTLY	no	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17			
		References: [Liu (1995), Ota & Ueda(1998)]					
		<ul style="list-style-type: none"> HyHIV-6: This paper compares the results of affinity constant (K_a) measurements of anti-p17 MABs using double Ab methods versus the faster, isotope-free BIAcore system, and results were found to be similar for HyHIV-(1–6), six MABs which all bind to the first alpha helix of p17, a functional domain for both membrane binding and nuclear localization [Ota & Ueda(1998)] 					
10	32/1.24.89	p17(17–22)	p17(17–22 IIIB)	EKIRLR	L	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> viral lysate					
		References: [Papsidero (1989)]					
		<ul style="list-style-type: none"> 32/1.24.89: Inhibited infectivity of cell free virus [Papsidero (1989)] 					

Table of HIV MAbs

11	3B10	p17(19–38)	p17(19–38 SIVmac)	IRLPGGKKKYMLKHVVWAA	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> inactivated virus		<i>Strain:</i> AGM TYO-7	<i>HIV component:</i> virus	
		References: [Otteken (1992)]				
		<ul style="list-style-type: none"> • 3B10: Recognized an epitope present on HIV-2/SIVmac (MAC251/32H) , SIVagm, HIV-1, and SIVmnd, demonstrating that the matrix protein of all nine HIV and SIV isolates tested in this study expresses at least one conserved immunogenic epitope recognized serologically [Otteken (1992)] 				
12	3E11	p17(19–38)	p17(19–38 SIVmac)	IRLPGGKKKYMLKHVVWAA	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> inactivated virus		<i>Strain:</i> AGM TYO-7	<i>HIV component:</i> virus	
		References: [Otteken (1992), Nilsen (1996)]				
		<ul style="list-style-type: none"> • 3E11: There is another MAb with this ID that recognizes integrase [Nilsen (1996)] • 3E11: Recognized an epitope present on HIV-2/SIVmac (MAC251/32H), SIVagm, HIV-1, and SIVmnd, demonstrating that the matrix protein of all nine HIV and SIV isolates tested in this study expresses at least one highly conserved immunogenic epitope [Otteken (1992)] 				
13	8H10	p17(30–52)	p17(30–52 JMH1)	KLKHIVWASRELERFAVNPGL- LE	Vaccine	murine(IgM)
	Vaccine:	<i>Vector/type:</i> peptide	<i>Strain:</i> JMH-1	<i>HIV component:</i> p17	<i>Stimulatory Agents:</i> BSA	
		References: [Ota (1999), Ota & Ueda(1999)]				
		<ul style="list-style-type: none"> • 8H10: This p17 MAb also can bind to the V3 loop [Ota (1999)] • 8H10: Inhibits viral replication of the HIV-1 infected MT-4 cells by decreasing p17 protein levels in the infected cells, and the effect of growing the 8H10 hybridoma in co-culture with HIV-1 infected MT-4 cells was studied [Ota & Ueda(1999)] 				
14	HyHIV-21	p17(30–52)	p17(30–52 JMH1)	KLKHIIWASRELERFAVNPGLLE	no Vaccine	murine(IgG2a)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17		
		References: [Liu (1995), Ota (1998)]				
		<ul style="list-style-type: none"> • HyHIV-21: epitope uncertain, based on the best estimate from JMH1 sequence – K_a is $3.6 \times 10^6 \text{ M}^{-1}$ for rec p17 – stains the surface of infected cells indicating the antigen is exposed at the cell surface –inhibited growth of HIV-1 JMH1 in MT-4 cells when added 24 hours after the initial culture [Ota (1998)] 				
15	B4f8	p17(51–65)	p17(51–65)	LETSEGCRQILGQLQ	no Vaccine	rat(IgG2a)
	Vaccine:	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		References: [Shang (1991)]				
		<ul style="list-style-type: none"> • B4f8: Did not bind live infected cells, only cells that had been made permeable with acetone [Shang (1991)] 				

Table of HIV MAbs

16	HyHIV-22	p17(52–83)	p17(53–87 JMH1)	ETSEGCRQILGQRQPSLQTGS- EELRSLYNTIH?	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> p17		
		References: [Liu (1995), Ota (1998)]				
		<ul style="list-style-type: none"> HyHIV-22: stains the surface of infected cells indicating the antigen is exposed at the cell surface – K_a is $2.3 \times 10^5 \text{ M}^{-1}$ for rec p17 [Ota (1998)] 				
17	12H-D3b3	p17(62–78)	p17(62–78)	GQLQPSLQTGSEELRSL	no Vaccine	rat(IgG2a)
	Vaccine:	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		References: [Shang (1991)]				
		<ul style="list-style-type: none"> 12H-D3b3: Did not bind live infected cells, only cells that had been made permeable with acetone [Shang (1991)] 				
18	12G-A8g2	p17(86–115)	p17(86–115)	YCVHQRIEIKDTKEALDKIEE- EQNKSKKKA	no Vaccine	rat(IgG2a)
	Vaccine:	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		References: [Shang (1991)]				
		<ul style="list-style-type: none"> 12G-A8g2: Bound to 30-mer, but not to internal peptides – did not bind live infected cells – antigenic domain known as HPG30 [Shang (1991)] 				
19	12G-D7h11	p17(86–115)	p17(86–115)	YCVHQRIEIKDTKEALDKIEE- EQNKSKKKA	no Vaccine	rat(IgG2a)
	Vaccine:	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		References: [Shang (1991)]				
		<ul style="list-style-type: none"> 12G-D7h11: Bound to 30-mer, but not to internal peptides – did not bind live infected cells – antigenic domain known as HPG30 [Shang (1991)] 				
20	12G-H1c7	p17(86–115)	p17(86–115)	YCVHQRIEIKDTKEALDKIEE- EQNKSKKKA	no Vaccine	rat(IgG)
	Vaccine:	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		References: [Shang (1991)]				
		<ul style="list-style-type: none"> 12G-H1c7: Bound to 30-mer, but not to internal peptides – did not bind live infected cells – antigenic domain known as HPG30 [Shang (1991)] 				
21	12I-D12g2	p17(86–115)	p17(86–115)	YCVHQRIEIKDTKEALDKIEE- EQNKSKKKA	no Vaccine	rat(IgG2a)
	Vaccine:	<i>Vector/type:</i> infected-cell lysate		<i>Strain:</i> IIIB	<i>HIV component:</i> virus	
		References: [Shang (1991)]				

Table of HIV MAbs

- 12I-D12g2: Bound to 30-mer, but not to internal peptides – did not bind live infected cells – antigenic domain known as HPG30 [Shang (1991)]

22	polyclonal	p17(86–115)	p17(86–115)	YSVHQRIDVKDTKEALEKIEE- EQNKSKKKA	L Vaccine	murine(IgA)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> Multiple B-clade <i>HIV component:</i> V3 loop, CD4BS, HPG30 <i>Stimulatory</i> <i>Agents:</i> cholera toxin adjuvant</p> <p>References: [Bukawa (1995)]</p> <ul style="list-style-type: none"> • Polyclonal secretory IgA antibody raised by oral mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to the V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa (1995)] 						
23	HyHIV-15	p17(87–115)	p17(87–115 JMH1)	SVHQRIDVKDTKEALEKIEE- EQNKSKKKA?	L Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> p17</p> <p>References: [Liu (1995), Ota (1998)]</p> <ul style="list-style-type: none"> • HyHIV-15: epitope uncertain, based on the best estimate from JMH1 sequence – K_a is $1.4 \times 10^7 \text{ M}^{-1}$ for rec p17 – stains the surface of infected cells indicating the antigen is exposed at the cell surface – inhibited growth of HIV-1 JMH1 in MT-4 cells when added 24 hours after the initial culture [Ota (1998)] 						
24	32/5.8.42	p17(dis 12–19 + 100–105)	p17(dis IIIB)	ELDRWEKI + ALDKIE	no Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> viral lysate <i>HIV component:</i> virus</p> <p>References: [Papsidero (1989)]</p> <ul style="list-style-type: none"> • 32/5.8.42: Inhibited infectivity of cell free virus – bound to two peptides, ELDRWEKI and ALDKIE, at positions 12–19 + 100–105 [Papsidero (1989)] 						
25	11H9	p17(101–115)	p17(101–115 SF2)	LEKIEEEQNKSKKKA?	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus</p> <p>Donor: R. B. Ferns and R. S. Tedder</p> <p>References: [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> • 11H9: Reactive against p18 and p55 [Ferns (1987)] • 11H9: UK Medical Research Council AIDS reagent: ARP344 						
26	3-H-7 (3H7)	p17(113–122)	p17(113–122 BH10)	KKAQQAAADT	L Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> IIIB</p> <p>References: [Niedrig (1989), Robert-Hebmann (1992b), Robert-Hebmann (1992a), Levin (1997)]</p> <ul style="list-style-type: none"> • 3-H-7: No cross-reactivity with HIV-2 ROD or SIV MAC by immunoblot [Niedrig (1989)] 						

- 3-H-7: Called 3H7 – using a bicistronic vector, an intracellular Fab intrabody, 3H7, can inhibit HIV-1 infection when expressed in the cytoplasm of dividing CD4+ T cells – HXBIIIIB and SI primary isolate virions from 3H7 expressing cells were far less infectious – 3H7 intrabody acts both at the stage of nuclear import and virus particle assembly [Levin (1997)]

27	C5126	p17(113–122)	p17(113–122 HXB2)	KKAQQAADT	no Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> viral lysate <i>HIV component:</i> virus</p> <p>References: [Hinkula (1990)]</p> <ul style="list-style-type: none"> • C5126: Defined epitope by peptide blocking of binding to native protein – WB reactive with p53 and p17 [Hinkula (1990)] 						
28	1D9	p17(119–132)	p17(121–134 SF2)	AAGTGNSSQVSQNY	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus</p> <p>Donor: R. B. Ferns and R. S. Tedder</p> <p>References: [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> • 1D9: Reactive against p18, but not p55 [Ferns (1987)] • 1D9: UK Medical Research Council AIDS reagent: ARP316 						
29	4C9	p17(119–132)	p18(121–134 SF2)	AAGTGNSSQVSQNY	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus</p> <p>Donor: R. B. Ferns and R. S. Tedder</p> <p>References: [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> • 4C9: Reactive against p18, but not p55 [Ferns (1987)] • 4C9: UK Medical Research Council AIDS reagent: ARP342 						
30	4H2B1	p17(119–132)	p17(121–134 SF2)	AAGTGNSSQVSQNY		murine(IgG1)
<p>Donor: R. B. Ferns and R. S. Tedder</p> <p>References: [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> • 4H2B1: Reactive against p18 and p55 of multiple isolates [Ferns (1987)] • 4H2B1: UK Medical Research Council AIDS reagent: ARP315 						
31	9G5	p17(119–132)	p17(121–134 SF2)	AAGTGNSSQVSQNY	Vaccine	murine(IgM)
<p>Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus</p> <p>Donor: R. B. Ferns and R. S. Tedder</p> <p>References: [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> • 9G5: Reactive against p18, but not p55 [Ferns (1987)] • 9G5: UK Medical Research Council AIDS reagent: ARP343 						
32	15–21	p17(121–132)	p17(121–132 BRU)	DTGHSSQVSQNY	no Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> BRU</p> <p>References: [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p>						

Table of HIV MAbs

33	31-11	p17(121-132)	p17(121-132 BRU)	DTGHSSQVSQNY	no Vaccine	murine(IgG)
	Vaccine:	<i>Strain:</i> BRU	References: [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]			
34	sc-FV p17	p17(121-132)	p17(121-132 BRU)	DTGHSSQVSQNY	Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Strain:</i> BRU	Ab type: C-term Donor: Paul Zhou, NIH, Bethesda, MD, USA			
		References: [Robert-Hebmann (1992a), Tewari (1998)]				
		<ul style="list-style-type: none"> • A single chain Ab (sc-FV) was made from an anti-p17 MAb, and intracellular binding of sc-FV resulted in inhibition of viral replication that was more pronounced when the sc-FV was expressed in the cytoplasm instead of the nucleus [Tewari (1998)] 				

Table 2: p24

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
35 3A6	p24(1–17)	p24(122–149 BH10)	TGHSSQVSQNYPIVQNIQGQM-VHQAISP	no	HIV-1 infection	human(IgG1 κ)
						<p>References: [Buchacher (1992), Buchacher (1994)]</p> <ul style="list-style-type: none"> • 3A6: The reactive peptide spans the p17/p24 border of gag [Buchacher (1994)] • 3A6: Human MAbs against were generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]
36 111/182	p24(1–20)	p24(134–153 IIIB)	PIVQNIQGQMVHQAISPRTL	no	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> β -galactosidase fusion protein	<i>Strain:</i> IIIB		<i>HIV component:</i> p24	
						<p>References: [Niedrig (1991)]</p> <ul style="list-style-type: none"> • 111/182: Test of specific evidence of cross-reactivity between HIV-1, HIV-2 and SIV MAC [Niedrig (1991)]
37 112/021	p24(1–20)	p24(134–153 IIIB)	PIVQNIQGQMVHQAISPRTL	no	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> β -galactosidase fusion protein	<i>Strain:</i> IIIB		<i>HIV component:</i> p24	
						<p>References: [Niedrig (1991)]</p> <ul style="list-style-type: none"> • 112/021: Test of specific evidence of cross-reactivity between HIV-1, HIV-2 and SIV MAC [Niedrig (1991)]
38 112/047	p24(1–20)	p24(134–153 IIIB)	PIVQNIQGQMVHQAISPRTL	no	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> β -galactosidase fusion protein	<i>Strain:</i> IIIB		<i>HIV component:</i> p24	
						<p>References: [Niedrig (1991)]</p> <ul style="list-style-type: none"> • 112/047: Test of specific evidence of cross-reactivity between HIV-1, HIV-2 and SIV MAC [Niedrig (1991)]
39 ID8F6	p24(11–25)	p24(143–157 BRU)	VHQAISPRTLNAWVK	no	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> inactivated virus	<i>Strain:</i> CBL-1		<i>HIV component:</i> virus	
						<p>Donor: R. B. Ferns and R. S. Tedder</p> <p>References: [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> • ID8F6: Reacted with both p55 and p24 – showed less than 75% homologous inhibition [Ferns (1987)] • ID8F6: UK Medical Research Council AIDS reagent: ARP348
40 F5-2	p24(14–23)	p24(14–23 HXB2)	AISPRTLNAW	no		murine()
						<p>References: [Kusk (1988), Kusk (1992)]</p> <ul style="list-style-type: none"> • F5-2: In HIV-1+ individuals, antibody to AISPRTLNAW is associated with increased rates of CD4 T-cell decline [Kusk (1988), Kusk (1992)]

Table of HIV MAbs

41	CB-13/5 (CB-mab- p24/13–15)	p24(21–25)	p24(152–156)	NAWVK	no		murine(IgG1 κ)
<p>References: [Grunow (1990), Franke (1992), Kuttner (1992), Glaser & Hausdorf(1996)]</p> <ul style="list-style-type: none"> • CB-13/5: It is not clear whether the MAbs CD-13/5 and CB-mab-p24/13–15 are the same, but from the shared references in the primary articles they seem to be (database note) • CB-13/5: Called CB-mab-p24/13–15 – the VDJ H and VJ L regions of CB-mab-p24/13–15 were sequenced [Kuttner (1992)] • CB-13/5: Inhibits spread of HIV-1 in cell cultures [Franke (1992)] • CB-13/5: Epitope described as VHQAISPRTLNAWVK – binding not affected by bound MAb CB-4/1 [Glaser & Hausdorf(1996)] 							
42	polyclonal	p24(44–60)	p24(176–192 LAI)	SEGATPQDLNNTMLNTVG	no	Vaccine	mouse(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein, virus-like particle <i>Strain:</i> LAI <i>HIV component:</i> p24, p17, p55 <i>Stimulatory Agents:</i> Freund’s adjuvant</p> <p>References: [Truong (1997)]</p> <ul style="list-style-type: none"> • An ELISA assay was used to study a panel of Gag peptides – mature p24 CA epitopes mapped to residues 176–192, 201–218, 233–253, 285–304, and were recognized by antibodies elicited by rp24CA – one p17MA epitope, residues 11–25, and one p24CA epitope, residues 176–192, were recognized by antibodies raised against anti-p55 virus-like particles, suggesting a different antigenic properties for p24CA and p17MA antibodies depending on whether they are produced against the mature soluble protein or the immature assembled form of the gag proteins [Truong (1997)] 							
43	3D3	p24(45–50)	p24(177–182 LAI)	EGATPQ		Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus</p> <p>Donor: R. B. Ferns and R. S. Tedder</p> <p>References: [Ferns (1987), Ferns (1989)]</p> <ul style="list-style-type: none"> • 3D3: Most broadly reactive of all the antibodies in this study[Ferns (1987)] • 3D3: UK Medical Research Council AIDS reagent: ARP314 							
44	CD-4/1 (CB- 4/1/1/F6)	p24(46–56)	p24(182–197)	GATPQDLNNTML	no	Vaccine	murine(IgG2a κ)
<p>Vaccine: <i>Vector/type:</i> β-galactosidase fusion protein <i>HIV component:</i> p24</p> <p>References: [Grunow (1990), Franke (1992), Hohne (1993), Glaser & Hausdorf(1996), Ehrhard (1996)]</p> <ul style="list-style-type: none"> • CD-4/1: Inhibits spread of HIV-1 in cell cultures [Franke (1992)] • CD-4/1: Affinity of CB-4/1 to native p24 is lower than to peptide or denatured p24 – proposed that the peptide binds in a loop conformation [Hohne (1993)] • CD-4/1: Unusual p24-MAb binding kinetics, with biphasic association – probably due to conformational changes in p24, not to p24 dimerization [Glaser & Hausdorf(1996)] • CD-4/1: Modification of p24 lysine residues by maleic anhydrid increased the affinity of CD-4/1, presumably due to conformational changes exposing a cryptic epitope [Ehrhard (1996)] 							

Table of HIV MAbs

45	15F8C7	p24(47–56)	p24(183–197)	ATPQDLNTML	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> purified HIV-1</p> <p>References: [Janvier (1990), Janvier1992]</p> <ul style="list-style-type: none"> • 15F8C7: Remapped to aa209–217 through Pepscan method – cross-reacts with HIV-2 [Janvier (1990)] – maps to aa203–217 through EIA pentadecapeptide [Janvier1992] 							
46	111/052	p24(51–60)	p24(183–192 IIIB)	DLNTMLNTVG	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> β-galactosidase fusion protein <i>Strain:</i> IIIB <i>HIV component:</i> p24</p> <p>References: [Niedrig (1991)]</p> <ul style="list-style-type: none"> • 111/052: Weak cross-reaction with HIV-2 on WB, otherwise not cross-reactive with HIV-2 or SIV MAC [Niedrig (1991)] 							
47	polyclonal	p24(51–82)	Gag(183–214 LAI)	DLNTMLNTVGGHQAAMQML-KETINEEAAEWDR	no	Vaccine	human(IgG)
<p>Vaccine: <i>Vector/type:</i> lipopeptide <i>Strain:</i> LAI <i>HIV component:</i> p24 <i>Stimulatory Agents:</i> QS21</p> <p>References: [Pialoux (2001)]</p> <ul style="list-style-type: none"> • 28 subjects were vaccinated with six HIV-1 peptides that were selected to be particularly rich in CTL epitopes, presented in lipopeptides with or without adjuvant QS21 – HIV-specific Ab responses were detected in 25/28 (89%), proliferative in 19/28 (79%), and CTL in 13/24 (54%) of testable volunteers – only 4/28 had Ab responses to this peptide, G1, 4/28 had proliferative responses, and no patient had a CTL response [Pialoux (2001)] 							
48	91–5	p24(64–75)	p24(196–207)	AAMQMLKETINE	no	HIV-1 infection	human(IgG1λ)
<p>References: [Gorny (1989), Tyler (1990), Robinson (1990b), Gorny (1998)]</p> <ul style="list-style-type: none"> • 91–5: Synthesized by immortalization of peripheral blood cells with Epstein-Barr virus [Gorny (1989)] • 91–5: Did not enhance HIV-1 IIIB infection [Robinson (1990b)] • 91–5: NIH AIDS Research and Reference Reagent Program: 1238 							
49	1109/01	p24(69–86)	p24(201–218 BRU)	LKETINEEAAEWDRVHPV	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> IIIB <i>HIV component:</i> virus</p> <p>References: [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p>							
50	14D4E11	p24(69–86)	p24(201–218 BRU)	LKETINEEAAEWDRVHPV	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> purified HIV-1</p> <p>References: [Janvier (1990), Robert-Hebmann (1992b), Robert-Hebmann (1992a), Janvier1992]</p> <ul style="list-style-type: none"> • 14D4E11: Mapped to aa209–217 through Pepscan method (original paper, AAEWDRVHP) – cross-reacts with HIV-2 [Janvier (1990)] and to aa203–217 through EIA pentadecapeptide [Janvier1992] 							

Table of HIV MAbs

51	1G5C8	p24(69–86)	p24(201–218 BRU)	LKETINEEAAEWDRVHPV	no Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> p24 References: [Janvier (1990), Robert-Hebmann (1992b), Robert-Hebmann (1992a), Janvier1992] • 1G5C8: Mapped to aa209–217 through Pepscan method (original paper, AAEWDRVHP) [Janvier (1990)] and to aa203–217 through EIA pentadecapeptide [Janvier1992]</p>						
52	47–2	p24(69–86)	p24(201–218 BRU)	LKETINEEAAEWDRVHPV	no Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> BRU References: [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p>						
53	714/01	p24(69–86)	p24(201–218 BRU)	LKETINEEAAEWDRVHPV	no Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> IIIB <i>HIV component:</i> virus References: [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p>						
54	polyclonal	p24(69–86)	p24(201–218 LAI)	LKETINEEAAEWDRVHPV	no Vaccine	mouse()
<p>Vaccine: <i>Vector/type:</i> recombinant protein, virus-like particle <i>Strain:</i> LAI <i>HIV component:</i> p24, p17, p55 <i>Stimulatory Agents:</i> Freund’s adjuvant References: [Truong (1997)] • An ELISA assay was used to study a panel of Gag peptides – mature p24 CA epitopes mapped to residues 176–192, 201–218, 233–253, 285–304, and were recognized by antibodies elicited by rp24CA – one p17MA epitope, residues 11–25, and one p24CA epitope, residues 176–192, were recognized by antibodies raised against anti-p55 virus-like particles, suggesting different antigenic properties for p24CA and p17MA antibodies depending on whether they are produced against the mature soluble protein or the immature assembled form of the gag proteins [Truong (1997)]</p>						
55	111/073	p24(71–81)	p24(203–213 IIIB)	ETINEEAAEWD	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> β-galactosidase fusion protein <i>Strain:</i> IIIB <i>HIV component:</i> p24 References: [Niedrig (1991)] • 111/073: cross-reactive between HIV-1, HIV-2 and SIV MAC by multiple assays [Niedrig (1991)]</p>						
56	113/038	p24(71–81)	p24(203–213 IIIB)	ETINEEAAEWD	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> β-galactosidase fusion protein <i>Strain:</i> IIIB <i>HIV component:</i> p24 References: [Niedrig (1991)] • 113/038: cross-reactive between HIV-1, HIV-2 and SIV MAC by multiple assays [Niedrig (1991)]</p>						
57	1-E-4	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> IIIB <i>HIV component:</i> virus References: [Niedrig (1989)] • 1-E-4: One of nine MAbs that bind to this peptide [Niedrig (1989)]</p>						

Table of HIV MAbs

58	1-E-9	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG)
<p>Vaccine: Strain: IIIB HIV component: virus</p> <p>References: [Niedrig (1989)]</p> <ul style="list-style-type: none"> • 1-E-9: One of nine MAbs that bind to this peptide [Niedrig (1989)] 						
59	10-E-7	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG1)
<p>Vaccine: Strain: IIIB HIV component: virus</p> <p>References: [Niedrig (1988), Niedrig (1989)]</p> <ul style="list-style-type: none"> • 10-E-7: Cross-reactive between HIV-1, HIV-2 and SIV [Niedrig (1988)] • 10-E-7: One of nine MAbs that bind to this peptide – cross-reactive with HIV-2 ROD and SIV MAC [Niedrig (1989)] 						
60	10-G-9	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG1)
<p>Vaccine: Strain: IIIB HIV component: virus</p> <p>References: [Niedrig (1988), Niedrig (1989)]</p> <ul style="list-style-type: none"> • 10-G-9: HIV-1 specific [Niedrig (1988)] • 10-G-9: One of nine MAbs that bind to this peptide [Niedrig (1989)] 						
61	11-C-5	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG1)
<p>Vaccine: Strain: IIIB HIV component: virus</p> <p>References: [Niedrig (1988), Niedrig (1989)]</p> <ul style="list-style-type: none"> • 11-C-5: HIV-1 specific [Niedrig (1988)] • 11-C-5: One of nine MAbs that bind to this peptide [Niedrig (1989)] 						
62	2-E-4	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG2a)
<p>Vaccine: Strain: IIIB HIV component: virus</p> <p>References: [Niedrig (1988), Niedrig (1989)]</p> <ul style="list-style-type: none"> • 2-E-4: Cross-reactive between HIV-1, HIV-2 and SIV by ELISA, HIV-1 and HIV-2 by WB [Niedrig (1988)] • 2-E-4: One of nine MAbs that bind to this peptide – cross-reactive with HIV-2 ROD [Niedrig (1989)] 						
63	2-H-4	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG1)
<p>Vaccine: Strain: IIIB HIV component: virus</p> <p>References: [Niedrig (1988), Niedrig (1989)]</p> <ul style="list-style-type: none"> • 2-H-4: Cross-reactive between HIV-1, HIV-2 and SIV by ELISA, HIV-1 and HIV-2 by WB [Niedrig (1988)] • 2-H-4: One of nine MAbs that bind to this peptide – cross-reactive with HIV-2 ROD [Niedrig (1989)] 						
64	8-D-2	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG2a)
<p>Vaccine: Strain: IIIB HIV component: virus</p>						

Table of HIV MAbs

<p>References: [Niedrig (1989), Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p> <ul style="list-style-type: none"> • 8-D-2: HIV-1 specific [Niedrig (1988)] • 8-D-2: One of nine MAbs that bind to this peptide [Niedrig (1989)] 						
65	8-G-9	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG)
<p>Vaccine: Strain: IIIB HIV component: virus</p> <p>References: [Niedrig (1989)]</p> <ul style="list-style-type: none"> • 8-G-9: One of nine MAbs that bind to this peptide [Niedrig (1989)] 						
66	8-H-7	p24(71–85)	p24(203–217)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG3)
<p>Vaccine: Strain: IIIB HIV component: virus</p> <p>References: [Niedrig (1988), Niedrig (1989), Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p> <ul style="list-style-type: none"> • 8-H-7: One of nine MAbs that bind to this peptide [Niedrig (1989)] 						
67	C5123	p24(71–85)	p24(203–217 HXB2)	ETINEEAAEWDRVHP	no Vaccine	murine(IgG1 κ)
<p>Vaccine: Vector/type: viral lysate HIV component: virus</p> <p>References: [Hinkula (1990)]</p> <ul style="list-style-type: none"> • C5123: Epitope defined by peptide blocking of binding to native protein – WB reactive with p53 and p24 [Hinkula (1990)] 						
68	1-B-7	p24(76–85)	p24(208–217 BH10)	EAAEWDRVHP	no Vaccine	murine(IgG1)
<p>Vaccine: Strain: IIIB</p> <p>References: [Niedrig (1988), Niedrig (1989)]</p> <ul style="list-style-type: none"> • 1-B-7: Reacts with two overlapping peptides, region of overlap is given – reacted with HIV-2 and SIV MAC [Niedrig (1989)] 						
69	3-B-7	p24(76–85)	p24(208–217 BH10)	EAAEWDRVHP	no Vaccine	murine(IgG1)
<p>Vaccine: Strain: IIIB</p> <p>References: [Niedrig (1988), Niedrig (1989)]</p> <ul style="list-style-type: none"> • 3-B-7: Reacts with two overlapping peptides, region of overlap is given – reacted with HIV-2 [Niedrig (1989)] 						
70	6-D-12	p24(76–85)	p24(208–217 BH10)	EAAEWDRVHP	no Vaccine	murine(IgG1)
<p>Vaccine: Strain: IIIB</p> <p>References: [Niedrig (1988), Niedrig (1989)]</p> <ul style="list-style-type: none"> • 6-D-12: Reacts with two overlapping peptides, region of overlap is given – reacted with HIV-2 [Niedrig (1989)] 						
71	6-E-7	p24(76–85)	p24(208–217 BH10)	EAAEWDRVHP	no Vaccine	murine(IgG1)
<p>Vaccine: Strain: IIIB</p> <p>References: [Niedrig (1988), Niedrig (1989)]</p> <ul style="list-style-type: none"> • 6-E-7: Reacts with two overlapping peptides, region of overlap is given – reacted with HIV-2 and SIV MAC [Niedrig (1989)] 						

Table of HIV MAbs

72	8-D-5	p24(76–85)	p24(208–217 BH10)	EAAEWDRVHP	no Vaccine	murine(IgG)
	Vaccine:	<i>Strain:</i> IIB				
		References: [Niedrig (1988), Niedrig (1989)]				
		<ul style="list-style-type: none"> • 8-D-5: Reacts with two overlapping peptides, region of overlap is given – bound only HIV-1 [Niedrig (1989)] 				
73	FF1	p24(76–90)	p24(208–222 HXB2)	EAAEWDRVHPVHAGP	no Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> inactivated virus				
		References: [Hinkula (1990)]				
		<ul style="list-style-type: none"> • FF1: Epitope defined by peptide blocking of binding to native protein – WB reactive with p53 and p24 [Hinkula (1990)] 				
74	113/072	p24(81–90)	p24(213–222 IIB)	DRVHPVHAGP	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> β -galactosidase fusion protein				
		<i>Strain:</i> IIB <i>HIV component:</i> p24				
		References: [Niedrig (1991)]				
		<ul style="list-style-type: none"> • 113/072: Weak cross-reaction with HIV-2 on WB, otherwise not cross-reactive with HIV-2 or SIV MAC [Niedrig (1991)] 				
75	25.3	p24(82–102)	p24(82–102)	RVHPVHAGPIAPGQMREPRGS	no	murine(IgG1 κ)
		References: [Momany (1996)]				
		<ul style="list-style-type: none"> • 25.3: Crystal structure of the CA protein bound to Fab 25.3 was solved – monomers form 7 alpha-helices arranged in a coiled-coil – Fab binds to a long antigenic peptide that separates the longest helices, with a salt bridge at CA 82 R, and interactions as far away as positions 100 and 102 [Momany (1996)] 				
76	13–102-100	p24(83–94)	p24(102–112 IIB)	HPVHAGPIAPG		murine(IgG)
		Donor: Advanced Technologies, Inc., Columbia, MD				
		References: [Parker (1996), Qian & Tomer(1998)]				
		<ul style="list-style-type: none"> • 13–102-100: Binding site (HPVHAGPIAPG) defined by epitope footprinting – first binding p24 to MAb, then allowing proteolytic cleavage to cleave unprotected residues, then performing mass spectrometry to identify protected residues of epitope [Parker (1996)] • 13–102-100: Affinity capillary electrophoresis was used to fine map this epitope, and the optimal peptide was defined as VHAGPI-APGIAP – this method uses migration time shifts to probe relative affinities of Abs – the antibody binds to the cyclophilin A binding domain [Qian & Tomer(1998)] 				
77	RL4.72.1	p24(87–101)	p24(219–233 BRU)	HAGPIAPGQMREPRG	no Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> inactivated virus <i>Strain:</i> clade D strain NDK <i>HIV component:</i> virus				
		References: [Tatsumi (1990), Robert-Hebmann (1992b), Robert-Hebmann (1992a)]				
		<ul style="list-style-type: none"> • RL4.72.1: Immunized with inactivated HIV NDK, D clade, reacts with B clade peptide [Robert-Hebmann (1992a)] 				
78	polyclonal	p24(101–121)	p24(233–253 LAI)	GSDIAGTTSTLQEIQIGWMTNL	no Vaccine	mouse()
	Vaccine:	<i>Vector/type:</i> recombinant protein, virus-like particle <i>Strain:</i> LAI <i>HIV component:</i> p24, p17, p55 <i>Stimulatory Agents:</i> Freund's adjuvant				

Table of HIV MAbs

References: [Truong (1997)]

- An ELISA assay was used to study a panel of Gag peptides – mature p24 CA epitopes mapped to residues 176–192, 201–218, 233–253, 285–304, and were recognized by antibodies elicited by rp24CA – one p17MA epitope, residues 11–25, and one p24CA epitope, residues 176–192, were recognized by antibodies raised against anti-p55 virus-like particles, suggesting a different antigenic properties for p24CA and p17MA antibodies depending on whether they are produced against the mature soluble protein or the immature assembled form of the gag proteins [Truong (1997)]

79	406/01	p24(101–121)	p24(233–253 BRU)	GSDIAGTTSTLQEIQGWMTNN	no Vaccine	murine(IgG)
	Vaccine:	<i>Strain:</i> IIIB				
		References: [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]				
80	38:9.6K (38:96K)	p24(121–130)	p24(253–262 HXB2)	NPPIPVGGEIY	no Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> recombinant protein <i>HIV component:</i> p24-p15				
		References: [Hinkula (1990)]				
		<ul style="list-style-type: none"> • 38:9.6K: Called 38:96K – epitope defined by peptide blocking of binding to native protein – WB reactive with p53 and p24 [Hinkula (1990)] • 38:9.6K: UK Medical Research Council AIDS reagent: ARP365 				
81	EB1A9	p24(121–135)	p24(253–267 LAI)	NPPIPVGGEIYKRWII	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus				
		Donor: R. B. Ferns and R. S. Tedder				
		References: [Ferns (1987), Ferns (1989)]				
		<ul style="list-style-type: none"> • EB1A9: Reacted with both p55 and p24 – showed less than 75% homologous inhibition [Ferns (1987)] • EB1A9: UK Medical Research Council AIDS reagent: ARP345 				
82	polyclonal	p24(121–152)	Gag(253–284 LAI)	NPPIPVGGEIYKRWIILGLNKI- VRMYSPTSILD	no Vaccine	human(IgG)
	Vaccine:	<i>Vector/type:</i> lipopeptide <i>Strain:</i> LAI <i>HIV component:</i> p24 <i>Stimulatory Agents:</i> QS21				
		References: [Pialoux (2001)]				
		<ul style="list-style-type: none"> • 28 subjects were vaccinated with six HIV-1 peptides that were selected to be particularly rich in CTL epitopes, presented in lipopeptides with or without adjuvant QS21 – HIV-specific Ab responses were detected in 25/28 (89%), proliferative in 19/28 (79%), and CTL in 13/24 (54%) of testable volunteers – 25/28 had Ab responses to this peptide, G2, 14/28 had proliferative responses, and CTL responses were detected [Pialoux (2001)] 				
83	30:3E5	p24(141–170)	p24(273–302 HXB2)	IVRMYSPTSILDIRQGPKPEF- RDYVDRFYK	Vaccine	murine(IgG1 λ)
	Vaccine:	<i>Vector/type:</i> recombinant protein <i>HIV component:</i> p24-p15				

Table of HIV MAbs

89	1A7	p24(152–172)	p24(152–172 SIVmac)	CVKQGPKEPFQSYVDRFYKSL	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> AGM TYO-7 <i>HIV component:</i> virus</p> <p>References: [Otteken (1992)]</p> <ul style="list-style-type: none"> • 1A7: Recognized an epitope present on HIV-2/SIVmac (MAC251/32H) and HIV-2smmH4, but not SIVagmTYO-1, HIV-1 IIIB or SIVmnd [Otteken (1992)] 						
90	1F6	p24(152–172)	p24(152–172 SIVmac)	CVKQGPKEPFQSYVDRFYKSL	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> AGM TYO-7 <i>HIV component:</i> virus</p> <p>References: [Otteken (1992)]</p> <ul style="list-style-type: none"> • 1F6: Recognized an epitope present on HIV-2/SIVmac (MAC251/32H) and HIV-2smmH4, but not SIVagmTYO-1, HIV-1 IIIB or SIVmnd [Otteken (1992)] 						
91	polyclonal	p24(153–172)	p24(285–304 LAI)	IRQGPKEPFRDYVDRFYKTL	no Vaccine	mouse()
<p>Vaccine: <i>Vector/type:</i> recombinant protein, virus-like particle <i>Strain:</i> LAI <i>HIV component:</i> p24, p17, p55 <i>Stimulatory Agents:</i> Freund's adjuvant</p> <p>References: [Truong (1997)]</p> <ul style="list-style-type: none"> • An ELISA assay was used to study a panel of Gag peptides – mature p24 CA epitopes mapped to residues 176–192, 201–218, 233–253, 285–304, and were recognized by antibodies elicited by rp24CA – one p17MA epitope, residues 11–25, and one p24CA epitope, residues 176–192, were recognized by antibodies raised against anti-p55 virus-like particles, suggesting a different antigenic properties for p24CA and p17MA antibodies depending on whether they are produced against the mature soluble protein or the immature assembled form of the gag proteins [Truong (1997)] 						
92	23A5G4	p24(153–172)	p24(285–304 IIIB)	IRQGPKEPFRDYVDRFYKTL	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> p24</p> <p>References: [Janvier (1990), Janvier1992, Janvier (1996)]</p> <ul style="list-style-type: none"> • 23A5G4: Mapped to aa209–217 through Pepsan method [Janvier (1990)] and to aa285–304 through EIA pentadecapeptide method [Janvier1992] • 23A5G4: A few sera which were able to bind the linear sequence 178–192, but not sequence 288–302 in an indirect peptide ELISA inhibited the binding of 23A5G4 to the native p24 [Janvier (1996)] 						
93	23A5G5	p24(153–172)	p24(285–304 BRU)	IRQGPKEPFRDYVDRFYKTL	no Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> p24</p> <p>References: [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]</p>						

Table of HIV MAbs

94	3D10G6	p24(153–172)	p24(285–304 IIIB)	IRQGPKEPFRDYVDRFYKTL	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> purified HIV-1				
		References: [Janvier (1990), Janvier1992]				
		<ul style="list-style-type: none"> • 3D10G6: Epitope cross-reacts with HIV-1 and HIV-2 – mapped to aa260–267 through Pepscan method [Janvier (1990)] and to aa285–304 through EIA pentadecapeptide method [Janvier1992] 				
95	F5-4	p24(153–175)	p24(153–174 HXB2)	IRQGPKEPFRDYVDRFYKTLR-AE	no	murine()
		References: [Kusk (1988), Kusk (1992)]				
		<ul style="list-style-type: none"> • F5-4: Binds to a location in the most hydrophilic region of p24 [Kusk (1988), Kusk (1992)] 				
96	MO9.42.2	p24(153–178)	p24(285–310 BRU)	IRQGPKEPFRDYVDRFYKTLR-AEQAS	no Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> virus	<i>Strain:</i> HIV2 ROD	<i>HIV component:</i> virus		
		References: [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]				
		<ul style="list-style-type: none"> • MO9.42.2: Reacts with HIV-1s, HIV-2s, and SIVs in rec protein ELISA [Robert-Hebmann (1992b)] 				
97	MO9.50.2	p24(153–178)	p24(285–310 BRU)	IRQGPKEPFRDYVDRFYKTLR-AEQAS	no Vaccine	murine(IgG)
	Vaccine:	<i>Strain:</i> HIV2 ROD				
		References: [Robert-Hebmann (1992b), Robert-Hebmann (1992a)]				
		<ul style="list-style-type: none"> • MO9.50.2: Reacts with HIV-1s, HIV-2s, and SIVs in rec protein ELISA [Robert-Hebmann (1992b)] 				
98	V10	p24(155–169)	p24(289–303 IIIB)	QGPKEPFRDYVDRFY	no virus	murine()
		References: [Matsuo (1992)]				
		<ul style="list-style-type: none"> • V10: Reacts with HIV-1 and SIV AGM analogous peptides [Matsuo (1992)] 				
99	V107	p24(155–177)	p24(289–311 IIIB)	QGPKEPFRDYVDRFYKTLRAE-QA	no virus	murine()
		References: [Matsuo (1992)]				
		<ul style="list-style-type: none"> • V107: Reacts with FIV, HIV-1 and SIV AGM analogous peptides [Matsuo (1992)] 				
100	12-B-4	p24(161–170)	p24(293–302 IIIB)	FRDYVDRFYK	no Vaccine	murine(IgG1)
	Vaccine:	<i>Strain:</i> IIIB	<i>HIV component:</i> virus			
		References: [Niedrig (1988), Niedrig (1989)]				
		<ul style="list-style-type: none"> • 12-B-4: Epitope is defined as the overlap between two HIV-1 reactive peptides – cross-reacts with HIV-2 ROD and SIV MAC [Niedrig (1988), Niedrig (1989)] 				

Table of HIV MAbs

101	C5122	p24(161–170)	p24(293–302 HXB2)	FRDYVDRFYK	no Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> viral lysate <i>HIV component:</i> virus				
		References: [Hinkula (1990)]				
		<ul style="list-style-type: none"> • C5122: Defined by peptide blocking of binding to native protein – WB reactive with p53 and p24 [Hinkula (1990)] 				
102	9A4C4	p24(170–188)	p24(303–317 IIIB)	KTLRAEQASQEVKNWMTET	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> p24				
		References: [Janvier (1990), Janvier1992, Robert-Hebmann (1992b), Robert-Hebmann (1992a)]				
		<ul style="list-style-type: none"> • 9A4C4: Mapped to aa260–267 through Pepsan method [Janvier (1990)] – and to aa303–317 through EIA pentadecapeptide method [Janvier1992] 				
103	11C10B10	p24(171–185)	p24(303–317 IIIB)	TLRAEQASQEVKNWM	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> p24				
		References: [Janvier (1990), Janvier1992]				
		<ul style="list-style-type: none"> • 11C10B10: Mapped to aa260–267 through Pepsan method [Janvier (1990)] and to aa303–317 through EIA pentadecapeptide method [Janvier1992] 				
104	11D11F2	p24(171–185)	p24(303–317 IIIB)	TLRAEQASQEVKNWM	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> p24				
		References: [Janvier (1990), Janvier1992]				
		<ul style="list-style-type: none"> • 11D11F2: Mapped to aa260–267 through Pepsan method [Janvier (1990)] and to aa303–317 through EIA pentadecapeptide method [Janvier1992] 				
105	CD12B4	p24(171–185)	p24(303–317 LAI)	TLRAEQASQEVKNWM	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus				
		Donor: R. B. Ferns and R. S. Tedder				
		References: [Ferns (1987), Ferns (1989)]				
		<ul style="list-style-type: none"> • CD12B4: Reacted with both p55 and p24 – strain-specific binding [Ferns (1987)] • CD12B4: UK Medical Research Council AIDS reagent: ARP346 				
106	BE3	p24(176–190)	p24(308–322 HXB2)	QASQEVKNWMTETLL	no Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> recombinant protein <i>HIV component:</i> p24-p15				
		Donor: B. Wahren				
		References: [Hinkula (1990)]				
		<ul style="list-style-type: none"> • BE3: Defined by peptide blocking of binding to native protein – WB reactive with p53 and p24 [Hinkula (1990)] • BE3: UK Medical Research Council AIDS reagent: ARP368 				

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107	L14	p24(176–190)	p24(308–322 HXB2)	QASQEVKNWMTETLL	no Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> p24-p15 Donor: B. Wahren References: [Hinkula (1990)] <ul style="list-style-type: none"> • L14: Defined by peptide blocking of binding to native protein – WB reactive with p53 and p24 [Hinkula (1990)] • L14: UK Medical Research Council AIDS reagent: ARP369 </p>						
108	108/03	p24(181–190)	p24(313–322 IIIB)	VKNWMTETLL	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> β-galactosidase fusion protein <i>Strain:</i> IIIB <i>HIV component:</i> p24 References: [Niedrig (1991)] <ul style="list-style-type: none"> • 108/03: Cross-reactive between HIV-1, HIV-2 and SIV MAC by multiple tests [Niedrig (1991)] </p>						
109	110/015	p24(181–190)	p24(313–322 IIIB)	VKNWMTETLL	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> β-galactosidase fusion protein <i>Strain:</i> IIIB <i>HIV component:</i> p24 References: [Niedrig (1991)] <ul style="list-style-type: none"> • 110/015: Cross-reactive between HIV-1, HIV-2 and SIV MAC by multiple tests [Niedrig (1991)] </p>						
110	32:32K	p24(199–222)	p24(331–354 HXB2)	KTILKALGPAATLEEMMTACQ-GVG	Vaccine	murine(IgG1 λ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> p24-p15 References: [Hinkula (1990)] <ul style="list-style-type: none"> • 32:32K: Epitope defined by peptide blocking of binding to native protein – WB reactive with p53 and p24 [Hinkula (1990)] • 32:32K: UK Medical Research Council AIDS reagent: ARP368 </p>						
111	C5200	p24(199–222)	p24(331–354 HXB2)	KTILKALGPAATLEEMMTACQ-GVG	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> viral lysate References: [Hinkula (1990)] <ul style="list-style-type: none"> • C5200: Epitope defined by peptide blocking of binding to native protein [Hinkula (1990)] </p>						
112	FH2	p24(201–215)	p24(333–347 HXB2)	ILKALGPAATLEEMM	no Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> p24-p15 References: [Hinkula (1990)] <ul style="list-style-type: none"> • FH2: Defined by peptide blocking of binding to native protein – WB reactive with p53 and p24 [Hinkula (1990)] </p>						
113	13B5	p24(205–214)	p24(205–213)	LGPAATLEEM	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> p24</p>						

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		Ab type: C-term	Donor: bioMerieux			
		References: [Berthet-Colominas (1999)]				
		<ul style="list-style-type: none"> • 13B5: Fab bound to p24 capsid for crystallization and study of p24's structure [Berthet-Colominas (1999)] 				
114	106/01	p24(211–230)	p24(343–362 IIIB)	LEEMMTACQGVGGPGHKARV	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> β -galactosidase fusion protein		<i>Strain:</i> IIIB	<i>HIV component:</i> p24	
		References: [Niedrig (1991)]				
		<ul style="list-style-type: none"> • 106/01: Cross-reactive between HIV-1, HIV-2 and SIV MAC by multiple tests [Niedrig (1991)] 				
115	LH-104-B	p24(225–230)	p24(357–362 BRU)	GHKARV	no Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> peptide		<i>Strain:</i> BRU		
		References: [Haaheim (1991)]				
		<ul style="list-style-type: none"> • LH-104-B: Binds exclusively with p55 (not p24), in contrast to LH-104-I [Haaheim (1991)] • LH-104-B: UK Medical Research Council AIDS reagent: ARP308 				
116	LH-104-I	p24(226–231)	p24(358–363 BRU)	HKARVL	no Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> peptide		<i>Strain:</i> BRU		
		References: [Haaheim (1991)]				
		<ul style="list-style-type: none"> • LH-104-I: Binds exclusively with p24 (not p55), in contrast to LH-104-B [Haaheim (1991)] • LH-104-I: UK Medical Research Council AIDS reagent: ARP321 				
117	polyclonal	p24()	p24()		no Vaccine	rabbit(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>Strain:</i> B subtype	<i>HIV component:</i> p24	
		References: [Gupta (2001)]				
		<ul style="list-style-type: none"> • Gag p24 is the mostly widely used HIV protein for serological based diagnostic kits — phage display libraries of HIV-1 p24 identified 2 epitope-rich regions: 70% of the clones that were identified using immunized rabbit sera had DNA fragments from the N-terminal region spanning 150–240 of Gag, and 30% from the carboxy-terminal region of p24 containing amino acids 310–360 — subtype B and C comparisons were made [Gupta (2001)] 				

Table 3: p2p7p1p6

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
118 LH-104-G	p2p7p1p6(1–5)	p24(363–368 BRU)	LAEAMS	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> BRU</p> <p>References: [Haaheim (1991)]</p> <ul style="list-style-type: none"> • LH-104-G: Reacts with both p24 and p55, in contrast to LH-104-I [Haaheim (1991)] • LH-104-G: UK Medical Research Council AIDS reagent: ARP320 • This peptide appears to overlap the p24-p2 cleavage site 						
119 i5B11	p2p7p1p6(19–28)	p7(5–14)	NFRNQRKIVK	no	Vaccine	rat(IgG2a)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> NCp7</p> <p>References: [Otake (1994), Tanchou (1994), Tanchou (1995)]</p> <ul style="list-style-type: none"> • i5B11: i5B11 and 15B11 may be two names for the same MAb • i5B11: Epitope mapped by ELISA and BIAcore – inhibits NCp7 primer tRNA binding [Tanchou (1994)] • i5B11: MAb reacts with NCp7, NCp15, and partially inhibits NCp7-tRNA interaction [Tanchou (1995)] 						
120 EC6	p2p7p1p6(45–54)	p15(408–417 HXB2)	PRKKGWCWCG	no	Vaccine	murine(IgG2a κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> p24-p15</p> <p>References: [Hinkula (1990)]</p> <ul style="list-style-type: none"> • EC6: Epitope defined by peptide blocking of binding to native protein – WB reactive with p53 [Hinkula (1990)] 						
121 M12	p2p7p1p6(45–54)	p15(408–417 HXB2)	PRKKGWCWCG	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> p24-p15</p> <p>References: [Hinkula (1990)]</p> <ul style="list-style-type: none"> • M12: There is a p15 and a gp120 MAb both called M12 • M12: Epitope defined by peptide blocking of binding to native protein – WB reactive with p53 [Hinkula (1990)] 						
122 DG8	p2p7p1p6(66–81)	p7(52–67)	RQANFLGKIWPSYKGR		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> NCp7</p> <p>References: [Tanchou (1995)]</p> <ul style="list-style-type: none"> • DG8: Binds proximal to the second zinc-finger, inhibits NCp7-tRNA interaction [Tanchou (1995)] 						
123 EB5	p2p7p1p6(66–81)	p7(52–67)	RQANFLGKIWPSYKGR		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> NCp7</p> <p>References: [Tanchou (1995)]</p>						

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- EB5: Binds proximal to the second zinc-finger – mutation at position 59 (Lys to Ser) results in 10-fold reduction in reactivity [Tanchou (1995)]

124	HH3	p2p7p1p6(66–81)	p7(52–67)	RQANFLGKIWPSYKGR	no	Vaccine	murine(IgG2b)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7					
		References: [Tanchou (1994), Tanchou (1995)]					
		• HH3: Epitopes mapped by ELISA and BIAcore – does not inhibit NCp7 primer tRNA binding [Tanchou (1994)]					
		• HH3: Binds proximal to the second zinc-finger [Tanchou (1995)]					
125	AD2	p2p7p1p6(78–86)	p7(64–72)	YKGRPGNFL	no	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7					
		References: [Tanchou (1995)]					
		• AD2: Binds at C term of NCp7 [Tanchou (1995)]					
126	CA5	p2p7p1p6(78–86)	p7(64–72)	YKGRPGNFL	no	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7					
		References: [Tanchou (1995)]					
		• CA5: Binds at C term of NCp7 [Tanchou (1995)]					
127	DF3	p2p7p1p6(78–86)	p7(64–72)	YKGRPGNFL	no	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7					
		References: [Tanchou (1995)]					
		• DF3: Binds at C term of NCp7 [Tanchou (1995)]					
128	EC3	p2p7p1p6(78–86)	p7(64–72)	YKGRPGNFL	no	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7					
		References: [Tanchou (1995)]					
		• EC3: Binds at C term of NCp7 [Tanchou (1995)]					
129	FC12	p2p7p1p6(78–86)	p7(64–72)	YKGRPGNFL	no	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7					
		References: [Tanchou (1995)]					
		• FC12: Binds at C term of NCp7, reacts with NCp15, inhibits NCp7-tRNA interaction [Tanchou (1995)]					
130	GE4	p2p7p1p6(78–86)	p7(64–72)	YKGRPGNFL	no	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7					
		References: [Tanchou (1995)]					
		• GE4: Binds at C term of NCp7 [Tanchou (1995)]					

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131	JB7	p2p7p1p6(78-86)	p7(64-72)	YKGRPGNFL	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> NCp7</p> <p>References: [Tanchou (1995)]</p> <ul style="list-style-type: none"> • JB7: Binds at C term of NCp7 [Tanchou (1995)] 							
132	JF11	p2p7p1p6(78-86)	p7(64-72)	YKGRPGNFL	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> NCp7</p> <p>References: [Tanchou (1994), Tanchou (1995)]</p> <ul style="list-style-type: none"> • JF11: Epitopes mapped by ELISA and BIAcore – does not inhibit NCp7 primer tRNA binding [Tanchou (1994)] • JF11: Binds at C term of NCp7 [Tanchou (1995)] 							

B Cell

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Table 4: **Gag**

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
133 183-H12-5C	Gag()	p24()		no		murine(IgG1)
<p>Donor: Bruce Chesebro and Kathy Wehrly, Rocky Mountain Laboratories, Hamilton, Montana</p> <p>References: [Chesebro (1992), Toohey (1995), Wehrly & Chesebro(1997)]</p> <ul style="list-style-type: none"> • 183-H12-5C: Cross-reacts with HIV1 and HIV-2 p24, and SIV p27 • 183-H12-5C: Used as antigen capture reagent for p24 ELISA [Chesebro (1992), Toohey (1995)] • 183-H12-5C: Cross-reacts with HIV1 and HIV-2 p24, and SIV p27 [Wehrly & Chesebro(1997)] • 183-H12-5C: NIH AIDS Research and Reference Reagent Program: 3537 						
134 241-D	Gag()	p24()		no		human(IgG1λ)
<p>Donor: Susan Zolla-Pazner (Zollas01@mcr6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (1989), Tyler (1990), Robinson (1991)]</p> <ul style="list-style-type: none"> • 241-D: An antibody by this name is available in the NIH AIDS Research and Reference Reagent Program, and they refer to the papers: [Gorny (1989), Tyler (1990), Robinson (1991)], but no p24 MAb by this name is discussed in these papers • 241-D: MH AIDS Research and Reference Reagent program: 1244 						
135 2A6	Gag()	p17()				()
<p>Donor: A. O. Arthur, Frederick Cancer Research and Development Center, Frederick, MD</p> <p>References: [Pincus (1998)]</p> <ul style="list-style-type: none"> • 2A6: Part of a panel of 17 MAbs used as controls testing for the dual specificity of MAb G11H3 for both p17 and mycoplasma [Pincus (1998)] 						
136 5E2.A3k	Gag(dis p24 1–158)	p24(dis 1–158 SF2)		no		murine(IgG1)
<p>Donor: Biodesign International, Kennebunk, Maine, USA</p> <p>References: [Hochleitner (2000a)]</p> <ul style="list-style-type: none"> • 5E2.A3k: The Ab binding site was studied with epitope excision (protein is bound in native conformation to immobilized MAb, then digested with proteolytic enzymes) and extraction (protein is digested then allowed to react with Ab), followed by mass spectroscopy, as well as lysine modification – the epitope is discontinuous, but involves the highly conserved N-term proline, and the antibody recognizes SIVs and HIV-2 as well as HIV-1 p24 [Hochleitner (2000a)] 						
137 71–31	Gag()	p24()		no		human(IgG1λ)
<p>References: [Gorny (1989), Robinson (1990b), Robinson (1991), Spear (1993), Gorny (1997), Gorny (1998), Bandres (1998)]</p> <ul style="list-style-type: none"> • 71–31: Did not enhance HIV-1 IIIB infection [Robinson (1990b)] • 71–31: No enhancing or neutralizing activity [Robinson (1991)] 						

- 71–31: Did not mediate deposition of complement component C3 on HIV infected cells [Spear (1993)]
- 71–31: Included as a negative control in studies that demonstrate that CXCR4 can bind to gp120 in the absence of CD4-gp120 interactions, and that this binding can be enhanced by Env deglycosylation [Bandres (1998)]
- 71–31: NIH AIDS Research and Reference Reagent Program: 530

138	91–6	Gag() p24(121–240 IIIB) References: [Gorny (1989), Robinson (1990b)]	no	HIV-1 infection	human(IgG1 λ)
		<ul style="list-style-type: none"> • 91–6: No enhancing activity for HIV-1 IIIB [Robinson (1990b)] • 91–6: NIH AIDS Research and Reference Reagent Program: 1239 			
139	98–4.3	Gag() p24() References: [Robinson (1991)]	no	HIV-1 infection	human(IgG1 λ)
		<ul style="list-style-type: none"> • 98–4.3: No enhancing or neutralizing activity [Robinson (1991)] 			
140	98–4.9	Gag() p24() References: [Gorny (1989)]	no	HIV-1 infection	murine(IgG3 λ)
141	AC2	Gag(dis) p7(dis) Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> NCp7 References: [Tanchou (1995)]	no	Vaccine	murine(IgG)
		<ul style="list-style-type: none"> • AC2: Binds NCp7 independent of Zn fingers, does not react with NCp15 [Tanchou (1995)] 			
142	anti-p24	Gag() p24() Vaccine: <i>Vector/type:</i> recombinant protein, virus-like particle <i>HIV component:</i> Gag, Pol, Nef, gp120 Donor: Intracel Co References: [Buonaguro (2001)]		Vaccine	murine(IgG)
		<ul style="list-style-type: none"> • Anti-p24: HIV-1 pr55 gag-based virus-like particles (VLP) carrying Nef and Pol open reading frames, as well as gp120 of the clade A isolate 94UG018, were created using a Baculovirus expression system to package additional ORFS into the VLP – anti-V3 and anti-p24 antibodies were used to assess the expression levels and Gag and gp120-TM were found to be expressed at comparable levels on the VLP 			
143	BC1071	Gag() p24() Donor: Aalto BioReagents References: [Schonning (1999)]	no	HIV-1 infection	murine()
		<ul style="list-style-type: none"> • BC1071: The stoichiometry of MAb neutralization was tested and MAb BC1071 was used in this study for virion quantitation [Schonning (1999)] 			

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144	BE10	Gag(dis)	p7(dis)	no Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7			
		References: [Tanchou (1995)]			
		<ul style="list-style-type: none"> • BE10: Binding NCp7 requires Zn fingers, does not react with NCp15, inhibits NCp7-tRNA interaction [Tanchou (1995)] 			
145	CD9	Gag(dis)	p7(dis)	no Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7			
		References: [Tanchou (1995)]			
		<ul style="list-style-type: none"> • CD9: Binds NCp7 independent of Zn fingers, does not react with NCp15 [Tanchou (1995)] 			
146	CH9B2	Gag()	p17()	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus			
		Donor: R. B. Ferns and R. S. Tedder			
		References: [Ferns (1987), Ferns (1989)]			
		<ul style="list-style-type: none"> • CH9B2: Reactive against p18 and p55 [Ferns (1987)] • CH9B2: UK Medical Research Council AIDS reagent: ARP349 			
147	ED8	Gag(dis)	p7(dis)	no Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> NCp7			
		References: [Tanchou (1995)]			
		<ul style="list-style-type: none"> • ED8: Binds NCp7 independent of Zn fingers, does not react with NCp15 [Tanchou (1995)] 			
148	EH12E1	Gag(dis)	p24(dis)	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus			
		Donor: R. B. Ferns and R. S. Tedder			
		References: [Ferns (1987), Ferns (1989)]			
		<ul style="list-style-type: none"> • EH12E1: Reacted with p55 and p24 in WB [Ferns (1987)] • EH12E1: UK Medical Research Council AIDS reagent: ARP313 			
149	G11G1	Gag()	p17()		rat()
		References: [Shang (1991), Pincus (1996)]			
		<ul style="list-style-type: none"> • G11G1: Immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but only if the antigen was expressed at the cell surface – ricin-G11G1 did not mediate cell killing [Pincus (1996)] 			

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150	G11H3	Gag(dis) p17(dis)			()
		References: [Shang (1991), Pincus (1998)]			
		<ul style="list-style-type: none"> • G11H3: This MAb is cross-reactive between p17 and mycoplasma – this antibody binds strain specifically to the variable lipoprotein (Vlp) F of <i>M. hyorhinis</i>, in the region of the carboxy-terminal repeat CGGSTPTPEQGNNQGGSTPTPEQNSQVSK – the p17 epitope is discontinuous, but p17 and Vlp F share the tetrapeptide SQVS [Pincus (1998)] 			
151	human sera	Gag() p24()		HIV-1 infection	human(IgG)
		References: [Binley (1997b)]			
		<ul style="list-style-type: none"> • Retention of anti-Env antibodies and loss of anti-Gag antibodies during disease progression was studied, and suggested to be the result of the loss of T-cell help and the unique ability of Env to stimulate B cells even in a backdrop of declining CD4 cells, because of the ability of Env to bind to the CD4 molecule [Binley (1997b)] 			
152	HyHIV-19	Gag(dis) p17(dis JMH1)		no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein <i>HIV component:</i> p17			
		References: [Liu (1995), Ota (1998)]			
		<ul style="list-style-type: none"> • HyHIV-19: Does not react with p17 peptides – K_a is $3.7 \times 10^6 \text{ M}^{-1}$ for rec p17 – inhibited growth of HIV-1 JMH1 in MT-4 cells when added 24 hours after the initial culture [Ota (1998)] 			
153	IE8G2	Gag() p24()		Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> inactivated virus <i>Strain:</i> CBL-1 <i>HIV component:</i> virus			
		Donor: R. B. Ferns and R. S. Tedder			
		References: [Ferns (1987), Ferns (1989)]			
		<ul style="list-style-type: none"> • IE8G2: Reacted with both p55 and p24 – broadly reactive – showed less than 75% homologous inhibition [Ferns (1987)] • IE8G2: UK Medical Research Council AIDS reagent: ARP347 			
154	LH-104-A	Gag(dis 284–289 + 351–356) p24(dis BRU)	DIRQGP + QGVGGP	no Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> peptide <i>HIV component:</i> p24			
		References: [Haaheim (1991)]			
		<ul style="list-style-type: none"> • LF-104-A: A 104 amino acid peptide was used to immunize mice – hexapeptide scans revealed two reactive p24 peptides – cross-competition studies indicated the region 270–286 [Haaheim (1991)] • LH-104-A: UK Medical Research Council AIDS reagent: ARP307 			
155	LH-104-C	Gag(dis 288–293 + 351–356) p24(dis BRU)	GPKEPF + QGVGGP	no Vaccine	murine(IgG3 κ)
	Vaccine:	<i>Vector/type:</i> peptide <i>HIV component:</i> p24			
		References: [Haaheim (1991)]			
		<ul style="list-style-type: none"> • LF-104-C: A 104 amino acid peptide was used to immunize mice – hexapeptide scans revealed two reactive p24 peptides – cross-competition studies indicated the region 351–373 [Haaheim (1991)] 			

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- LH-104-C: UK Medical Research Council AIDS reagent: ARP309

156	polyclonal	Gag()	Gag(LAI)		Vaccine	murine()
	Vaccine:	<i>Vector/type:</i> DNA prime with recombinant protein boost		<i>Strain:</i> LAI	<i>HIV component:</i> Gag, Tat, Nef	<i>Stimulatory Agents:</i> IL18
		References: [Billaut-Mulot (2001)]				
		<ul style="list-style-type: none"> • DNA vaccinated BALB/c mice primed and boosted with a multiepitopic vaccine with IL18 showed lymphoproliferative and CTL responses – co-administration of IL18 increased T-cell responses but decreased anti-HIV antibody levels 				
157	polyclonal	Gag()	p24(SF2)		Vaccine	murine()
	Vaccine:	<i>Vector/type:</i> recombinant protein microparticles		<i>Strain:</i> SF2	<i>HIV component:</i> gp120, p24	<i>Stimulatory Agents:</i> PLG+MF59
		References: [O'Hagan (2000)]				
		<ul style="list-style-type: none"> • Microparticles were used as an adjuvant for entrapped HIV-1 gp120 and induced strong serum IgG responses in mice – polylactide co-glycolide polymer (PLG) microparticles in combination with MF59 had the highest Ab response and also induced p24 specific CTL [O'Hagan (2000)] 				
158	polyclonal	Gag()	Gag(SF2)		Vaccine	mouse, guinea pig, macaque()
	Vaccine:	<i>Vector/type:</i> DNA, recombinant protein croparticles, aluminum phosphate, MF-59		<i>Strain:</i> SF2	<i>HIV component:</i> p55	<i>Stimulatory Agents:</i> PLG mi-
		References: [O'Hagan (2001)]				
		<ul style="list-style-type: none"> • DNA vaccines of codon-optimized Env and Gag genes driven by CMV promotors absorbed on to PLG microparticles were more effective than naked DNA at eliciting strong Ab responses (more rapid, higher titer, more stable), comparable to gp120 in MF-59 [O'Hagan (2001)] 				
159	polyclonal	Gag()	p55()		no Vaccine	mouse()
	Vaccine:	<i>Vector/type:</i> recombinant protein, virus-like particle		<i>Strain:</i> LAI	<i>HIV component:</i> V3, CD4BS, p55	
		References: [Truong (1996)]				
		<ul style="list-style-type: none"> • Antibodies raised against recombinant anti-p55 virus-like particles with the p24 region 196–226 deleted, bearing inserts of either the V3 or the CD4BS regions of gp120 were studied – no neutralizing responses, weak Env and strong Gag responses were elicited – the major homology region (MHR) and proximal regions were found to be required for capsid assembly [Truong (1996)] 				
160	polyclonal	Gag()	p24()		no Vaccine	rat()
	Vaccine:	<i>Vector/type:</i> gp120 depleted whole killed virus whole virus		<i>Strain:</i> HZ321 (subtype A env, subtype G gag)	<i>HIV component:</i>	
		<i>Stimulatory Agents:</i> CpG, Freund's adjuvant				
		References: [Moss (2000)]				

- Lewis rats co-immunized with HIV-1 antigen in Freund's and with immunostimulatory sequences CpG stimulated increased IFN γ expressing CD4+ and CD8+ T cells and anti-p24 antibodies relative to antigen in Freund's without CpG

161	V7-8	Gag() p24()	no HIV-1 infection	murine(IgG3 κ)
<p>References: [Robinson (1990b), Montefiori (1991)]</p> <ul style="list-style-type: none"> • V7-8: Did not enhance HIV-1 IIIB infection [Robinson (1990b)] • V7-8: Reacted with HIV-1IIIB, RF, and MN [Montefiori (1991)] • V7-8: NIH AIDS Research and Reference Reagent Program: 381 				

Table 5: **Protease**

MAB ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
162 1696	Protease(1–7)	Pro(1–7 BH10)	PQIYLWQ		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> Protease</p> <p>Ab type: N-term References: [Lescar (1999)]</p> <ul style="list-style-type: none"> • 1696: MAb binds to HIV-1 and HIV-2, putative epitopes are PQIYLWQ and PQFSLWK respectively – Pro1 is critical, QIYLWQR residues 2–8 do not compete without it – MAb disrupts catalytic activity – crystal structure of Fab at 3 Å resolution reveals a deep cavity lined by acidic and hydrophobic residues – the binding region is located within the region required for dimerization and the Fab structure could serve as a basis for drug design targeting this region [Lescar (1999)] 						
163 10E7	Protease(36–46)	Pro(38–45 HXB2)	MSLPGRWKPKM	no	Vaccine	hamster(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Protease</p> <p>References: [Croix (1993)]</p> <ul style="list-style-type: none"> • 10E7: Immunodominant region of protease in Armenian hamster (but only weakly reactive in people, see: [Bjorling1992]) – peptide MSLPGRWKP blocks protease binding [Croix (1993)] 						
164 F11.2.32	Protease(36–46)	Pro(36–46 BH10)	MSLPGRWKPKM		Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> Protease</p> <p>Ab type: flap region References: [Lescar (1996), Lescar (1997), Lescar (1999)]</p> <ul style="list-style-type: none"> • F11.2.32: Binding leads to significant inhibition in proteolytic activity – crystal structure of Fab-peptide was determined to 2.2 Å resolution – bound peptide shows no structural similarity to the corresponding segment in native protease suggesting binding may distort protein structure [Lescar (1997)] • F11.2.32: Distortion may occur in the flap region of the protein, important for regulating access of substrate to the catalytic site [Lescar (1999)] 						
165 13E1	Protease(38–45)	Pro(38–45 HXB2)	LPGRWKPK	no	Vaccine	hamster(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Protease</p> <p>References: [Croix (1993)]</p> <ul style="list-style-type: none"> • 13E1: LPGRWKPK is the core of the epitope – binds to MSLPGRWKPKM with slightly higher affinity [Croix (1993)] 						
166 8B11	Protease(38–45)	Pro(38–45 HXB2)	LPGRWKPK	no	Vaccine	hamster(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Protease</p> <p>References: [Croix (1993)]</p> <ul style="list-style-type: none"> • 8B11: LPGRWKPK is the core of the epitope – binds to MSLPGRWKPKM with slightly higher affinity [Croix (1993)] 						

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167	8C10	Protease(38–45)	Pro(38–45 HXB2)	LPGRWKPK	no Vaccine	hamster(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Protease		
		References: [Croix (1993)]				
		<ul style="list-style-type: none"> ● 8C10: LPGRWKPK is the core of the eptiope – binds to MSLPGRWKPKM with sightly higher affinity [Croix (1993)] 				
168	8G5	Protease(38–45)	Pro(38–45 HXB2)	LPGRWKPK	no Vaccine	hamster(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Protease		
		References: [Croix (1993)]				
		<ul style="list-style-type: none"> ● 8G5: LPGRWKPK is the core of the eptiope – binds to MSLPGRWKPKM with sightly higher affinity [Croix (1993)] 				

B Cell

Table of HIV MAbs

Table 6: **RT**

169	1E8	RT(65–73)	RT(65–73)	KKDSTKWRK	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> RT <i>Stimulatory Agents:</i> nitrocellulose</p> <p>References: [Wu (1993), Gu (1996)]</p> <ul style="list-style-type: none"> • 1E8: Inhibits RT activity, binding site overlaps with two AZT resistance mutations [Wu (1993)] • 1E8: Significantly inhibits DNA polymerase activity of RT by hindering binding of dNTPs – additive or synergistic RT inhibition with nevirapine and delavirdine [Gu (1996)] 						
170	1.152 B3	RT(294–302)	RT(294–302)	PLTEEAEELE	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> RT</p> <p>References: [Orvell (1991)]</p> <ul style="list-style-type: none"> • 1.152 B3: Weakly positive by immunofluorescence – binding inhibits RT enzymatic activity [Orvell (1991)] 						
171	1.158 E2	RT(294–302)	RT(294–302)	PLTEEAEELE	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> RT</p> <p>References: [Orvell (1991)]</p> <ul style="list-style-type: none"> • 1.158 E2: Negative by immunofluorescence – binding inhibits RT enzymatic activity [Orvell (1991)] 						
172	31D6	RT(294–318)	RT(294–319)	PLTEEAEELELAENREILKEPVHGVY	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> <i>E. coli</i> Trp fusion protein <i>HIV component:</i> RT</p> <p>References: [Szilvay (1992)]</p> <ul style="list-style-type: none"> • 31D6: Strong inhibitor of RT, > 50% inhibition [Szilvay (1992)] 						
173	31G8	RT(294–318)	RT(294–319)	PLTEEAEELELAENREILKEPVHGVY	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> <i>E. coli</i> Trp fusion protein <i>HIV component:</i> RT</p> <p>References: [Szilvay (1992)]</p> <ul style="list-style-type: none"> • 31G8: Weak inhibitor of RT, reactive by immunofluorescence [Szilvay (1992)] 						
174	32E7	RT(294–318)	RT(294–319)	PLTEEAEELELAENREILKEPVHGVY	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> <i>E. coli</i> Trp fusion protein <i>HIV component:</i> RT</p> <p>References: [Szilvay (1992)]</p> <ul style="list-style-type: none"> • 32E7: Weak inhibitor of RT, reactive by immunofluorescence [Szilvay (1992)] 						
175	33D5	RT(294–318)	RT(294–319)	PLTEEAEELELAENREILKEPVHGVY	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> <i>E. coli</i> Trp fusion protein <i>HIV component:</i> RT</p> <p>References: [Szilvay (1992)]</p> <ul style="list-style-type: none"> • 33D5: Weak inhibitor of RT, reactive by immunofluorescence [Szilvay (1992)] 						

Table of HIV MAbs

176	5B2	RT(294–318)	RT(294–319)	PLTEEALELELAENREILKEPVHGVY	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> <i>E. coli</i> Trp fusion protein		<i>HIV component:</i> RT		
		References: [Szilvay (1992)]				
		<ul style="list-style-type: none"> • 5B2: There is an RT specific Ab [Szilvay (1992)] and a gp41 specific Ab [Tian (2001)] both called 5B2 • 5B2: Weak inhibitor of RT, reactive by immunofluorescence [Szilvay (1992)] • 5B2: UK Medical Research Council AIDS reagent: ARP3018 				
177	polyclonal	RT(295–304)	RT(295–304 PV22)	LTEEALELELA	no HIV-1 infection	human(IgG)
		References: [Grimison & Laurence(1995)]				
178	1.153 G10	RT(350–354)	RT(350–354)	KTGKY	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> RT		
		References: [Orvell (1991)]				
179	RTMAb8	RT(376–383)	RT(532–539)	TTESIVIW	no Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> RT		
		References: [Tisdale (1988), Ferns (1991)]				
180	1D4A3	RT(384–387)	RT(540–543)	GKIP	no Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> RT		
		References: [Ferns (1991)]				
181	RT6H	RT(384–387)	RT(540–543)		no Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> RT		
		References: [Ferns (1991)]				
182	1.160 B3	RT(442–450)	RT(442–450)	VDGAANRET	no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> RT		
		References: [Orvell (1991)]				
183	polyclonal	RT(521–531)	RT(521–531 PV22)	IIEQLIKKEKV	no HIV-1 infection	human(IgG)
		References: [Grimison & Laurence(1995)]				
184	C2003	RT(536–549)	RT(703–716 BH10)	VPAHKGIGGNEQVD	no Vaccine	rabbit(IgG)
	Vaccine:	<i>Vector/type:</i> peptide	<i>Strain:</i> BH10			

Table 7: **Integrase**

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
189 1C4	Integrase(1–16)	Integrase(1–16 HXB2)	FLDGIDKAQDEHEKYH	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase</p> <p>Ab type: N-term References: [Haugan (1995), Nilsen (1996)]</p> <ul style="list-style-type: none"> • 1C4: MAb interferes with integrase binding to DNA [Haugan (1995)] • 1C4: One of a large set of MAbs that interact with the N-terminal part of integrase: 1C4, 2C11, 2E3, 3E11, 3F9, 5F8, 6G5, 7B6, 7C6 – these MAbs inhibit end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] 						
190 2C11	Integrase(1–16)	Integrase(1–16 HXB2)	FLDGIDKAQDEHEKYH	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase</p> <p>Ab type: N-term References: [Nilsen (1996)]</p> <ul style="list-style-type: none"> • 2C11: One of a large set of MAbs that interact with the N-terminal part of integrase: 1C4, 2C11, 2E3, 3E11, 3F9, 5F8, 6G5, 7B6, 7C6 – these MAbs inhibit end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] 						
191 2E3	Integrase(1–16)	Integrase(1–16 HXB2)	FLDGIDKAQDEHEKYH	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase</p> <p>Ab type: N-term References: [Nilsen (1996), Ovod (1992)]</p> <ul style="list-style-type: none"> • 2E3: There are two MAbs called 2E3 – the other one binds to Nef [Ovod (1992)] • 2E3: One of a large set of MAbs that interact with the N-terminal part of integrase: 1C4, 2C11, 2E3, 3E11, 3F9, 5F8, 6G5, 7B6, 7C6 – these MAbs inhibit end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] 						
192 3E11	Integrase(1–16)	Integrase(1–16 HXB2)	FLDGIDKAQDEHEKYH	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase</p> <p>Ab type: N-term References: [Otteken (1992), Nilsen (1996)]</p> <ul style="list-style-type: none"> • 3E11: There is another MAb with this ID that recognizes p17 [Otteken (1992)] • 3E11: Recognized an epitope present on HIV-2/SIVmac, SIVagm, HIV-1, and SIVmnd [Otteken (1992)] • 3E11: One of a large set of MAbs that interact with the N-terminal part of integrase: 1C4, 2C11, 2E3, 3E11, 3F9, 5F8, 6G5, 7B6, 7C6 – these MAbs inhibit end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] 						

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193	3F9	Integrase(1–16)	Integrase(1–16 HXB2)	FLDGIDKAQDEHEKYH	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase Ab type: N-term References: [Nilsen (1996)] <ul style="list-style-type: none"> • 3F9: One of a large set of MAbs that interact with the N-terminal part of integrase: 1C4, 2C11, 2E3, 3E11, 3F9, 5F8, 6G5, 7B6, 7C6 – these MAbs inhibit end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] </p>							
194	5F8	Integrase(1–16)	Integrase(1–16 HXB2)	FLDGIDKAQDEHEKYH	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase Ab type: N-term References: [Haugan (1995), Nilsen (1996)] <ul style="list-style-type: none"> • 5F8: There is another MAb with this ID that recognizes and unknown protein in HIV [Pinter (1995)] • 5F8: MAb interferes with integrase binding to DNA [Haugan (1995)] • 5F8: One of a large set of MAbs that interact with the N-terminal part of integrase: 1C4, 2C11, 2E3, 3E11, 3F9, 5F8, 6G5, 7B6, 7C6 – these MAbs inhibit end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] </p>							
195	6G5	Integrase(1–16)	Integrase(1–16 HXB2)	FLDGIDKAQDEHEKYH	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase Ab type: N-term References: [Nilsen (1996)] <ul style="list-style-type: none"> • 6G5: One of a large set of MAbs that interact with the N-terminal part of integrase: 1C4, 2C11, 2E3, 3E11, 3F9, 5F8, 6G5, 7B6, 7C6 – these MAbs inhibit end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] </p>							
196	7B6	Integrase(1–16)	Integrase(1–16 HXB2)	FLDGIDKAQDEHEKYH	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase Ab type: N-term References: [Nilsen (1996)] <ul style="list-style-type: none"> • 7B6: One of a large set of MAbs that interact with the N-terminal part of integrase: 1C4, 2C11, 2E3, 3E11, 3F9, 5F8, 6G5, 7B6, 7C6 – these MAbs inhibit end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] </p>							
197	7C6	Integrase(1–16)	Integrase(1–16 HXB2)	FLDGIDKAQDEHEKYH	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase Ab type: N-term References: [Nilsen (1996)] <ul style="list-style-type: none"> • 7C6: One of a large set of MAbs that interact with the N-terminal part of integrase: 1C4, 2C11, 2E3, 3E11, 3F9, 5F8, 6G5, 7B6, 7C6 – these MAbs inhibit end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] </p>							

Table of HIV MABs

198	6C5	Integrase(17–38)	Integrase(17–38 HXB2)	SNWRAMASDFNLPPVVAKEIV- A	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase Ab type: N-term References: [Haugan (1995), Nilsen (1996)] <ul style="list-style-type: none"> • 6C5: MAb interferes with integrase binding to DNA [Haugan (1995)] • 6C5: This MAb inhibits end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] </p>							
199	4D6	Integrase(42–55)	Integrase(42–55 HXB2)	KCQLKGEAMHGQVD	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase Ab type: N-term References: [Haugan (1995), Nilsen (1996)] <ul style="list-style-type: none"> • 4D6: MAb interferes with integrase binding to DNA [Haugan (1995)] • 4D6: This MAb inhibits end processing and DNA joining, and reduces reintegration activity [Nilsen (1996)] </p>							
200	7–16 (7–19)	Integrase(50–159)	Integrase(50–159 HXB2)		no	Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> chimeric maltose binding protein (MBP) <i>Strain:</i> IIIB <i>HIV component:</i> Integrase Ab type: Integrase catalytic core Donor: Yoshihiro Kitamura, Div of Mol Genetics, Nat Inst of Infectious Diseases, Musashimurayama, Japan References: [Ishikawa (1999)] <ul style="list-style-type: none"> • 7–16: Binds to the central catalytic domain – the paper seems to sometimes call this antibody 7–16, sometimes 7–19, a possible typo [Ishikawa (1999)] </p>							
201	4F6	Integrase(56–102)	Integrase(56–102 HXB2)	CSPGIWQLDCTHLEGKVLV- AVHVASGYIEAEVIPAETGQE- TAYFLL	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase Ab type: Integrase catalytic core References: [Haugan (1995), Nilsen (1996)] <ul style="list-style-type: none"> • 4F6: MAb binding had minimal effects on Integrase <i>in vitro</i> activities [Nilsen (1996)] • 4F6: MAb interferes with integrase binding to DNA [Haugan (1995)] </p>							
202	8G4	Integrase(dis 22– 31 + 82–101)	Integrase(dis 12–42 HXB2)	MASDFNLPPV + GYIEAEVI- PAETGQETAYFI?	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase References: [Haugan (1995), Nilsen (1996)] <ul style="list-style-type: none"> • 8G4: This MAb reacted strongly with peptides IN(12–31) and IN(22–42), and less strongly with peptide IN(82–101) – it did not react with a deletion mutant of positions 17–38 – this MAb inhibits end processing and DNA joining, but had little effect on integration activities [Nilsen (1996)] </p>							

Table of HIV MAbs

- 8G4: MAb interferes with integrase binding to DNA [Haugan (1995)]

203	anti-K159	Integrase(151–163)	Integrase(163–175)	VESMNKELKKIIG		Vaccine	rabbit(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> Integrase</p> <p>References: [Maroun (1999)]</p> <ul style="list-style-type: none"> ● anti-K159: Both the peptide K159, SQGVVESMNKELKKIIGQVRDQAEHLKTA, and the Abs raised against this peptide inhibit Integrase activity – K159 was found to fulfill condition of minimal number of helical heptads to achieve the formation of a stable coiled-coil structure – Integrase is proposed to function as a dimer interacting in this region [Maroun (1999)] 							
204	5D9	Integrase(186–250)	Integrase(186–250 HXB2)		no	Vaccine	murine(IgG1κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase</p> <p>Ab type: Integrase DNA binding domain References: [Nilsen (1996)]</p> <ul style="list-style-type: none"> ● 5D9: MAb binding had minimal effects on Integrase <i>in vitro</i> activities [Nilsen (1996)] ● 5D9: While C-term and N-term anti-Integrase MAbs interfere with Integrase-DNA binding, 5D9 which binds more centrally, does not [Haugan (1995)] 							
205	8–6	Integrase(211–227)	Integrase(211–227 HXB2)	KELQKQITKIQNFRVYY	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> chimeric maltose binding protein (MBP) <i>Strain:</i> IIIB <i>HIV component:</i> Integrase</p> <p>Donor: Yoshihiro Kitamura, Div of Mol Genetics, Nat Inst of Infectious Diseases, Musashimurayama, Japan</p> <p>References: [Ishikawa (1999)]</p> <ul style="list-style-type: none"> ● 8–6: Antibody binds proximal to the DNA binding region [Ishikawa (1999)] 							
206	19 (2–19, scAb2–19)	Integrase(228–236)	Integrase(228–236 LAI)	RDSRNPLWK	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Integrase</p> <p>Ab type: Integrase References: [Bizub-Bender (1994), Levy-Mintz (1996), Kitamura (1999)]</p> <ul style="list-style-type: none"> ● 19: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized – 19 has a low binding affinity [Bizub-Bender (1994)] ● 19: Called 2–19, scAb2–19 is a single-chain Ab made from MAb 2–19 –acts intra-cellularly to block infection at low MOI by binding to integrase – scAb interfered with the folding of Gag-Pol polyprotein, the Ab did not affect viral production in LAI transfected cells, but the virus produced was less infectious – authors suggest that the epitope may be conformational [Kitamura (1999)] 							
207	2–19	Integrase(228–236)	Integrase(228–236 HXB2)	RDSRNPLWK	no	Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> chimeric maltose binding protein (MBP) <i>Strain:</i> IIIB <i>HIV component:</i> Integrase</p>							

		Ab type: Integrase DNA binding domain	Donor: Yoshihiro Kitamura, Div of Mol Genetics, Nat Inst of Infectious Diseases, Musashimurayama, Japan				
		References: [Ishikawa (1999)]					
		<ul style="list-style-type: none"> • 2–19: MAb inhibits RT-Integrase interaction, and the terminal cleavage and strand transfer functions of Integrase, but not the disintegration activity [Ishikawa (1999)] 					
208	8–22	Integrase(237–252)	Integrase(237–252 HXB2)	GPAKLLWKGEAVVIQ	no	Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> chimeric maltose binding protein (MBP) <i>Strain:</i> IIIB <i>HIV component:</i> Integrase Ab type: Integrase DNA binding domain Donor: Yoshihiro Kitamura, Div of Mol Genetics, Nat Inst of Infectious Diseases, Musashimurayama, Japan References: [Ishikawa (1999)] <ul style="list-style-type: none"> • 8–22: MAb inhibits the terminal cleavage and strand transfer functions of Integrase, but not the disintegration activity [Ishikawa (1999)] 					
209	4–20	Integrase(253–261)	Integrase(253–261 HXB2)	DNSDIKVVP	no	Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> chimeric maltose binding protein (MBP) <i>Strain:</i> IIIB <i>HIV component:</i> Integrase Ab type: Integrase DNA binding domain Donor: Yoshihiro Kitamura, Div of Mol Genetics, Nat Inst of Infectious Diseases, Musashimurayama, Japan References: [Ishikawa (1999)] <ul style="list-style-type: none"> • 4–20: Inhibits the terminal cleavage and strand transfer functions of Integrase, but not the disintegration activity [Ishikawa (1999)] 					
210	6–19	Integrase(261–270)	Integrase(261–270 HXB2)	RRKAKIIRD	no	Vaccine	murine(IgG2b)
		Vaccine: <i>Vector/type:</i> chimeric maltose binding protein (MBP) <i>Strain:</i> IIIB <i>HIV component:</i> Integrase Ab type: Integrase DNA binding domain Donor: Yoshihiro Kitamura, Div of Mol Genetics, Nat Inst of Infectious Diseases, Musashimurayama, Japan References: [Ishikawa (1999)] <ul style="list-style-type: none"> • 6–19: Inhibits the terminal cleavage and strand transfer functions of Integrase, but not the disintegration activity [Ishikawa (1999)] 					
211	7C3	Integrase(262–271)	Integrase(262–271 HXB2)	RRKAKIIRDY	no	Vaccine	murine(IgG1 κ)
		Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase References: [Haugan (1995), Nilsen (1996)] <ul style="list-style-type: none"> • 7C3: MAb interferes with integrase binding to DNA [Haugan (1995)] • 7C3: A set of three MAbs recognize an epitope in this region, 7C3, 7F11, and 8E5 – all three HIV-1 MAbs cross-react with HIV-2 Integrase – these MAbs inhibit end-processing, DNA joining and reintegration, and had little effect on disintegration [Nilsen (1996)] 					

Table of HIV MAbs

212	7F11	Integrase(262–271)	Integrase(262–271 HXB2)	RRKAKIIRDY	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase</p> <p>References: [Nilsen (1996), Lasky (1987)]</p> <ul style="list-style-type: none"> • 7F11: A set of three MAbs recognize an epitope in this region, 7C3, 7F11, and 8E5 – all three HIV-1 MAbs cross-react with HIV-2 Integrase – these MAbs inhibit end-processing, DNA joining and reintegration, and had little effect on disintegration [Nilsen (1996)] • 7F11: There is another MAb with this name that binds to gp120 [Lasky (1987)] 							
213	8E5	Integrase(262–271)	Integrase(262–271 HXB2)	RRKAKIIRDY	no	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HXB2 <i>HIV component:</i> Integrase</p> <p>References: [Haugan (1995), Nilsen (1996)]</p> <ul style="list-style-type: none"> • 8E5: MAb interferes with integrase binding to DNA [Haugan (1995)] • 8E5: A set of three MAbs recognize an epitope in this region, 7C3, 7F11, and 8E5 – all three HIV-1 MAbs cross-react with HIV-2 Integrase – these MAbs inhibit end-processing, DNA joining and reintegration, and had little effect on disintegration [Nilsen (1996)] 							
214	MAb 35	Integrase(264–273)	Integrase(264–273)	KAKIIRDYGK	no	Vaccine	murine(IgG κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Integrase</p> <p>References: [Barsov (1996), Acel (1998)]</p> <ul style="list-style-type: none"> • MAb 35: There appears to be two different Integrase Abs with similar names: MAb 35 and 35 [Barsov (1996), Bizub-Bender (1994)] • MAb 35: Although MAb 35 does not inhibit HIV-1 IN, Fab 35 inhibits 3'-end processing, strand transfer and disintegration [Barsov (1996)] • MAb 35: Integrase was shown to have intrinsic DNA polymerase activity that can catalyze gap repair – MAb 35 inhibits this activity [Acel (1998)] 							

Table 8: Pol

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
215 12	Pol()	Integrase(1–58)		no	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Integrase</p> <p>References: [Bizub-Bender (1994), Levy-Mintz (1996)]</p> <ul style="list-style-type: none"> • 12: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized – the Zn finger motif is in the binding region – MAbs 12, 13 and 35 form a competition group [Bizub-Bender (1994)] • 12: Used for the creation of single-chain variable antibody fragments (SFvs) for internal cellular expression – neutralization of Integrase activity occurs prior to integration, whether the Ab is expressed in the nucleolus or the cytoplasm – relative binding affinity to IN: 12 > 17 = 33 > 21 > 4 [Levy-Mintz (1996)] 						
216 13	Pol()	Integrase(1–58)		no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Integrase</p> <p>References: [Bizub-Bender (1994)]</p> <ul style="list-style-type: none"> • 13: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized – the Zn finger motif is in the binding region – MAbs 12, 13 and 35 form a competition group [Bizub-Bender (1994)] 						
217 14	Pol()	Integrase(1–58)		no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Integrase</p> <p>References: [Bizub-Bender (1994)]</p> <ul style="list-style-type: none"> • 14: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized – the Zn finger motif is in the binding region – MAbs 14 and 17 form a competition group [Bizub-Bender (1994)] 						
218 16	Pol(dis)	Integrase(dis)		no	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Integrase</p> <p>References: [Bizub-Bender (1994)]</p> <ul style="list-style-type: none"> • 16: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized [Bizub-Bender (1994)] 						
219 17	Pol()	Integrase(1–58)		no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Integrase</p> <p>References: [Bizub-Bender (1994), Levy-Mintz (1996)]</p> <ul style="list-style-type: none"> • 17: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized – the Zn finger motif is in the binding region – MAbs 14 and 17 form a competition group [Bizub-Bender (1994)] • 17: Used for the creation of single chain variable antibody fragments (SFvs) for internal cellular expression – neutralization of Integrase activity occurs prior to integration, whether the Ab is expressed in the nucleolus or the cytoplasm – relative binding affinity to IN: 12 > 17 = 33 > 21 > 4 [Levy-Mintz (1996)] 						

Table of HIV MAbs

220	1C12B1	Pol()	RT(431–521)		Vaccine	murine()
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> RT		
		References: [Ferns (1991)]				
		<ul style="list-style-type: none"> • 1C12B1: Recognized both p66 and p51 in Western blot, binds to C terminus [Ferns (1991)] • 1C12B1: UK Medical Research Council AIDS reagent: ARP384 				
221	21	Pol()	Integrase(58–141)	no	Vaccine	murine(IgG2b)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Integrase		
		References: [Bizub-Bender (1994), Levy-Mintz (1996)]				
		<ul style="list-style-type: none"> • 21: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized [Bizub-Bender (1994)] • 21: Used for the creation of single chain variable antibody fragments (SFvs) for internal cellular expression – neutralization of Integrase activity occurs prior to integration, whether the Ab is expressed in the nucleolus or the cytoplasm – relative binding affinity to IN: 12 > 17 = 33 > 21 > 4 [Levy-Mintz (1996)] 				
222	32	Pol()	Integrase(259–288)	no	Vaccine	murine(IgG2b)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Integrase		
		References: [Bizub-Bender (1994)]				
		<ul style="list-style-type: none"> • 32: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized – MAbs 32 and 33 form a competition group [Bizub-Bender (1994)] 				
223	33	Pol()	Integrase(259–288)	no	Vaccine	murine(IgG2b)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Integrase		
		References: [Bizub-Bender (1994), Levy-Mintz (1996)]				
		<ul style="list-style-type: none"> • 33: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized – MAbs 32 and 33 form a competition group [Bizub-Bender (1994)] • 33: Used for the creation of single chain variable antibody fragments (SFvs) for internal cellular expression – neutralization of Integrase activity occurs prior to integration, whether the Ab is expressed in the nucleolus or the cytoplasm – relative binding affinity to IN: 12 > 17 = 33 > 21 > 4 [Levy-Mintz (1996)] 				
224	35	Pol()	Integrase(1–58)	no	Vaccine	murine(IgG2b)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Integrase		
		References: [Bizub-Bender (1994)]				
		<ul style="list-style-type: none"> • 35: There appears to be two Integrase Abs with similar names: MAb 35 and 35 [Barsov (1996), Bizub-Bender (1994)] • 35: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized – the Zn finger motif is in the binding region – MAbs 12, 13 and 35 form a competition group [Bizub-Bender (1994)] 				
225	3D12	Pol(dis)	RT(dis)		Vaccine	murine(IgG2a)
	Vaccine:	<i>Vector/type:</i> vaccinia		<i>HIV component:</i> RT		

		References: [Chiba (1997)]				
		• 3D12: There is an anti-Nef MAb that also has this name (see [Chiba (1997)])				
226	3F10	Pol(dis) RT(dis)			Vaccine	murine(IgG2a)
	Vaccine:	<i>Vector/type:</i> vaccinia <i>HIV component:</i> RT				
		References: [Chiba (1997)]				
227	4	Pol() Integrase(141–172)	no		Vaccine	murine(IgG2b)
	Vaccine:	<i>Vector/type:</i> recombinant protein <i>HIV component:</i> Integrase				
		References: [Bizub-Bender (1994), Levy-Mintz (1996)]				
		• 4: There is another MAb with this ID that reacts with gp41 [Oldstone (1991), Bizub-Bender (1994)]				
		• 4: BALBc mice were immunized with rec integrase, hybridomas expressing anti-integrase Abs were generated, and the antibodies characterized – 4 has a low binding affinity [Bizub-Bender (1994)]				
		• 4: Used for the creation of single chain variable antibody fragments (SFvs) for internal cellular expression – neutralization of Integrase activity occurs prior to integration, whether the Ab is expressed in the nucleolus or the cytoplasm – relative binding affinity to IN: 12 > 17 = 33 > 21 > 4 [Levy-Mintz (1996)]				
228	6B9	Pol(dis) RT(dis)			Vaccine	murine(IgG2a)
	Vaccine:	<i>Vector/type:</i> vaccinia <i>HIV component:</i> RT				
		References: [Chiba (1997)]				
229	7C4	Pol(dis) RT(dis)			Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> vaccinia <i>HIV component:</i> RT				
		References: [Chiba (1997)]				
		• 7C4: Dose-dependent inhibition of polymerase activity of RT of strains IIIB, Bru and IMS-1, but not HIV-2 strains GH-1 or LAV-2 or SIV strains MAC or MND [Chiba (1997)]				
230	anti-HIV-1 RT	Pol() RT()				murine(IgG)
		References: [di Marzo Veronese (1986), Maciejewski (1995), Wainberg & Gu(1995)]				
		• anti-HIV-1 RT: Cloned heavy and light chains to express Fab intracellularly, preventing HIV infection <i>in vitro</i> – this MAb was broadly cross-reactive with clinical strains and even HIV-2 [Maciejewski (1995)]				
		• Commentary on Maciejewski <i>et al.</i> [Wainberg & Gu(1995)]				
231	polyclonal	Pol() p55()	no		Vaccine	Rhesus macaque()
	Vaccine:	<i>Vector/type:</i> virus-like particle <i>HIV component:</i> Pr55gag, anchored gp120, V3+CD4 linear domains				
		References: [Wagner (1998)]				

Table of HIV MAbs

- A VLP is a non-infectious virus-like particle self-assembled from HIV Pr55 gag – macaques were immunized with VLPs bound to either gp120 or V3+CD4 linear domains – gag and env CTL specific CTL were stimulated in each case, and Ab response to gag and gp120 and was elicited, but the gp120 neutralizing response occurred only with whole gp120, not V3+CD4 – despite the CTL and Ab response, immunized macaques were infected by intervenous challenge with SHIV chimeric challenge stock [Wagner (1998)]

232	polyclonal	Pol()	RT()		Vaccine	murine()
	Vaccine:	<i>Vector/type:</i> DNA		<i>HIV component:</i> Gag, Pol, Vif, Env	<i>Stimulatory Agents:</i> B7, IL-12	
		References: [Kim (1997)]				
		<ul style="list-style-type: none"> • 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, as well as Ab response detected by ELISA 				
233	polyclonal	Pol()	RT(203–219)		Vaccine	murine(IgA)
	Vaccine:	<i>Vector/type:</i> Salmonella		<i>HIV component:</i> RT		
		References: [Burnett (2000)]				
		<ul style="list-style-type: none"> • A live attenuated bacterial vaccine, Salmonella SL3261-pHART, with an inserted HIV RT gene fragment in the Lpp-OmpA-HIV fusion protein, induced a lymphoproliferative Th response and fecal RT-specific IgA in BALB/c mice [Burnett (2000)] 				
234	RT-4	Pol()	RT()		no	murine(IgG2b)
		References: [Li (1993), Gu (1996)]				
		<ul style="list-style-type: none"> • RT-4: Increased nevirapine and delavirdine inhibition, no effect on AZT inhibition [Gu (1996)] 				
235	RT7O	Pol()	RT(231–315)		Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> RT		
		Donor: B. Ferns and R. Tedder				
		References: [Ferns (1991)]				
		<ul style="list-style-type: none"> • RT7O: Conformational epitope located centrally in the protein – inhibited RT enzyme activity and thus may bind close to the active site of the enzyme [Ferns (1991)] • RT7O: UK Medical Research Council AIDS reagent: ARP381 				
236	RT7U	Pol(dis)	RT(dis 231–315)		Vaccine	murine()
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> RT		
		Donor: B. Ferns and R. Tedder				
		References: [Ferns (1991)]				
		<ul style="list-style-type: none"> • RT7U: Has a conformational epitope – reacts with p66 and p51 in WB [Ferns (1991)] • RT7U: UK Medical Research Council AIDS reagent: ARP380 				

Table 9: **Vif**

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
237 TG002	Vif(34-47)	Vif(34-47)	KARGWFYRHHYESP?	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Vif</p> <p>Donor: Transgene</p> <ul style="list-style-type: none"> • TG002: This antibody was raised in response to a rec Vif protein derived from <i>E. coli</i> • TG002: NIH AIDS Research and Reference Reagent Program: 2746 						
238 TG001	Vif(176-192)	Vif(176-192)	KPQKTKGHRGSHTMNGH?	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Vif</p> <p>Ab type: C-term Donor: Transgene</p> <ul style="list-style-type: none"> • TG001: This antibody was raised in response to a rec Vif protein derived from <i>E. coli</i> • TG001: NIH AIDS Research and Reference Reagent Program: 2745 						
239 polyclonal	Vif()	Vif()			Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> DNA <i>HIV component:</i> Gag, Pol, Vif, Env <i>Stimulatory Agents:</i> B7, IL-12</p> <p>References: [Kim (1997)]</p> <ul style="list-style-type: none"> • 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, as well as an Ab response detected by ELISA 						

Table of HIV MAbs

Table 10: **Tat**

MAB ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
240 NT3/2D1.1	Tat(2–15)	Tat()	EPVDPNLEPWNHPS		Vaccine	murine(IgG1a)
<p><i>Vaccine:</i> Vector/type: peptide HIV component: Tat</p> <p>Ab type: N-term References: [Dingwall (1989)]</p> <ul style="list-style-type: none"> • NT3/2D1.1: Immunoprecipitates and immunoblots HIV-1 Tat protein [Dingwall (1989)] • NT3/2D1.1: UK Medical Research Council AIDS reagent: ARP352 						
241 1.2	Tat(2–17)	Tat(1–16)	EPVDPRLEWKHPGSQ			()
<p>References: [Ovod (1992), Ranki (1995)]</p> <ul style="list-style-type: none"> • 1.2: Weak expression of Tat observed in HIV+ brain tissue sample, in contrast to Nef [Ranki (1995)] 						
242 1D9D5	Tat(2–21)	Tat()	EPVDPRLEWKHPGSQPKTA		Vaccine	murine(IgG1)
<p><i>Vaccine:</i> Vector/type: recombinant protein HIV component: Tat</p> <p>Ab type: N-term References: [Mhashilkar (1995), Valvatne (1996)]</p> <ul style="list-style-type: none"> • 1D9D5: Single chain antibodies, intrabodies, were engineered that can be stably expressed in the cytoplasm of mammalian cells – co-expression of an N-term Tat intrabody can inhibit transactivation of an HIV LTR-CAT construct and block import into nucleus, but intrabody specific for exon 2 did not inhibit activity [Mhashilkar (1995)] • 1D9D5: Exogenously delivered Tat can efficiently transactivate an HIV-LTR-CAT construct in HeLa cells in the presence of 1D9D5, suggesting when considered with the results of [Mhashilkar (1995)], that free Tat and not Ab bound is taken up by cells [Valvatne (1996)] 						
243 1D2F11	Tat(dis 49–86)	Tat(dis)	RKKRRQRRRPPQGSQTHQVSL-SKQPTSQSRGDPTGPKE		Vaccine	murine(IgG1)
<p><i>Vaccine:</i> Vector/type: recombinant protein HIV component: Tat</p> <p>Ab type: C-term References: [Valvatne (1996)]</p> <ul style="list-style-type: none"> • 1D2F11: MAb did not bind shorter peptides – this MAb inhibited exogenously delivered Tat transactivation of an HIV-LTR-CAT construct in HeLa cells by inhibition of cellular uptake of Tat [Valvatne (1996)] 						
244 2D9E7	Tat(49–86)	Tat()	RKKRRQRRRPPQGSQTHQVSL-SKQPTSQSRGDPTGPKE		Vaccine	murine(IgG1)
<p><i>Vaccine:</i> Vector/type: recombinant protein HIV component: Tat</p> <p>Ab type: C-term References: [Valvatne (1996)]</p> <ul style="list-style-type: none"> • 2D9E7: MAb did not bind shorter peptides – this MAb inhibited exogenously delivered Tat transactivation of an HIV-LTR-CAT construct in HeLa cells by inhibition of cellular uptake of Tat, but less efficiently than MAbs 1D2F11 or 4B4C4 [Valvatne (1996)] 						

Table of HIV MAbs

245	4B4C4 (4B4)	Tat(49–86)	Tat()	RKKRRQRRRPPQGSQTHQVSL- SKQPTSQSRGDPTGPKE	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Tat Ab type: C-term References: [Valvatne (1996), Jensen (1997)] <ul style="list-style-type: none"> • 4B4C4: MAb did not bind shorter peptides – this MAb inhibited exogenously delivered Tat transactivation of an HIV-LTR-CAT construct in HeLa cells by inhibition of cellular uptake of Tat [Valvatne (1996)] </p>						
246	5G7D8	Tat(49–86)	Tat()	RKKRRQRRRPPQGSQTHQVSL- SKQPTSQSRGDPTGPKE	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Tat Ab type: C-term References: [Valvatne (1996)] <ul style="list-style-type: none"> • 5G7D8: MAb did not bind shorter peptides – this MAb inhibited exogenously delivered Tat transactivation of an HIV-LTR-CAT construct in HeLa cells by inhibition of cellular uptake of Tat, but less efficiently than 1D2F11 or 4B4C4 [Valvatne (1996)] </p>						
247	NT2/4D5.24	Tat(73–86)	Tat()	PTSQPRGDPTGPKE	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> Tat Ab type: C-term References: [Dingwall (1989)] <ul style="list-style-type: none"> • NT2/4D5.24: Immunoprecipitates and immunoblots HIV-1 Tat protein [Dingwall (1989)] </p>						
248	L-anti-Tat	Tat()	Tat()	L P (when lipidated)	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Tat Donor: AGMED, Inc., Bedford, MA USA References: [Cruikshank (1997)] <ul style="list-style-type: none"> • L-anti-Tat: Lipidated antibody can be taken up by cells and effectively block IIIB and primary virus HIV-1 replication in actively and latently infected cells [Cruikshank (1997)] </p>						
249	2D9D5	Tat()	Tat()		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Tat Ab type: C-term References: [Mhashilkar (1995)] <ul style="list-style-type: none"> • 2D9D5: Single chain antibodies, intrabodies, were engineered that can be stably expressed in the cytoplasm of mammalian cells – co-expression of C-term intrabody did not inhibit transactivation of an HIV LTR-CAT construct, in contrast to MAb 1D9D5 [Mhashilkar (1995)] </p>						

B Cell

Table of HIV MAbs

Table 11: **Rev**

MAB ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
250 4G9	Rev(5–15)	Rev(5–15)	SGDSDEELIRT?		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Rev</p> <p>References: [Jensen (1997)]</p> <ul style="list-style-type: none"> • 4G9: Mapped binding location by protein footprinting [Jensen (1997)] 						
251 Ab2	Rev(32–50)	Rev(32–49 BRU)	EGTRQARRNRRRWREERQR		Vaccine	(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Rev</p> <p>Donor: Tony Lowe and Jonathan Karn, MRC Center, Cambridge</p> <p>References: [Henderson & Percipalle(1997)]</p> <ul style="list-style-type: none"> • Ab2: The Ab2 binding site overlaps the nuclear localization signal – Ab2 binding to Rev was blocked by bound HIV RNA – the cellular protein importin-β can bind in this Arg rich region – atypically, the Rev binds specifically to importin-β, but not to the importin-β-importin-alpha dimer [Henderson & Percipalle(1997)] 						
252 10.1	Rev(33–48)	Rev(33–48)	GTRQARRNRRRWREER?			()
<p>References: [Ovod (1992), Ranki (1994), Ranki (1995)]</p> <ul style="list-style-type: none"> • 10.1: Binds to the RRE – polyclonal anti-Rev Ab detected Rev in astrocytes in 4/5 brain autopsy samples, but only one of these was positive using 10.1, suggesting most Rev was bound to RRE [Ranki (1995)] 						
253 3H6	Rev(38–43)	Rev(38–44)	RRNRRR		Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Rev</p> <p>References: [Orsini (1995)]</p> <ul style="list-style-type: none"> • 3H6: There is another MAB with this ID that recognizes gp41 [Pinter (1995)] • 3H6: Directed against nucleolar localization/RRE binding domain – antigenic domain tentative, MAb failed to bind a RRNRRR Rev deletion mutant [Orsini (1995)] 						
254 8E7	Rev(70–84)	Rev(70–84)	PVPLQLPPLERLTLTD		Vaccine	murine(IgG2a κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Rev</p> <p>References: [Kalland (1994a), Kalland (1994b), Szilvay (1995), Jensen (1997), Boe (1998)]</p> <ul style="list-style-type: none"> • 8E7: 8E7 worked in indirect immunofluorescence and also detected Rev in WB assays – used to detect localization of Rev in several compartments including the nucleoli, nucleoplasm, perinuclear zone, and cytoplasm – Rev co-localized with host cell factors known to assemble on nascent transcripts – Rev shuttles continuously between cytoplasmic and nucleoplasmic compartments [Kalland (1994a), Kalland (1994b), Szilvay (1995)] • 8E7: Peptide interaction mapped to aa 70–84, 75–88 – protein footprint to 65–88 [Jensen (1997)] 						

- 8E7: HIV-1 RNA and Rev localize to the same region in the nucleoplasm, but the splicing factor SC-35 localizes in different speckles with the nucleoplasm than Rev – intron containing β -globin was distributed similarly to HIV-1, suggesting Rev and HIV-1 RNAs interact at putative sites of mRNA transcriptions and splicing [Boe (1998)]

255	9G2 (9G2G4D6E8:)	Rev(70–84)	Rev(70–84)	PVPLQLPPLERLTLD	Vaccine	murine(IgG2a κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Rev</p> <p>Donor: Anne Marie Szilvay</p> <p>References: [Kalland (1994a), Jensen (1997)]</p> <ul style="list-style-type: none"> • 9G2: Worked in indirect immunofluorescence and also detected Rev in WB assays – used to detect localization of Rev throughout the cell [Kalland (1994a)] • 9G2: Peptide interaction mapped to aa 70–84, 75–88 – protein footprint to 65–88 [Jensen (1997)] • 9G2: Called 9G2G4D6E8: UK Medical Research Council AIDS reagent: ARP3058 						
256	Ab4	Rev(72–91)	Rev(72–91 BRU)	PLQLPPLERLTLDNCNEDCGT	Vaccine	(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Rev</p> <p>Donor: Tony Lowe and Jonathan Karn, MRC Center, Cambridge</p> <p>References: [Henderson & Percipalle(1997)]</p> <ul style="list-style-type: none"> • Ab4: The binding site overlaps the nuclear export signal – binding was not blocked by bound HIV RNA and may be accessible for protein interaction [Henderson & Percipalle(1997)] 						
257	3G4	Rev(90–116)	Rev(90–116)	TSGTQGVGSPQILVESPTVLE-SGTKE?	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Rev</p> <p>References: [Orsini (1995)]</p> <ul style="list-style-type: none"> • 3G4: Binds to a region that can be dispensed with and still retain Rev function [Orsini (1995)] 						
258	1G10 (IG10F4)	Rev(96–105)	Rev(95–105)	GVGSPQILVE	Vaccine	murine(IgG2b κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Rev</p> <p>Donor: Anne Marie Szilvay</p> <p>References: [Kalland (1994a)]</p> <ul style="list-style-type: none"> • 1G10: Bound Rev in indirect immunofluorescence and also detected Rev in WB – used to detect localization of Rev throughout the cell [Kalland (1994a)] • 1G10: Peptide interaction mapped to aa 91–105, 96–110 and a protein footprint to aa 10–20, and 95–105 [Jensen (1997)] • 1G10: Called IG10F4: UK Medical Research Council AIDS reagent: ARP3060 						

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259	1G7	Rev(96–105)	Rev(95–105)	GVGSPQILVE	Vaccine	murine(IgG2b κ)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Rev		
		References: [Kalland (1994a), Jensen (1997)]				
		<ul style="list-style-type: none"> • 1G7: Worked in indirect immunofluorescence and also detected Rev in WB – used to detect localization of Rev throughout the cell [Kalland (1994a)] • 1G7: Peptide interaction mapped to aa 91–105, 96–110 and a protein footprint to aa 95–105 [Jensen (1997)] 				
260	Ab3	Rev(102–116)	Rev(102–116 BRU)	ILVESPTVLESDKTE	Vaccine	(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Rev		
		Donor: Tony Lowe and Jonathan Karn, MRC, Cambridge				
		References: [Henderson & Percipalle(1997)]				
		<ul style="list-style-type: none"> • Ab3: This binding site is at the carboxy end of Rev – Ab3 binding was not blocked by bound HIV RNA [Henderson & Percipalle(1997)] 				
261	2G2	Rev(dis)	Rev(dis)		Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Rev		
		References: [Orsini (1995)]				
		<ul style="list-style-type: none"> • 2G2: Does not bind to any of a set of glutathione S-transferase (GST) Rev fusion proteins, or to Rev in a RIPA buffer, suggesting a conformational epitope [Orsini (1995)] 				

Table 12: gp160

MAb ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
262 M85	gp160(30–51)	gp120(30–51 LAI)	ATEKLWVTVYYGVPVWKEAT-TT	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> Env</p> <p>Ab type: C1 Donor: Fulvia di Marzo Veronese</p> <p>References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Ditzel (1997), Wyatt (1997)]</p> <ul style="list-style-type: none"> • M85: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese (1992)] • M85: C1 domain – mutation 40 Y/D impairs binding – the relative affinity for denatured/native gp120 is < .01, suggesting conformational component [Moore (1994c)] • M85: Binding inhibited by MAb 4D4#85, enhanced by conformationally sensitive anti-V3 MAb 5G11, and some anti-18 MAbs [Moore & Sodroski(1996)] • M85: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt (1997)] 						
263 7E2/4	gp160(31–50)	gp120(31–50 LAI)	TEKLWVTVYYGVPVWKEATT		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Env</p> <p>Ab type: C1 Donor: S. Ranjbar, NIBSC, UK</p> <p>References: [Moore (1994c)]</p> <ul style="list-style-type: none"> • 7E2/4: C1 domain – the relative affinity for denatured/native gp120 is .07, suggesting conformational component [Moore (1994c)] • 7E2/4: UK Medical Research Council AIDS reagent: ARP3050 						
264 4D4#85	gp160(41–50)	gp120(LAI)	GVPVWKEATT		Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C1 Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA</p> <p>References: [Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Wyatt (1997), Binley (1998)]</p> <ul style="list-style-type: none"> • 4D4#85: C1 domain – the relative affinity, denatured/native gp120 is 0.1 – mutation 45 W/S impairs binding [Moore (1994c)] • 4D4#85: Inhibits binding of C1 MAb M85, C1-C5 discontinuous epitope MAbs 181 and 212A, and CD4 binding induced MAbs 48d and 17b [Moore & Sodroski(1996)] • 4D4#85: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31–50, are deleted [Wyatt (1997)] • 4D4#85: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)] 						

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265	M92	gp160(41–50)	gp120(31–50 LAI)	GVPVWKEATT	no	Vaccine	rat(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> Env</p> <p>Ab type: C1 Donor: Fulvia di Marzo Veronese</p> <p>References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d)]</p> <ul style="list-style-type: none"> • M92: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)] • M92: The relative affinity for denatured/native gp120 is 1 [Moore (1994c)] 							
266	M86	gp160(42–61)	gp120(42–61 LAI)	VPVWKEATTTLFCASDAKAY	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> Env</p> <p>Ab type: C1 Donor: Fulvia di Marzo Veronese</p> <p>References: [di Marzo Veronese (1992), Moore (1994c)]</p> <ul style="list-style-type: none"> • M86: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese (1992)] • M86: C1 domain – the relative affinity for denatured/native gp120 is 1 [Moore (1994c)] 							
267	polyclonal	gp160(51–70)	Env(42–61 LAI)	LFCASDAKAYDTEVHNVWAT	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>HIV component:</i> Env</p> <p>Ab type: C1 References: [Collado (2000)]</p> <ul style="list-style-type: none"> • Vaccinia p14 can elicit NAb and p39 tends to be immunodominant, so these two proteins were fused to regions of HIV-1 Env – reduced glycosylation was noted when p14 or p39 was placed in the N-term region of the fusion protein – chimeric proteins shifted the Env Ab response from V3 to either a C1 or C4 domain, depending on the construct – all chimeric Env proteins: 14kEnv, 39kEnv, and Env39k elicited a strong Ab response to the C1 region of gp120 (LFCASDAKAYDTEVHNVWAT), and Env39k mounted a strong response to the C4 region (KAMYAPPISGQIRCSSNITG) [Collado (2000)] 							
268	133/237	gp160(61–70)	gp120(51–70 LAI)	YDTEVHNVWA	L	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [Niedrig (1992b), Moore (1994c), Moore (1994d)]</p> <ul style="list-style-type: none"> • 133/237: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig (1992b)] • 133/237: The relative affinity, denatured/native gp120 is 1.4 – mutation of position 69 W/L impairs binding [Moore (1994c)] 							
269	133/290	gp160(61–70)	gp120(61–70 LAI)	YDTEVHNVWA	L	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [Niedrig (1992b), Thali (1993), Moore (1994c), Moore (1994d), Wyatt (1995), Binley (1997a), Wyatt (1997), Binley (1998)]</p> <ul style="list-style-type: none"> • 133/290: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig (1992b)] • 133/290: The relative affinity for denatured/native gp120 is 2.2 – mutation in position 69 W/L impairs binding [Moore (1994c)] 							

- 133/290: Used for antigen capture assay, either to bind gp120 to the ELISA plate, or to quantify bound gp120 [Wyatt (1995)]
- 133/290: Reciprocal binding inhibition with the antibody 522–149, that binds to a discontinuous epitope – binding is enhanced by some C5 and C1 binding site antibodies [Moore & Sodroski(1996)]
- 133/290: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt (1997)]
- 133/290: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

270	133/11	gp160(64–78)	gp120(64–78)	EVHNVWATHACVPTD	L	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [Niedrig (1992b)]</p> <ul style="list-style-type: none"> • 133/11: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig (1992b)] 							
271	D/3G5	gp160(73–82)	gp120(73–82 LAI)	ACVPTDPNPQ	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [Bristow (1994)]</p> <ul style="list-style-type: none"> • D/3G5: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)] 							
272	D/6A11	gp160(73–82)	gp120(73–82 LAI)	ACVPTDPNPQ	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [Bristow (1994)]</p> <ul style="list-style-type: none"> • D/6A11: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)] 							
273	D/5E12	gp160(73–92)	gp120(73–92 LAI)	ACVPTDPNPQEVVLNVNVTEN	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [Bristow (1994)]</p> <ul style="list-style-type: none"> • D/5E12: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)] 							
274	L5.1	gp160(79–93)	gp120(89–103 IIIB)	PNPQEVVLNVNVTENF		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> gp160</p> <p>Ab type: C1 References: [Akerblom (1990)]</p>							

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275	4A7C6	gp160(81–90)	gp120(81–90 LAI)	PQEVVLVNVNT		Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>HIV component:</i> Env			
		Ab type: C1		Donor: R. Tedder			
		References: [Thiriart (1989), Thali (1993), Moore & Ho(1993), Moore (1994c), Moore (1994d), Moore & Sodroski(1996)]					
		<ul style="list-style-type: none"> • 4A7C6: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • 4A7C6: The relative affinity for denatured/native gp120 is 7.9 – mutation 88 N/P impairs binding [Moore (1994c)] • 4A7C6: C1 region epitope (88 N/P substitutions abrogates binding), but substitutions 380 G/F and 420 I/R also impaired binding [Moore (1994d)] • 4A7C6: Reciprocal binding inhibition with the antibody 133/192 – enhanced by anti-C5 antibodies, and C1 antibody 135/9[Moore & Sodroski(1996)] • 4A7C6: UK Medical Research Council AIDS reagent: ARP 360 					
276	1D10	gp160(81–100)	gp120(81–100 LAI)	PQEVVLVNVNTENFDMWKNDM	L	Vaccine	rat()
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>Strain:</i> IIIB		<i>HIV component:</i> gp120	
		Ab type: C1		References: [Dowbenko (1988), Berman (1991), Nakamura (1992), Moore (1994c)]			
		<ul style="list-style-type: none"> • 1D10: Cross-blocks 5B3 in IIIB-rsgp160 ELISA – type specific in rgp120 ELISA binding [Nakamura (1992)] • 1D10: The relative affinity for denatured/native gp120 is 13 – mutation 88 N/P impairs binding [Moore (1994c)] 					
277	B242	gp160(83–92)	gp120(83–92 LAI)	EVVLVNVNTEN		no Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>Strain:</i> NL43		<i>HIV component:</i> gp160	
		Ab type: C1		References: [Bristow (1994)]			
		<ul style="list-style-type: none"> • B242: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys [Bristow (1994)] 					
278	133/192	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	L	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> protein		<i>Strain:</i> IIIB		<i>HIV component:</i> gp120	
		Ab type: C1		Donor: Matthias Niedrig			
		References: [Niedrig (1992b), Moore (1993b), Moore (1994c), Moore & Sodroski(1996), Trkola (1996a), Binley (1997a), Binley (1998)]					
		<ul style="list-style-type: none"> • 133/192: Epitope seems complex, binds multiple peptides – weak neutralization of lab strain [Niedrig (1992b)] • 133/192: The relative affinity for denatured/native gp120 is 1.8 [Moore (1994c)] • 133/192: C1 region – substitutions 76P/Y, 113 D/A or R, 117 K/W, 420 I/R, 427 W/S impair binding, other substitutions enhanced binding [Moore (1994d)] • 133/192: Reciprocal binding inhibition with the antibody 4A7C6 – enhanced by some anti-C5 and-C1 antibodies [Moore & Sodroski(1996)] • 133/192: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)] 					

- 133/192: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

279	489.1(961)	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	Vaccine	murine(IgG)
Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env						
Ab type: C1 Donor: C. Bruck, SKB, Belgium						
References: [Moore (1994c)]						
<ul style="list-style-type: none"> • 489.1(961): C1 region – The relative affinity for denatured/native gp120 is 1 [Moore (1994c)] • 489.1(961): NIH AIDS Research and Reference Reagent Program: 961 						
280	5B3	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	no Vaccine	murine(IgG)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> gp160						
Ab type: C1 References: [Berman (1991), Nakamura (1992), Beretta & Dalgleish(1994), Moore (1994c)]						
<ul style="list-style-type: none"> • 5B3: Blocks gp120 -CD4 binding [Berman (1991)] • 5B3: Cross-blocks 1D10 in competitive IIIB-rsgp160 ELISA – no neutralization – blocks IIIB-gp120 sCD4 binding – localized binding to residues 72–106 [Nakamura (1992)] • 5B3: The relative affinity of denatured/native gp120 is 8.3 [Moore (1994c)] 						
281	B10	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	Vaccine	murine(IgG1)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160						
Ab type: C1 References: [Abacioglu (1994), Moore (1994c)]						
<ul style="list-style-type: none"> • B10: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu (1994)] • B10: The relative affinity for denatured/native gp120 is 0.4 [Moore (1994c)] • B10: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.) 						
282	B2	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	Vaccine	murine(IgG2b)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160						
Ab type: C1 References: [Thali (1993), Abacioglu (1994), Moore (1994c), Moore (1994d), Binley (1997a)]						
<ul style="list-style-type: none"> • B2: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu (1994)] • B2: The relative affinity for denatured/native gp120 is 1.4 [Moore (1994c)] • B2: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.) 						
283	C6 (Ch6)	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	Vaccine	murine(IgG1)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160						
Ab type: C1 References: [Pincus & McClure(1993), Abacioglu (1994), Moore (1994c), Pincus (1996)]						
<ul style="list-style-type: none"> • C6: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu (1994)] • C6: The relative affinity for denatured/native gp120 is 0.9 [Moore (1994c)] 						

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- C6: There is FNM/FDM polymorphism in LAI-based peptides – N is essential (J. P. Moore, per. comm.)
- C6: Called Ch6 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus (1996)]
- C6: NIH AIDS Research and Reference Reagent Program: 810

284	MF49.1	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C1 References: [Thiriart (1989), Moore (1994c)]</p> <ul style="list-style-type: none"> • MF49.1: The relative affinity of denatured/native gp120 is 3.8 [Moore (1994c)] 						
285	T1.1	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>HIV component:</i> gp160</p> <p>Ab type: C1 References: [Akerblom (1990), Broliden (1990), Moore (1994c)]</p> <ul style="list-style-type: none"> • T1.1: Also reacted in solid phase with gp120(234–248) NGTGPCTNVSTQCT [Akerblom (1990)] • T1.1: No ADCC activity – reactive peptide: NVTENFNMWKNDMVEQ, IIIB [Broliden (1990)] • T1.1: C1 region – the relative affinity for denatured/native gp120 is 1 [Moore (1994c)] 						
286	T7.1	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C1 References: [Akerblom (1990), Bolmstedt (1990), Moore (1994c), Moore (1994d)]</p> <ul style="list-style-type: none"> • T7.1: The relative affinity of denatured/native gp120 is 4.0 [Moore (1994c)] 						
287	T9	gp160(91–100)	gp120(91–100 LAI)	ENFDMWKNDM	Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C1 Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula</p> <p>References: [Akerblom (1990), Bolmstedt (1990), Moore (1994c), Moore (1994d), Binley (1997a)]</p> <ul style="list-style-type: none"> • T9: The relative affinity of denatured/native gp120 is 7.9 [Moore (1994c)] • T9: C1 region – 45 W/S, 88 N/P, 256 S/Y, 262 N/T, 475 M/S, 485 1.83, and 491 I/F enhanced binding, no substitution tested significantly inhibited [Moore (1994d)] 						
288	GV4D3	gp160(92–100)	gp120(92–100 IIIB)	NFNMWKNDM	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> protein-Ab complex <i>HIV component:</i> gp120 complexed with MAb M77</p> <p>Ab type: C1 References: [Denisova (1996)]</p> <ul style="list-style-type: none"> • GV4D3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV4H4 and GV5F9 are homologous to GV4D3 and were generated in the same experiment [Denisova (1996)] 						

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289	B27	gp160(93–96)	gp120(94–97 BH10)	FNMW	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> NL43 <i>HIV component:</i> gp160</p> <p>Ab type: C1 References: [Abacioglu (1994), Bristow (1994)]</p> <ul style="list-style-type: none"> • B27: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)] • B27: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIB:NL43, MicroGenSys [Bristow (1994)] 						
290	B9	gp160(93–96)	gp120(93–96 LAI)	FNMW	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p> <p>Ab type: C1 References: [Abacioglu (1994)]</p> <ul style="list-style-type: none"> • B9: Binds C1 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)] 						
291	B35	gp160(93–98)	gp120(94–99 BH10)	FNMWKN	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p> <p>Ab type: C1 References: [Abacioglu (1994)]</p> <ul style="list-style-type: none"> • B35: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)] 						
292	D/4B5	gp160(93–101)	gp120(93–101 LAI)	FNMWKNDMV	no Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [Bristow (1994)]</p> <ul style="list-style-type: none"> • D/4B5: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)] 						
293	D/5A11	gp160(93–101)	gp120(93–101 LAI)	FNMWKNDMV	no Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [Bristow (1994)]</p> <ul style="list-style-type: none"> • D/5A11: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)] 						
294	D/6B2	gp160(93–101)	gp120(93–101 LAI)	FNMWKNDMV	no Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [Bristow (1994)]</p> <ul style="list-style-type: none"> • D/6B2: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)] 						
295	B18	gp160(101–110)	gp120(101–110 LAI)	VEQMHEDIIS	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p>						

- 37.1.1: The relative affinity for denatured/native gp120 is 8.6 – mutations 113 D/R (not D/A) and 117 K/W impair binding [Moore (1994c)]
- 37.1.1: UK Medical Research Council AIDS reagent: ARP327

300	6D8	gp160(101–120)	gp120(101–120 LAI)	VEQMHEDIISLWDQSLKPCV	Vaccine	rat()
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120						
Ab type: C1 References: [Dowbenko (1988), Nakamura (1992), Moore (1994c)]						
• 6D8: Highly cross-reactive with multiple stains by rgp120 ELISA [Nakamura (1992)]						
• 6D8: The relative affinity for denatured/native gp120 is 15 – mutations 113 D/R and 113 D/A impair binding [Moore (1994c)]						
301	M96	gp160(101–120)	gp120(101–120 LAI)	VEQMHEDIISLWDQSLKPCV	no Vaccine	rat(IgG2a)
Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> Env						
Ab type: C1 Donor: Fulvia di Marzo Veronese						
References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d)]						
• M96: Immunoblot reactive for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)]						
• M96: C1 region – the relative affinity for denatured/native gp120 is 6 [Moore (1994c)]						
302	MF119.1	gp160(101–120)	gp120(101–120 LAI)	VEQMHEDIISLWDQSLKPCV	Vaccine	murine(IgG)
Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env						
Ab type: C1 References: [Thiriart (1989), Moore (1994c)]						
• MF119.1: The relative affinity for denatured/native gp120 is 30 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore (1994c)]						
303	MF4.1	gp160(101–120)	gp120(101–120 LAI)	VEQMHEDIISLWDQSLKPCV	Vaccine	murine(IgG)
Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env						
Ab type: C1 References: [Thiriart (1989), Moore (1994c)]						
• MF4.1: The relative affinity for denatured/native gp120 is 8 [Moore (1994c)]						
304	MF53.1	gp160(101–120)	gp120(101–120 LAI)	VEQMHEDIISLWDQSLKPCV	Vaccine	murine(IgG)
Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env						
Ab type: C1 References: [Thiriart (1989), Moore (1994c)]						
• MF53.1: The relative affinity for denatured/native gp120 is 10 [Moore (1994c)]						
305	MF58.1	gp160(101–120)	gp120(101–120 LAI)	VEQMHEDIISLWDQSLKPCV	Vaccine	murine(IgG)
Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env						
Ab type: C1 References: [Thiriart (1989), Moore (1994c)]						

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306	MF77.1	gp160(101–120)	gp120(101–120 LAI)	VEQMHEDIISLWDQSLKPCV	Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C1 References: [Thiriart (1989), Moore (1994c)]</p> <ul style="list-style-type: none"> • MF77.1: The relative affinity for denatured/native gp120 is 11 [Moore (1994c)] 						
307	T2.1	gp160(101–120)	gp120(101–120 LAI)	VEQMHEDIISLWDQSLKPCV	Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C1 Donor: Lennart Akerblom, Britta Wahren and Jorma Hinkula</p> <p>References: [Akerblom (1990), Bolmstedt (1990), Moore (1994c), Moore (1994d)]</p> <ul style="list-style-type: none"> • T2.1: The relative affinity for denatured/native gp120 is .27 – mutations 113 D/R, 106 E/A, and 117 D/A impair binding [Moore (1994c)] 						
308	11/65 (11/65a/5h)	gp160(dis 102–121)	gp120(dis 311–321 HXB10)	EQMHEDIISLWDQSLKPCVK	Vaccine	rat(IgG2b)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>Ab type: C1 References: [McKeating (1992a), McKeating (1993b), Peet (1998)]</p> <ul style="list-style-type: none"> • 11/65: Binds only soluble gp120, not virion bound – used to quantify gp120 shedding – (numbering is incorrect in original?) [McKeating (1992a)] • 11/65: Called 11/65a/5h – The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – 11/65 was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)] • 11/65: UK Medical Research Council AIDS reagent: ARP3076 						
309	W1	gp160(102–121)	gp120(102–121 LAI)	EQMHEDIISLWDQSLKPCVK	Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C1 Donor: D. Weiner, U. Penn.</p> <p>References: [Moore (1994c)]</p> <ul style="list-style-type: none"> • W1: The relative affinity for denatured/native gp120 is 6 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore (1994c)] 						
310	T11	gp160(102–125)	gp120(102–125)	EQMHEDIISLWDQSLKPCVKL-TPL	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: C1 Donor: R. Doms, Univ. of Pennsylvania</p> <p>References: [Earl (1994), Jagodzinski (1996)]</p> <ul style="list-style-type: none"> • T11: Generated during a study of the influence of the oligomeric structure of Env in determining the repertoire of the Ab response – an oligomer with no gp120/gp41 cleavage site was used as the immunogen [Earl (1994)] 						

- T11: The sulfated polysaccharide, curdlan sulfate (CRDS), binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop from gp120 results in more potent T11 inhibition by CRDS [Jagodzinski (1996)]

311	GV1A8	gp160(105–113)	gp120(105–113 IIIB)	HEDIISLWD		Vaccine	murine()
	Vaccine:	<i>Vector/type:</i> protein-Ab complex		<i>HIV component:</i> gp120 complexed with MAb M77			
		Ab type: C1		References: [Denisova (1996)]			
		<ul style="list-style-type: none"> • GV1A8: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV7A4 and GV5H5 are homologous to GV1A8 and were generated in the same experiment [Denisova (1996)] 					
312	11	gp160(111–120)	gp120(101–120 LAI)	LWDQSLKPCV		Vaccine	murine(IgG)
	Vaccine:	<i>Strain:</i> LAI		<i>HIV component:</i> Env			
		Ab type: C1		References: [Thiriart (1989), Moore (1994c)]			
		<ul style="list-style-type: none"> • 11: The relative affinity for denatured/native gp120 is 7.8 – mutation 113 D/R impairs binding [Moore (1994c)] 					
313	12G10	gp160(111–120)	gp120(101–120 LAI)	LWDQSLKPCV		Vaccine	murine(IgG)
	Vaccine:	<i>Strain:</i> LAI		<i>HIV component:</i> Env			
		Ab type: C1		References: [Thiriart (1989), Moore (1994c)]			
		<ul style="list-style-type: none"> • 12G10: The relative affinity for denatured/native gp120 is 17 – mutation 117 K/W impairs binding [Moore (1994c)] 					
314	135/9 (87–135/9)	gp160(111–120)	gp120(111–120 LAI)	LWDQSLKPCV	L	Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> protein	<i>Strain:</i> IIIB	<i>HIV component:</i> gp120			
		Ab type: C1		Donor: Matthias Niedrig			
		References: [Niedrig (1992b), Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Trkola (1996a), Binley (1997a), Kropelin (1998)]					
		<ul style="list-style-type: none"> • 135/9: Defines the epitope as gp120(114–123) MHEDIISLWD (core LWD?) – weak neutralization of lab strain [Niedrig (1992b)] • 135/9: The relative affinity for denatured/native gp120 is 15 – mutation 113 D/R impairs binding to native and denatured, 113 D/A only to denatured [Moore (1994c)] • 135/9: Substitutions 106 E/A, 113 D/A or R, and 117 K/W impair binding, some substitutions enhance binding [Moore (1994d)] • 135/9: Binding is enhanced by some anti-C1 and anti-C5 antibodies – enhances binding of some anti-V3, anti-C4 and anti-V2 MAbs – 135/9 binds to predicted alpha-helix in C1 [Moore & Sodroski(1996)] • 135/9: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)] • 135/9: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)] • 135/9: Noted to bind to C1 peptide HEDIISLWDQSLK – blocks gp120 interaction with CD4+ cells – blocking activity is additive when combined with antibodies which bind in the C4 region of gp120 (F105, 388/389, and b12) [Kropelin (1998)] 					

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315	7C10	gp160(111–120)	gp120(101–120 LAI)	LWDQSLKPCV		Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C1 References: [Thiriart (1989), Moore (1994c)]</p> <ul style="list-style-type: none"> • 7C10: The relative affinity for denatured/native gp120 is 5.8 – mutation 117 K/W impairs binding [Moore (1994c)] 							
316	C4	gp160(111–120)	gp120(101–120 LAI)	LWDQSLKPCV		Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p> <p>Ab type: C1 Donor: George Lewis</p> <p>References: [Abacioglu (1994), Moore & Ho(1993), Moore (1994c)]</p> <ul style="list-style-type: none"> • C4: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • C4: C1 region – epitope boundaries mapped by peptide scanning, BH10 core IISLW [Abacioglu (1994)] • C4: The relative affinity for denatured/native gp120 is 10 [Moore (1994c)] 							
317	MF46.1	gp160(111–120)	gp120(101–120 LAI)	LWDQSLKPCV		Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C1 References: [Thiriart (1989), Moore (1994c)]</p> <ul style="list-style-type: none"> • MF46.1: The relative affinity for denatured/native gp120 is 8.5 [Moore (1994c)] 							
318	6D5	gp160(122–141)	gp120(122–141 LAI)	LTPLCVSLKCTDLKNDTNTN		Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: V2 Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA</p> <p>References: [Moore (1994c), Moore (1994d)]</p> <ul style="list-style-type: none"> • 6D5: The relative affinity for denatured/native gp120 is 15 – mutations Δ119–205 and 125 L/G impair binding [Moore (1994c)] 							
319	B33	gp160(123–142)	gp120(123–142 LAI)	TPLCVSLKCTDLGNATNTNS	no	Vaccine	murine(IgG2b κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> NL43 <i>HIV component:</i> gp160</p> <p>Ab type: V2 Donor: Daniels</p> <p>References: [Abacioglu (1994), Bristow (1994)]</p> <ul style="list-style-type: none"> • B33: There are two MAbs in the literature named B33, see also gp160(727–734) [Abacioglu (1994)] • B33: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)] • B27: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys [Bristow (1994)] • B33: UK Medical Research Council AIDS reagent: ARP304, gp160/41 binding 							
320	polyclonal (VEI1)	gp160(131–151)	Env(131–151)	CTDLKNDTNTNSSSGRMMME-K		HIV-1 infection	human()
<p>References: [Carlos (1999)]</p>							

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

321	2H1B	gp160(155–161)	gp120(370–376 HIV2ROD)	RNISFKA	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> HIV-2 ROD</p> <p>Ab type: C3 References: [Matsushita (1995)]</p> <ul style="list-style-type: none"> • 2H1B: Binds in WB, but binds poorly to Env on the cell surface [Matsushita (1995)] 							
322	697-D (697D, 697-30D, 697/30D)	gp160(dis 161– 180)	gp120(dis 161–180 IIIB)	ISTSIRGKVQKEYAFFYKLD	P (weak)	HIV-1 infection	human(IgG1λ)
<p>Ab type: V2 Donor: Susan Zolla-Pazner (Zollas01@mcr6.med.nyu) (NYU Med. Center) or Cellular Products Inc, Buffalo NY</p> <p>References: [Gorny (1994), Forthal (1995), Moore & Ho(1995), Trkola (1996a), Binley (1997a), Fouts (1997), Parren (1997b), Nyambi (1998), Stamatatos & Cheng-Mayer(1998), Gorny (2000), Hioe (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 697-D: Conformational with weak reactivity to V2 peptide ISTSIRGKVQKEYAFFYKLD – neutralized 3/4 primary isolates, but none of 4 lab strains – V2 substitutions 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192–194 YSL/GSS abrogate binding – anti-C4 MAbs G3-536 and G45–60 enhance binding – mild oxidation of carbohydrate moieties inhibits binding [Gorny (1994)] • 697-D: Not neutralizing, no ADCC activity, and no viral enhancing activity [Forthal (1995)] • 697-D: Review: called 697/30D – neutralizes some primary, but not lab adapted strains [Moore & Ho(1995)] • 697-D: Partial inhibition of gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)] • 697-D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 697-D bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)] • 697-D: Does not neutralize TCLA strains but neutralizes some primary isolates weakly [Parren (1997b)] • 697-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – V2 Abs 697-D, 1361, and 1357 tended to bind weakly with a similar pattern of specificity to virions, and bound well to soluble gp120: weak binding to 1/4 B clade viruses (CA5), and weak binding to viruses from subtype A and D [Nyambi (1998)] • 697-D: Called 697-30D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V2 MAbs G3.4, G3.136, or 687–30D [Stamatatos & Cheng-Mayer(1998)] • 697-D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V2 MAbs 697-D, 1357 and 1361 favored the monomer by approximately 2 fold[Gorny (2000)] 							

Table of HIV MAbs

- 697-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – V2 MAb 697-D did not effect proliferation [Hioe (2000)]
- 697-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 anti-V2 MAbs, which showed weak and sporadic binding, with the most frequent binding to C and D clades [Nyambi (2000)]

323	6C4/S	gp160(162–169)	gp120(BH10)	STSIRGKV		Vaccine	()
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>Donor: S. Ranjbar (NIBSC, UK)</p> <p>References: [Moore (1993a)]</p> <ul style="list-style-type: none"> • 6C4/S: UK Medical Research Council AIDS reagent: ARP3049 							
324	C108G	gp160(162–169)	gp120(162–169 HXB2)	STSIRGKV	L	HIV-1 infection	chimpanzee(IgG1κ)
<p>Donor: S. Tilley, Public Health Research Institute, NY, NY</p> <p>References: [Warrier (1994), Wu (1995), Warrier (1995), Warrier (1996), Ugolini (1997), Mondor (1998), Alsmadi & Tilley(1998)]</p> <ul style="list-style-type: none"> • C108G: Chimps were infected with HIV-1 IIIB, and this high affinity MAb gave potent neutralization of HIV-1 IIIB – binding not affected by reduction of disulfide bonds – binding disrupted by removal of N-linked glycans – peptide binding lower affinity than glycosylated Env [Warrier (1994)] • C108G: Strain specificity: LAI, Bal, HXB2 – conformational character – glycosylation site at 160 critical – mutation of conserved glycosylation site at 156 increased epitope exposure [Wu (1995)] • C108G: Characterization of MAb variable region [Warrier (1995)] • C108G: Synergistic neutralization of HIV-1 when combined with anti-V3 MAbs 0.5β and C311E, or anti-CD4BS MAbs, 1125H and 5145A – neutralization further enhanced by presence of both 1125H and 0.5β [Warrier (1996)] • C108G: Viral binding inhibition by C108G was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)] • C108G: Inhibits HX10 binding to both CD4 positive and negative HeLa cells[Mondor (1998)] • C108G: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against only IIIB – this is first demonstration of ADCC directed by a V2 specific MAb [Alsmadi & Tilley(1998)] 							
325	10/76b	gp160(162–170)	gp120(162–171 BH10)	STSIRGKVQ	L (HXB10)	Vaccine	rat(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>References: [McKeating (1993b), McKeating (1993a), Shotton (1995), Wu (1995), McKeating (1996)]</p> <ul style="list-style-type: none"> • 10/76b: R to L substitution abrogated binding – human sera recognize epitope [McKeating (1993b)] • 10/76b: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton (1995)] 							

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

326	11/41e	gp160(162–170)	gp120(162–171)	STSIRGKVQ	L (HXB10)	Vaccine	rat(IgG1)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120							
References: [McKeating (1993b), Shotton (1995), Wu (1995)]							
<ul style="list-style-type: none"> • 11/41e: R to L abrogated binding – human sera recognize the epitope [McKeating (1993b)] • 11/41e: Included in cross-competition and neutralization studies [Shotton (1995)] • 11/41e: HX10 strain specificity – binds native and deglycosylated gp120 [Wu (1995)] 							
327	11/4b	gp160(162–170)	gp120(162–171)	STSIRGKVQ	L (HXB10)	Vaccine	rat(IgG2a)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120							
References: [McKeating (1993b), Shotton (1995), Wu (1995), Moore & Sodroski(1996)]							
<ul style="list-style-type: none"> • 11/4b: A mutation R166L abrogated binding – human sera recognize epitope [McKeating (1993b)] • 11/4b: Cross-competes with MAbs 10/76b and 11/4c – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton (1995)] • 11/4b: HXB10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (1995)] • 11/4b: Linear V2 epitope – reciprocal binding enhancement of anti-V2 discontinuous epitope antibodies (in contrast to BAT085) and CD4 inducible antibody 48d. Reciprocal inhibits BAT085 binding – inhibits CRA-3 binding CRA-3 does not inhibit 11/4b [Moore & Sodroski(1996)] 							
328	RSD-33	gp160(162–170)	gp120(162–171)	STSIRGKVQ		Vaccine	()
Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120							
Donor: R. Daniels (NIMR, UK)							
References: [Moore (1993a)]							
329	11/4c (11/4c/1j/4j)	gp160(162–170)	gp120(152–181)	STSIRGKVQ	L (HXB2)	Vaccine	rat(IgG2a)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120							
Ab type: V2 References: [McKeating (1993b), Wu (1995), Shotton (1995), Peet (1998)]							
<ul style="list-style-type: none"> • 11/4c: R to L substitution abrogated binding – human sera recognize epitope [McKeating (1993b)] • 11/4c: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (1995)] • 11/4c: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton (1995)] 							

Table of HIV MAbs

- 11/4c: Called 11/4c/1j/4j – The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – 11/4c was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 11/4c: UK Medical Research Council AIDS reagent: ARP3035

330	12b	gp160(162–181)	gp120(162–181)	STSIRGKVQKEYAFFYKLDI	L (HXB10)	Vaccine	rat(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>Ab type: V2 References: [Shotton (1995), McKeating (1996)]</p> <ul style="list-style-type: none"> • 12b: V2 MAb neutralized HXB2 – position 179–180 LD to DL abrogates binding – competes with 60b, but not 74 [Shotton (1995)] • 12b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)] 							
331	G3-136 (G3.136)	gp160(dis 170–180)	gp120(dis 170–180 IIIB)	QKEYAFFYKLD	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: V2 Donor: Tanox Biosystems Inc and David Ho, ADARC, NY</p> <p>References: [Fung (1992), Pirofski (1993), Thali (1993), Moore & Ho(1993), Moore (1993a), Yoshiyama (1994), Sattentau & Moore(1995), Stamatatos & Cheng-Mayer(1995), Moore & Sodroski(1996), Pognard (1996a), Binley (1997a), Stamatatos (1997), Ditzel (1997), Wyatt (1997), Parren (1998a), Stamatatos & Cheng-Mayer(1998), Ly & Stamatatos(2000)]</p> <ul style="list-style-type: none"> • G3-136: V2 region – binds and neutralizes IIIB and RF in CEM-SS cells, but not MN – neutralization activity against a few primary isolates in PBMC – sCD4 binding inhibits binding (contrast with BAT085) – deglycosylation or reduction of gp120 by DTT diminishes reactivity [Fung (1992)] • G3-136: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)] • G3-136: Marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore (1993a)] • G3-136: Binding enhanced by selected antibodies to C1, C4, C5, V3 and anti-CD4 binding site MAbs – enhances binding of selected V3, C4 and anti-CD4 binding site MAbs [Moore (1993a)] • G3-136: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity [Yoshiyama (1994)] • G3-136: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V2 loop epitopes are somewhat occluded in the oligomeric gp120 epitopes on the virion surface relative to the gp120 monomer as indicated by an increase in the half-maximal binding values to macrophage-tropic isolates SF162 and SF128a – anti-V2 MAbs G3-4 and G3.136 don't bind to T-cell tropic SF2 [Stamatatos & Cheng-Mayer(1995)] • G3-136: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)] 							

- G3-136: Described epitope as STSIRGKVKEYAFFYKLDI – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT123 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50–69, in contrast to anti-V3 MAbs [Poignard (1996a)]
- G3-136: Called G3.136 – does not mediate gp120 virion dissociation in contrast to anti-V2 MAb G3-4 – not neutralizing for SF162 or SF128A in either primary macrophages or PBMC [Stamatatos (1997)]
- G3-136: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- G3-136: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- G3-136: Called G3.136 – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V2 MAbs G3.4, G3.136, or 687–30D [Stamatatos & Cheng-Mayer(1998)]
- G3-136: Called G3.136 – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391–95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]

332	G3-4 (G3.4)	gp160(dis 170–180)	gp120(dis 170–180 BH10)	QKEYAFFYKLD	L	Vaccine	murine(IgG2b κ)
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Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: V2 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Ho (1991a), Ho (1992), Fung (1992), McKeating (1992a), Moore & Ho(1993), Sullivan (1993), Sattentau (1993), Thali (1993), Moore (1993a), Moore (1994b), Gorny (1994), Thali (1994), Yoshiyama (1994), Stamatatos & Cheng-Mayer(1995), Wu (1995), Sattentau & Moore(1995), Jagodzinski (1996), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Stamatatos (1997), Ditzel (1997), Wyatt (1997), Parren (1998a), Stamatatos & Cheng-Mayer(1998), Ly & Stamatatos(2000), Srivastava (2002)]

- G3-4: Binding is sensitive to removal of glycans by endo H – 50% neutralization of 4/9 primary isolates – has conformational features [Ho (1991a)]
- G3-4: Neutralizes IIIB and RF, not MN – blocks sCD4-gp120, not as potent as MAb 15e – V2 binding MAbs BAT085 and G3-136 block G3-4 gp120 binding – sensitive to reduction of gp120 by DTT [Ho (1992)]
- G3-4: Substitutions in residues 176 to 184 affect MAb recognition – substitutions in V2 can result in gp120-gp41 dissociation [Sullivan (1993)]
- G3-4: Increased binding in the presence of sCD4 [Sattentau (1993)]
- G3-4: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- G3-4: V2 region, marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore (1993a)]

Table of HIV MAbs

- G3-4: Conformationally sensitive – sporadic cross-reactivity among, and outside, B clade gp120s [Moore (1994b)]
- G3-4: Weakly neutralizing, IC 50 = 53 $\mu\text{g/ml}$ [Gorny (1994)]
- G3-4: gp41 mutation (582 A/T) that reduces neutralization of anti-CD4 binding site MAbs does not alter G3-4s ability to neutralize [Thali (1994)]
- G3-4: Neutralizes RF – substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity and result in neutralization escape [Yoshiyama (1994)]
- G3-4: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V2 loop epitopes are somewhat occluded in the oligomeric gp120 epitopes on the virion surface relative to the gp120 monomer as indicated by an increase in the half-maximal binding values to macrophage-tropic isolates SF162 and SF128a – anti-V2 MAbs G3-4 and G3.136 don't bind to T-cell tropic SF2 [Stamatatos & Cheng-Mayer(1995)]
- G3-4: Reactive with BH10, RF, and MN – binds native, but not denatured or deglycosylated gp120, binds to deglycosylated V1V2 fusion protein, suggesting importance of glycans outside the V1V2 region [Wu (1995)]
- G3-4: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes Hx10 cell-free virus [Sattentau & Moore(1995)]
- G3-4: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop from gp120 results in more potent G3-4 binding inhibition by CRDS – G3-4 epitope described as 176–184 FYKLDIPI and 191–193 YSL [Jagodzinski (1996)]
- G3-4: Binding enhanced by selected antibodies to C1, C4, C5, V3 and anti-CD4 binding site MAbs – enhances binding of selected V3, C4 and anti-CD4 binding site MAbs [Moore & Sodroski(1996)]
- G3-4: Described epitope as STSIRGKVKEYAFFYKLDI – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT085 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50–69, in contrast to anti-V3 MAbs [Poignard (1996a)]
- G3-4: Called G3.4 – mediates gp120 virion dissociation in contrast to anti-V2 MAb G3-136 – not neutralizing for SF162 or SF128A in either primary macrophages or PBMC [Stamatatos (1997)]
- G3-4: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- G3-4: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- G3-4: Called G3.4 – Deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V2 MAbs G3.4, G3.136, or 687–30D [Stamatatos & Cheng-Mayer(1998)]
- G3-4: Called G3.4 – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391–95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]

- G3-4: Called G3.4 – Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – G3.4 recognized o-gp140 [Srivastava (2002)]

333	BAT085 (BAT-085)	gp160(171–180)	gp120(170–180 IIIB)	KEYAFFYKLD	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* inactivated virus *Strain:* IIIB *HIV component:* virus

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

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

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

334	60b	gp160(172–181)	gp120(172–181 HXB2)	EYAFFYKLDI	no	Vaccine	rat(IgG2b)
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Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

References: [Shotton (1995)]

Table of HIV MAbs

- 60b: V2 MAb did not neutralize HXB2 – bound to rgp120 in ELISA – substitutions 179–180 LD/DL and 191–193 YSL/GSS abrogate binding, as do changes outside the minimum epitope – competes with 12b, but not 74 [Shotton (1995)]

335	74	gp160(172–181)	gp120(172–181)	EYAFFYKLDI	no	Vaccine	rat(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>References: [Shotton (1995)]</p> <ul style="list-style-type: none"> • 74: V2 MAb did not neutralize HXB2 – did not bind rgp120 ELISA – position 179–180 LD to DL abrogates binding, as do changes outside the minimum epitope – does not compete with 60b or 12b, and is enhanced by two conformation dependent MAbs [Shotton (1995)] 							
336	38/12b	gp160(172–191)	gp120(172–191 HXB2)	EYAFFYKLDIIPIDNDTTSY		Vaccine	rat()
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>References: [Wu (1995)]</p> <ul style="list-style-type: none"> • 38/12b: Broad specificity: HXB2, MN, SF162 – binds native and deglycosylated gp120 [Wu (1995)] 							
337	38/60b	gp160(172–191)	gp120(172–191 HXB2)	EYAFFYKLDIIPIDNDTTSY		Vaccine	rat()
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>References: [Wu (1995)]</p> <ul style="list-style-type: none"> • 38/60b: Strain specificity: HXB2 – binds native and deglycosylated gp120 [Wu (1995)] 							
338	polyclonal (VEI2)	gp160(176–196)	Env()	FYKLDIVPIDNTTTSYRLISC		HIV-1 infection	human()
<p>References: [Carlos (1999)]</p> <ul style="list-style-type: none"> • Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)] 							
339	322–151	gp160(211–221)	gp120(201–220 LAI)	EPIPIHYCAPA		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Env</p> <p>Donor: G. Robey, Abbot Labs</p> <p>References: [Moore (1994c), Moore (1994d)]</p> <ul style="list-style-type: none"> • 322–151: The relative affinity denatured/native gp120 is 30 [Moore (1994c)] 							
340	3D3.B8	gp160(211–221)	gp120(211–220 LAI)	EPIPIHYCAPA		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Env</p>							

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346	J3	gp160(222–231)	gp120(222–231 LAI)	GFAILKCNNK	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> LAI Donor: J. Hoxie, U. Penn. References: [Moore (1994c), Cook (1994)]</p> <ul style="list-style-type: none"> • J3: The relative affinity denatured/native gp120 is 30 [Moore (1994c)] • J3: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook (1994)] 						
347	1006–30-D	gp160(236–245)	gp120(241–251)	KGSCKNVSTV		human(IgG1λ)
<p>Ab type: C2 References: [Hioe (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 1006–30-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – C2 MAbs 1006–30-D and 847-D did not effect proliferation [Hioe (2000)] • 847-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including two C2 MAbs – the binding of anti-C2 MAbs was weak to isolates from clades B, C, D, E, F, G, and they did not bind to isolates from subtypes A and H – epitope is suggested to be in a 20 amino acid peptide KGSCKNVSTVQCTHGIRPVV [Nyambi (2000)] 						
348	847-D	gp160(236–245)	gp120(241–251)	KGSCKNVSTV		human(IgG1λ)
<p>Ab type: C2 References: [Hioe (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 847-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – C2 MAbs 1006–30-D and 847-D did not effect proliferation [Hioe (2000)] • 847-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including two C2 MAbs – the binding of anti-C2 MAbs was weak to isolates from clades B, C, D, E, F, G, and they did not bind to isolates from subtypes A and H – epitope is suggested to be in a 20 amino acid peptide KGSCKNVSTVQCTHGIRPVV [Nyambi (2000)] 						
349	MF169.1	gp160(252–261)	gp120(242–261 LAI)	RPVVSTQLLL	Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env References: [Thiriart (1989), Moore (1994c), Moore (1994d)]</p> <ul style="list-style-type: none"> • MF169.1: The relative affinity denatured/native gp120 is 11 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore (1994c)] 						
350	MF170.1	gp160(252–261)	gp120(242–261 LAI)	RPVVSTQLLL	Vaccine	murine(IgG)
<p>Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env References: [Thiriart (1989), Moore (1994c), Moore (1994d)]</p> <ul style="list-style-type: none"> • MF170.1: The relative affinity denatured/native gp120 is 15 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding to denatured and native gp120, and 262N/T, 269 E/L and 281 A/V to only native gp120 [Moore (1994c)] 						

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351	MF87.1	gp160(252–261)	gp120(242–261 LAI)	RPVVSTQLLL	Vaccine	murine(IgG)
<p>Vaccine: Strain: LAI HIV component: Env</p> <p>References: [Thiriart (1989), Moore (1994c)]</p> <ul style="list-style-type: none"> • MF87.1: The relative affinity denatured/native gp120 is 10 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore (1994c)] 						
352	213.1	gp160(252–261)	gp120(242–261 LAI)	RPVVSTQLLL	Vaccine	murine(IgG1)
<p>Vaccine: Vector/type: recombinant protein HIV component: Env</p> <p>Ab type: C2 Donor: Claudine Bruck</p> <p>References: [Thiriart (1989), Moore & Ho(1993), Moore (1994c)]</p> <ul style="list-style-type: none"> • 213.1: Bound preferentially to denatured IIIB and SF2 gp120 [Moore & Ho(1993)] • 213.1: The relative affinity denatured/native gp120 is 100 – mutations 252 R/W, 257 T/G or T/R impair binding [Moore (1994c)] • 213.1: UK Medical Research Council AIDS reagent: ARP334 						
353	B12	gp160(252–271)	gp120(252–271 LAI)	RPVVSTQLLLNGSLAEEEEVV	Vaccine	murine(IgG)
<p>Vaccine: Vector/type: recombinant protein Strain: LAI HIV component: gp160</p> <p>Ab type: C2 References: [Moore (1994c)]</p> <ul style="list-style-type: none"> • B12: C2 region – the relative affinity for denatured/native gp120 is 27 – mutations 257 T/R and 262 N/T impair binding [Moore (1994c)] 						
354	B13 (Bh13)	gp160(252–271)	gp120(252–271 LAI)	RPVVSTQLLLNGSLAEEEEVV	Vaccine	murine(IgG2a)
<p>Vaccine: Vector/type: recombinant protein Strain: LAI HIV component: gp160</p> <p>Ab type: C2 Donor: George Lewis, Institute of Human Virology, Baltimore MD, USA</p> <p>References: [Pincus & McClure(1993), Moore & Ho(1993), Moore (1994c), Abacioglu (1994), Moore (1994d), Pincus (1996), Connor (1998)]</p> <ul style="list-style-type: none"> • B13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • B13: The relative affinity for denatured/native gp120 is 30 – mutations 257 T/R and 269 E/L impair binding [Moore (1994c)] • B13: C2 region – epitope boundaries mapped by peptide scanning, core epitope: TQLLLN [Abacioglu (1994)] • B13: Called Bh13 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus (1996)] 						
355	C13	gp160(252–271)	gp120(252–271 LAI)	RPVVSTQLLLNGSLAEEEEVV	Vaccine	murine(IgG1)
<p>Vaccine: Vector/type: recombinant protein Strain: LAI HIV component: gp160</p> <p>Ab type: C2 Donor: George Lewis</p> <p>References: [Moore & Ho(1993), Moore (1994c), Abacioglu (1994)]</p> <ul style="list-style-type: none"> • C13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • C13: The relative affinity for denatured/native gp120 is 36 – mutations 257 T/R, 267 E/L, and 269 E/L impair binding [Moore (1994c)] 						

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- C13: Epitope boundary extended to RPVVSTQLLLNGSLAEEEEVVIR, to take into account the effect of a point mutation [Abacioglu (1994)]
- C13: NIH AIDS Research and Reference Reagent Program: 1209

356	M89	gp160(252–271)	gp120(252–271 LAI)	RPVVSTQLLLNGSLAEEEEVV	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> Env</p> <p>Ab type: C2 Donor: Fulvia di Marzo Veronese</p> <p>References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d)]</p> <ul style="list-style-type: none"> • M89: Immunoblot reactive, RIP negative, for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)] • M89: C2 region – the relative affinity for denatured/native gp120 is >30 – mutations 257 T/R and 269 E/L impair binding [Moore (1994c)] 							
357	B21	gp160(257–262)	gp120(257–262 BH10)	TQLLLN		Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p> <p>Ab type: C2 References: [Abacioglu (1994)]</p> <ul style="list-style-type: none"> • B21: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)] 							
358	B23	gp160(257–262)	gp120(257–262 BH10)	TQLLLN		Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p> <p>Ab type: C2 References: [Abacioglu (1994)]</p> <ul style="list-style-type: none"> • B23: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)] 							
359	B24	gp160(257–262)	gp120(257–262 BH10)	TQLLLN		Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p> <p>Ab type: C2 References: [Abacioglu (1994)]</p> <ul style="list-style-type: none"> • B24: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)] 							
360	B25	gp160(257–262)	gp120(257–262 BH10)	TQLLLN		Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p> <p>Ab type: C2 References: [Abacioglu (1994)]</p> <ul style="list-style-type: none"> • B25: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)] 							

Table of HIV MAbs

361	B3	gp160(257–262)	gp120(257–262 BH10)	TQLLLN	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160 Ab type: C2 References: [Abacioglu (1994)] <ul style="list-style-type: none"> • B3: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)] </p>						
362	B26	gp160(257–263)	gp120(257–263 BH10)	TQLLNG	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160 Ab type: C2 References: [Abacioglu (1994)] <ul style="list-style-type: none"> • B26: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)] </p>						
363	B29	gp160(257–263)	gp120(257–263 BH10)	TQLLNG	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160 Ab type: C2 References: [Abacioglu (1994)] <ul style="list-style-type: none"> • B29: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)] </p>						
364	B36	gp160(257–263)	gp120(257–263 BH10)	TQLLNG	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160 Ab type: C2 References: [Abacioglu (1994)] <ul style="list-style-type: none"> • B36: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)] </p>						
365	110.E	gp160(262–281)	gp120(262–281 LAI)	NGSLAEEEEVVIRSVNFTDNA	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> Env Ab type: C2 Donor: F. Traincard References: [Moore (1994c), Moore (1994d)] <ul style="list-style-type: none"> • 110.E: The relative affinity for denatured/native gp120 is 7.3 [Moore (1994c)] </p>						
366	110.C	gp160(271–280)	gp120(271–280 LAI)	VIRSVNFTDN	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> Env Ab type: C2 Donor: F. Traincard, Hybridolabs, Institut Pasteur References: [Moore (1994c), Moore (1994d), Valenzuela (1998)] <ul style="list-style-type: none"> • 110.C: The relative affinity for denatured/native gp120 is 1 [Moore (1994c)] • 110.C: Only slightly reduces LAI viral binding or entry into CEM cells [Valenzuela (1998)] </p>						

Table of HIV MAbs

367	IIIB-V3-26	gp160(291-307)	gp120(299-304 IIIB)	SVEINCTRPNNNTRKSI	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> IIIB Ab type: V3 References: [Laman (1992)] <ul style="list-style-type: none"> • IIIB-V3-26: Binds to the base of the V3 loop on denatured gp120 [Laman (1992)] </p>							
368	IIIB-V3-21 (V3-21)	gp160(294-299)	gp120(299-304 IIIB)	INCTRP	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> IIIB Ab type: V3 Donor: J. Laman References: [Laman (1992), Laman (1993), Valenzuela (1998)] <ul style="list-style-type: none"> • IIIB-V3-21: Binds to the base of the V3 loop on denatured gp120 [Laman (1992)] • IIIB-V3-21: Binds to NP40 treated gp120, and epitope is probably obscured by local glycosylation [Laman (1993)] • IIIB-V3-21: Does not block HIV-1 LAI binding or entry into CEM cells [Valenzuela (1998)] • IIIB-V3-21: UK Medical Research Council AIDS reagent: ARP3048 • IIIB-V3-21: NIH AIDS Research and Reference Reagent Program: 1725 </p>							
369	polyclonal	gp160(296-327)	gp120(MN)	CNYNKRKRRIHIGPGRAFYTTL NIIGTIC	L		rabbit(IgA,IgG)
<p>Ab type: V3 References: [FitzGerald (1998)] <ul style="list-style-type: none"> • Polyclonal response to MN, or Thai E V3 loop inserted into Pseudomonas Exotoxin for vaccination – inserts of 14 or 26 amino acids were used from MN or a Thai E strain, constrained by disulfide bond – sera from vaccinated rabbit were reactive with strain-specific gp120 – administration to mucosal surfaces elicits IgA [FitzGerald (1998)] </p>							
370	polyclonal	gp160(297-320)	gp120()	NYNKRKRRIHIGPGRAFYTTL	L	HIV-1 infection, Vaccine	human()
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> cocktail <i>HIV component:</i> V3 Ab type: V3 References: [Bartlett (1998)] <ul style="list-style-type: none"> • V3 peptide vaccine (MN, RF, EV91, and Can0A) with a C4 helper T cell epitope were used to vaccinate HLA-B7 HIV-infected patients – V3 Ab levels and the anti-HIV proliferative response, but no decrease in HIV-1 RNA levels or increase in CD4 levels was observed [Bartlett (1998)] </p>							
371	polyclonal	gp160(297-320)	gp120()	NYNKRKRRIHIGPGRAFYTTL		HIV-1 exposed seronegative	human(IgA)
<p>Ab type: V3 References: [Kaul (1999)] <ul style="list-style-type: none"> • HIV-1 Env-specific mucosal IgA found in genital track of 16/21 HIV-1 resistant chronically exposed Kenyan sex workers – 11/21 had detectable Th responses [Kaul (1999)] </p>							

372	polyclonal	gp160(297–331)	Env(303–335 LAI)	TRPNNNTRKSIHIGPGRAFYA- TGEIIGDIRQAH	no	Vaccine	human(IgG)
<p>Vaccine: <i>Vector/type:</i> lipopeptide <i>Strain:</i> LAI <i>HIV component:</i> V3 <i>Stimulatory Agents:</i> QS21</p> <p>Ab type: V3 References: [Pialoux (2001)]</p> <ul style="list-style-type: none"> • 28 subjects were vaccinated with six HIV-1 peptides that were selected to be particularly rich in CTL epitopes, presented in lipopeptides with or without adjuvant QS21 – HIV-specific Ab responses were detected in 25/28 (89%), proliferative in 19/28 (79%), and CTL in 13/24 (54%) of testable volunteers – 14/28 had non-neutralizing Ab responses to this peptide (E), 7/24 had proliferative responses, and multiple CTL responses were detected [Pialoux (2001)] 							
373	MO97/V3	gp160(299–308)	gp120(299–308 IIIB)	PNNNTRKSIR	no	<i>in vitro</i> stimulation	human(IgM)
<p>Ab type: V3 References: [Ohlin (1992)]</p> <ul style="list-style-type: none"> • MO97: Generated through <i>in vitro</i> stimulation of uninfected-donor lymphocytes with rpB1 (IIIB Env 286–467) [Ohlin (1992)] 							
374	polyclonal	gp160(299–331)	gp120(306–338 BH10)	PNNNTRKSIRIQRGPGRAFVT- IGKIGNMRQAHC	L	Vaccine	rabbit(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> BH10</p> <p>Ab type: V3 References: [Neurath & Strick(1990)]</p> <ul style="list-style-type: none"> • 21 V3 loop variant peptides spanning this region were tested and serological cross-reactivity correlated with divergence [Neurath & Strick(1990)] 							
375	55/11	gp160(300–315)	gp120(300–315)	NNNTRKRIRIQRGPGR?			()
<p>Ab type: V3 References: [Peet (1998)]</p> <ul style="list-style-type: none"> • 55/11: 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 55/11 binding was abrogated by V3 serine substitutions in the V3 loop – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)] 							
376	8/38c (8/38/1c)	gp160(300–315)	gp120(300–315 HXB10)	NNNTRKRIRIQRGPGR	L	Vaccine	rat(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>Ab type: V3 Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK</p> <p>References: [McKeating (1992a), Sattentau & Moore(1995), Jeffs (1996), Parren (1998a), Peet (1998)]</p> <ul style="list-style-type: none"> • 8/38c: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating (1992a)] • 8/38c: Binds equally well to monomer and oligomer, less rapid association rate than other anti-V3 antibodies, and an associated less potent neutralization of lab strains [Sattentau & Moore(1995)] • 8/38c: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs (1996)] • 8/38c: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)] 							

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- 8/38c: Called 8/38/1c: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 8/38c binding was diminished by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 8/38c: UK Medical Research Council AIDS reagent: ARP3039

377	8/64b	gp160(300–315)	gp120(300–315 HXB10)	NNNTRKRIRIQRGPGGR	L	Vaccine	rat(IgM)
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Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V3 **References:** [McKeating (1992a), Peet (1998)]

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

378	polyclonal	gp160(300–322)	gp120(IIIB)	CNNTRKSIRIQRGPGRAFVTI- GK	L		guinea pig(IgG)
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Ab type: V3 **Donor:** D. Bolognesi and T. Matthews, Duke University

References: [Allaway (1993)]

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

379	polyclonal	gp160(300–328)	Env()	NNNTRKSIRIGPGRAFYTGD- IGNIRQ		HIV-1 infection	human()
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Ab type: V3 **References:** [Carlos (1999)]

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

380	9284 (NEA 9284)	gp160(301–312)	gp120(307–318 IIIB)	NNTRKSIRIQRG	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* inactivated virus *Strain:* IIIB *HIV component:* virus

Ab type: V3 **Donor:** Dupont de Nemours, Les Ulis, France or Wilmington, Delaware

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

- 9284: IIIB type-specific binding and neutralization [Skinner (1988b)]
- 9284: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 9284: Single amino acid substitutions in the C4 region (427 W/V or W/S) or at the base of the V3 loop (298 R/G) can significantly increase binding and neutralization– position 427 is also important for CD4 binding and anti-CD4 binding site MAbs [Wyatt (1992)]
- 9284: Increased binding in the presence of sCD4 [Sattentau (1993)]
- 9284: Inhibits C4 region antibodies (G3-299, G3-519) which have conformational requirements [Moore (1993b)]
- 9284: Peptide RIQRGPGRAFVTIGKIGNMRQA – Reacts with three human brain proteins of 35, 55, 110 kd – called NEA-9284 [Trujillo (1993)]
- 9284: Does not bind MN gp120, just IIIB [VanCott (1994)]
- 9284: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali (1994)]
- 9284: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook (1994)]
- 9284: Binding domain aa 301–310: TRKSIRIQRG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: R306T, R309T and R313G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5β – called NEA9284 [Okada (1994)]
- 9284: Did not neutralize infection of HIV/HTLV-I pseudotype [Sorensen (1994)]
- 9284: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains – neutralizes cell-free virus Hx10 [Sattentau & Moore(1995)]
- 9284: Used to monitor HIV-1 Env expression in infected H9 cells, binds native and reduced gp120s similarly [VanCott (1995)]
- 9284: Binds V3 loop – anti-C1 MAbs 133/290 and 135/9 enhance binding – reciprocal binding inhibition of other anti-V3 MAbs [Moore & Sodroski(1996)]
- 9284: V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50–69, in contrast to anti-V2 MAbs [Poignard (1996a)]
- 9284: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao (1997)]
- 9284: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- 9284: In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, 9284 was found to have an inaccessible epitope on the oligomeric form of Env and anti-V3 MAbs were found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan more efficiently than HIV-BRU [Schonning (1998)]

381	polyclonal	gp160(301–325)	gp120(IIIB)	NNTRKSIRIQRGPGRAFVTIG- KIGN	L	Vaccine	murine(IgA)
<i>Vaccine: Vector/type:</i> peptide <i>Strain:</i> IIIB <i>Stimulatory Agents:</i> cholera toxin adjuvant							
Ab type: V3 References: [Bukawa (1995)]							

Table of HIV MAbs

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

382	polyclonal	gp160(301–325)	gp120(IIIB)	NNTRKSIRIQRGPGRAFVTIG- KIGN	L	Vaccine	murine(IgA22a)
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Vaccine: *Vector/type:* DNA *Strain:* IIIB *HIV component:* Env, Rev *Stimulatory Agents:* QS-21

Ab type: V3 **References:** [Sasaki (1998)]

- An anti-Env response was sought, and co-expression of Rev was required – intramuscular versus nasal vaccination with DNA vaccine with a QS-21 adjuvant was studied – QS-21 enhanced the IgG2a response mediated via Th1 cytokines IFN γ and IL-2 [Sasaki (1998)]

383	polyclonal	gp160(302–318)	Env()	NTRKSIHIGPGRIFY	L P	HIV-1 infection	human()
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Ab type: V3 **References:** [Bongertz (2001)]

- Non-transmitting mothers had an increased frequency of high neutralizing plasma Ab titers against HIV-1 MN (1:50 dilution, >90% neutralization, 33/88 pregnant women), compared to plasma from transmitting mothers (0/8 pregnant women) – non-transmitting mothers also had more potent neutralization against primary isolates from transmitting mothers, but neutralization of autologous virus was comparable for non-transmitting (7/13) and transmitting mothers (2/4) [Bongertz (2001)]

384	MAG 109	gp160(302–321)	gp120(302–321 BH10)	NTRKSIRIQRGPGRAFVTIG	L	Vaccine	murine()
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Vaccine: *Vector/type:* sCD4-gp120 complex *Strain:* HXB2 *HIV component:* gp120

Ab type: V3 **References:** [Kang (1994)]

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

385	MAG 49 (#49)	gp160(302–321)	gp120(302–321 BH10)	NTRKSIRIQRGPGRAFVTIG	L	Vaccine	murine()
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Vaccine: *Vector/type:* sCD4-gp120 complex *Strain:* HXB2 *HIV component:* gp120

Ab type: V3 **References:** [Kang (1994), Moore & Sodroski(1996)]

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

386	MAG 53	gp160(302–321)	gp120(302–321 BH10)	NTRKSIRIQRGPGRAFVTIG	L	Vaccine	murine()
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Vaccine: *Vector/type:* sCD4-gp120 complex *Strain:* HXB2 *HIV component:* gp120

Ab type: V3 **References:** [Kang (1994)]

Table of HIV MAbs

391	C311E	gp160(304–313)	gp120(309–316 MN)	RKRIHIGP	L	HIV-1 infection	chimpanzee(IgG1)
<p>Ab type: V3 References: [Warrier (1996), Alsmadi & Tilley(1998)]</p> <ul style="list-style-type: none"> • C311E: Chimps were infected with HIV-1 IIIIB, and this resulting MAb gave synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier (1996)] • C311E: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIIB, MN, SF-2, and RF – C311E bound and directed lysis against all four strains [Alsmadi & Tilley(1998)] 							
392	907	gp160(304–314)	gp120(309–318)	RKSIRIQRGPG	L	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIIB <i>HIV component:</i> gp160</p> <p>References: [Chesebro & Wehrly(1988), Pincus (1989), Pincus (1991), Pincus (1996)]</p> <ul style="list-style-type: none"> • 907: Strain specific binding, and neutralization of only the LAV strain [Chesebro & Wehrly(1988)] • 907: Coupled to ricin A chain (RAC), MAb 907 inhibited protein synthesis and cell growth in HIV-infected cells [Pincus (1989)] • 907: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIIB strain-specific [Pincus (1991)] • 907: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)] 							
393	924	gp160(304–314)	gp120(309–318 IIIIB)	RKSIRIQRGPG		Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIIB <i>HIV component:</i> gp160</p> <p>Ab type: V3 References: [Chesebro & Wehrly(1988), Pincus (1991), Pincus & McClure(1993), Pincus (1993), Cook (1994), Pincus (1996), Pincus (1998)]</p> <ul style="list-style-type: none"> • 924: HIV IIIIB strain specific [Chesebro & Wehrly(1988)] • 924: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIIB strain-specific [Pincus (1991)] • 924: MAb was coupled to ricin A chain (RAC) – immunotoxin efficacy was not significantly decreased by sCD4, although the efficacy of gp41 MAb immunotoxins <i>in vitro</i> increased 30-fold by sCD4 [Pincus & McClure(1993)] • 924: Ab response in IIIIB lab workers was compared to gp160 LAI vaccine recipients – MAb 924 was used as a control – infected lab workers and a vaccinia gp160 vaccine had strong V3 MAb response, but alum absorbed rec gp160 did not generate anti-V3 response [Pincus (1993)] • 924: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer <i>in vitro</i> [Cook (1994)] • 924: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)] 							
394	polyclonal	gp160(304–318)	gp120(304–318 LAI)	RKSIRIQRGPGRAFV		<i>in vitro</i> stimulation	human(IgG,IgM)
<p>Ab type: V3 References: [Chin (1995)]</p> <ul style="list-style-type: none"> • Mimicking the humoral immune response <i>in vitro</i> supports isotype switching – human IgG MAbs were generated from naive donors [Chin (1995)] 							

395	polyclonal	gp160(304–318)	gp120(304–318 LAI)	RKSIRIQRGPGRAFV		Vaccine	human(IgG,IgM)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> LAI</p> <p>Ab type: V3 References: [Zafiroopoulos (1997)]</p> <ul style="list-style-type: none"> • IgG to IgM isotype switching in response to primary and secondary peptide vaccinations was studied – the immunogen contained a V3 loop fragment and a tetanus toxin helper epitope [Zafiroopoulos (1997)] 							
396	10F10	gp160(304–320)	gp120(MN)	RKRIHIGPGRAFYTT	L	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> MN <i>HIV component:</i> gp120</p> <p>Ab type: V3 References: [Duarte (1994)]</p> <ul style="list-style-type: none"> • 2C4: Putative epitope lies within IHIGPGRAFYT – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAFYTT) peptides, lower affinity for SF2 [Duarte (1994)] 							
397	2C4	gp160(304–320)	gp120(MN)	RKRIHIGPGRAFYTT	L (MN)	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> MN</p> <p>Ab type: V3 References: [Duarte (1994)]</p> <ul style="list-style-type: none"> • 2C4: Putative epitope lies within IHIGPGRAFYT – neutralizes MN, not IIIB and SF2 – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAFYTT) peptides, lower affinity for SF2 [Duarte (1994)] 							
398	412-D (412–10D, 412, 412D)	gp160(304–320)	gp120(MN)	RKRIHIGPGRAFYTT	L	HIV-1 infection	human(IgG1 κ)
<p>Ab type: V3 Donor: Susan Zolla-Pazner (Zollas01@mrcr6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (1993), Spear (1993), VanCott (1994), Fontenot (1995), Gorny (1998), Nyambi (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 412-D: Neutralizes MN, does not bind SF2 or HXB2 – not reactive with hexa or heptapeptides by Pepscan [Gorny (1993)] • 412-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear (1993)] • 412-D: Called 412–10D – relatively rapid dissociation and weak homologous neutralization [VanCott (1994)] • 412-D: Called 412 – The tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot (1995)] • 412-D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 412-D has a relatively fast dissociation, thus low affinity among V3 MAbs [Gorny (1998)] • 412-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 412-D was bound only to B clade virions and to D clade MAL [Nyambi (1998)] • 412-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)] • 412-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 391.5, 412 and 418, all selected with MN V3 peptide – the core amino acids HIGPGR tended to be critical for reactivity in this group [Zolla-Pazner (1999b)] 							

Table of HIV MAbs

- 412-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 412-D showed limited reactivity [Nyambi (2000)]

399	polyclonal	gp160(304–320)	gp120(MN)	RKRIHIGPGRAFYT	L (MN ALA-1)	HIV-1 infection	human()
<p>Ab type: V3 References: [Spear (1994)]</p> <ul style="list-style-type: none"> • 40% of antibody in serum that can bind to native viral proteins on MN-infected cells can be blocked by the peptide RKRIHIGP-GRAFYT, which can also block 75–95% of the complement activation on HIV infected cells [Spear (1994)] 							
400	CGP 47 439	gp160(304–322)	gp120()		L	Vaccine	human(Ig)
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: V3 References: [Liou (1989), Safrit (1993), Gunthard (1994), Gauduin (1998), Jacobson(1998)]</p> <ul style="list-style-type: none"> • CGP 47 439: passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus – CGP 47 439 is a BAT123-human Ig chimera [Safrit (1993)] • CGP 47 439: Phase I/IIA clinical trial studying multidose tolerability, immunogenicity and pharmacokinetic responses – GP 47 439 was well tolerated, serum t_{1/2} was 8–16 days, and a virus burden reduction was noted in some patients [Gunthard (1994)] • CGP 47 439: Post-exposure passive transfer of murine BAT123 can confer protection to hu-PBL-SCID mice challenged with HIV-1 LAI – this protection is not elicited by CGP 47 439, suggesting that the protection is mediated by complement – the protective ability of BAT123 is lost when mice were treated with cobra venom factor, which inactivates serum complement – in this circumstance complement activation provided a protective advantage [Gauduin (1998)] • CGP 47 439: Review of passive immunotherapy, summarizing [Gunthard (1994)] in relation to other studies [Jacobson(1998)] 							
401	178.1 (178.1.1)	gp160(305–309)	gp120(305–309 BH10)	KSIRI	L	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> yeast derived gp160</p> <p>Ab type: V3 Donor: C. Thiriart, Smith Kline and MRC AIDS reagent project</p> <p>References: [Thiriart (1989), Back (1993), Moore & Ho(1993), Cook (1994)]</p> <ul style="list-style-type: none"> • 178.1: Reacts to gp120 and gp160 in RIPA EIA and immunoblot [Thiriart (1989)] • 178.1: Called 178.1.1 – conformational, does not bind well to denatured gp120 [Moore & Ho(1993)] • 178.1: gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662–675 is ELDKWANLWNWFNI [Back (1993)] • 178.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer <i>in vitro</i> – binding of GalCer to gp120 inhibited but did not completely block MAb binding [Cook (1994)] • 178.1: UK Medical Research Council AIDS reagent: ARP331 							

402 257-D (257, gp160(dis 305– gp120(dis MN) KRIHI L HIV-1 infection human(IgG1λ)
 257–2-D-IV, 309)
 257-D-IV,
 257, 257–
 2D, 257D)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)

References: [Gorny (1991), D'Souza (1991), Karwowska (1992b), Gorny (1993), Cavacini (1993a), Spear (1993), D'Souza (1994), VanCott (1994), Stamatatos & Cheng-Mayer(1995), D'Souza (1995), Zolla-Pazner (1995), Schutten (1995a), Schutten (1995b), Fontenot (1995), Wisniewski (1996), Schutten (1996), Schutten (1997), Stamatatos (1997), Hill (1997), LaCasse (1998), Yang (1998), Gorny (1998), Stamatatos & Cheng-Mayer(1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Beddows (1999), Oggioni (1999), Nyambi (2000), Park (2000), York (2001)]

- 257-D: Called 257–2-D-IV – potent neutralizing MAb [D'Souza (1991)]
- 257-D: Reacts with MN, NY5, CDC4 and SF2, does not cross-react with RF, WM52, or HXB2 [Karwowska (1992b)]
- 257-D: Neutralizes MN – binds SF2: KSIYI – specificity: MN, SF2, NY5, RF. [Gorny (1993)]
- 257-D: Additive MN or SF2 neutralization when combined with CD4 binding site MAb F105 – does not neutralize RF [Cavacini (1993a)]
- 257-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG – complement mediated virolysis of MN, but not in the presence of sCD4 [Spear (1993)]
- 257-D: Included a multi-lab study for antibody characterization and assay comparison – best NAb against MN, but not IIIB [D'Souza (1994)]
- 257-D: Potent MN neutralization, slow dissociation constant [VanCott (1994)]
- 257-D: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V3 loop epitopes were less accessible to Ab binding on the virion surface than in the gp120 monomer, particularly for macrophage-tropic isolates SF162 and SF128a, relative to T-cell tropic SF2 – sCD4 association with gp120 better revealed this V3 epitope on TCLA SF2 and dual tropic (MU3) viruses than on macrophage tropic isolates [Stamatatos & Cheng-Mayer(1995)]
- 257-D: Called 257-D-IV – could neutralize MN and closely related JRCSF, but not 2 B subtype and 1 D subtype primary isolates in a multi-laboratory study involving 11 labs [D'Souza (1995)]
- 257-D: In serotyping study using flow-cytometry, bound only to virus with KRIHI [Zolla-Pazner (1995)]
- 257-D: Only inhibition of SI phenotype virus, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten (1995a)]
- 257-D: Comparable affinity for SI and NSI viruses, in contrast to MAb MN215 [Schutten (1995b)]
- 257-D: 257-D is V H5 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisniewski (1996)]
- 257-D: IIIB neutralizing MAbs *in vitro* fail to neutralize in a mouse model *in vivo* [Schutten (1996)]
- 257-D: Neutralized (>90%) an SI-env chimeric virus and enhanced (>200%) an NSI-env chimeric virus [Schutten (1997)]
- 257-D: Binds less extensively than MAb 391–95D on the surface of HIV-1 isolates SF162 and SF128A – neutralizes less potently than 391–95D – stronger neutralization of primary macrophage targets than PBMC [Stamatatos (1997)]

Table of HIV MAbs

- 257-D: Called 257 – gp120 can inhibit MIP-1 α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill (1997)]
- 257-D: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by anti-V3 MAbs – the primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse (1998)]
- 257-D: A neutralization assay was developed based on hemi-nested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang (1998)]
- 257-D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 257-D has a slow dissociation, thus the highest affinity among V3 MAbs [Gorny (1998)]
- 257-D: Called 257D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V3 MAbs 391–95D or 257D [Stamatatos & Cheng-Mayer(1998)]
- 257-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 257-D: MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 257-D: rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – 257-D bound rgp120 W61D but could only neutralize the W61D isolate following T-cell line adaptation [Beddows (1999)]
- 257-D: Study of a live-vector mucosal vaccine that expresses HIV-1 V3 domains using the bacterium *Streptococcus gordonii* which can express heterologous Ag and can colonize the oral cavity and vagina of mice – 268-D and 257-D recognized *S. gordonii* expressing the V3 domain of MN – the vaccine stimulated V3-specific IgG2a in mice [Oggioni (1999)]
- 257-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 257-D showed intermediate reactivity [Nyambi (2000)]
- 257-D: Called 257D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- 257-D: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization, suggesting that the change in TCLA lines that make them more susceptible to NAb alters some step after binding [York (2001)]
- 257-D: UK Medical Research Council AIDS reagent: ARP3023
- 257-D: NIH AIDS Research and Reference Reagent Program: 1510

403	311-11-D (311-11D, 311, 311D, 311-D)	gp160(305-313)	gp120()	KRIHIGP	L	HIV-1 infection	human(IgG1 λ)
<p>Ab type: V3 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (1991), Gorny (1993), Spear (1993), Gorny (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 311-11-D: Neutralizes MN – binds SF2: KSIYIGP [Gorny (1993)] • 311-11-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear (1993)] • 311-11-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)] • 311-11-D: MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)] • 311-11-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 311-11D showed weak reactivity [Nyambi (2000)] 							
404	41148D	gp160(305-313)	gp120(MN)	KRIHIGP	L	HIV-1 infection	human(IgG1)
<p>Ab type: V3 References: [Pinter (1993b), Alsmadi & Tilley(1998)]</p> <ul style="list-style-type: none"> • 41148D: Neutralizes less potently than 4117C, reacts with MN, IIIB, SF2 [Pinter (1993b)] • 41148D: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against strains IIIB, MN, SF-2, comparable to 4117C, however 41148D is 10x less efficient at neutralization, showing ADCC and neutralization don't always correlate [Alsmadi & Tilley(1998)] 							
405	391/95-D (391-95D, 391.5, 391/95D)	gp160(dis 305-318)	gp120(dis MN)	KRIHIGPGRAFY	L	HIV-1 infection	human(IgG1 κ)
<p>Ab type: V3 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (1991), Gorny (1993), Fontenot (1995), Stamatatos & Cheng-Mayer(1995), Seligman (1996), Stamatatos (1997), Stamatatos & Cheng-Mayer(1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Ly & Stamatatos(2000), Park (2000)]</p> <ul style="list-style-type: none"> • 391/95-D: Neutralizes MN – binds to SF2, not IIIB [Gorny (1993)] • 391/95-D: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V3 loop epitopes were less accessible to Ab binding on the virion surface than in the gp120 monomer, particularly for macrophage-tropic isolates SF162 and SF128a, relative to T-cell tropic SF2 – sCD4 association with gp120 better revealed this V3 epitope on macrophage tropic and dual tropic (MU3) viruses, but not in TCLA SF2 [Stamatatos & Cheng-Mayer(1995)] 							

Table of HIV MAbs

- 391/95-D: Competition ELISAs with serial deletions estimated the epitope to be KRIHIGPGRAFY – unconstrained peptide had higher affinity than cyclic [Seligman (1996)]
- 391/95-D: Called 391–95D – binds more extensively than MAb 257-D on the surface of HIV-1 isolates SF162 and SF128A – neutralizes more potently than 257-D – stronger neutralization of primary macrophage targets than PBMC – binding post-gp120-sCD4 association related to anti-V3 Abs neutralizing capacity [Stamatatos (1997)]
- 391/95-D: Called 391–95D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V3 MAbs 391–95D or 257D [Stamatatos & Cheng-Mayer(1998)]
- 391/95-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 391/95-D: Called 391.5 – MAb peptide-reactivity pattern clustered with immunological related MAbs: 391.5, 412 and 418, all selected with MN V3 peptide – the core amino acids HIGPGR tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 391/95-D: Called 391–95D – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391–95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]
- 391/95-D: Called 391/95D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]

406 Aw	gp160(305–320)	gp120(Gun-1wt)	KSITIGPGRAFHAI	L	Vaccine	rat()
Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> Gun-1 <i>HIV component:</i> V3						
Ab type: V3 References: [McKnight (1995)]						
• Aw: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – Aw gives weak neutralization of both wildtype and v strains [McKnight (1995)]						
407 Bw	gp160(305–320)	gp120(Gun-1wt)	KSITIGPGRAFHAI	L	Vaccine	rat()
Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> Gun-1 <i>HIV component:</i> V3						
Ab type: V3 References: [McKnight (1995)]						
• Bw: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – Bw gives weak neutralization of only the wildtype strain, does not bind to variant [McKnight (1995)]						
408 DO142–10 (DO 142–10)	gp160(305–320)	gp120(MN)	KRIHIGPGRAFYTT	L	HIV-1 infection	human Fab(IgG1)
Ab type: V3 References: [Seligman (1996), Ditzel (1997), Parren (1997b), Parren & Burton(1997), Parren (1998a), Sullivan (1998a)]						

- DO142–10: Fab fragment – competition ELISAs with serial deletions defined the epitope KRIHIGPGRAFYTT [Seligman (1996)]
- DO142–10: Phage expression libraries panned against MN peptide were used to select Fab DO142–10 – Fab binds MN gp120, but not a primary isolate rec gp120 [Ditzel (1997)]
- DO142–10: Neutralizes TCLA strains but not primary isolates [Parren (1997b)]
- DO142–10: Binds to gp120 MN and an MN V3 peptide with equal affinity, but binds a consensus B peptide and JRCSF less well, and to IIIB gp120 not at all [Parren & Burton(1997)]
- DO142–10: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142–10 > DA48 > L17) was markedly different that Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142–10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- DO124–10: The HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – Fab Ab fragment DO124–10 also enhances YU2 entry, ruling out Fc interactions or Env cross-linking as a mechanism – while DO124–10 enhances YU2 entry 6-fold, it neutralizes HXBc2 under identical conditions [Sullivan (1998a)]

409	Dv	gp160(305–320)	gp120(Gun-1v)	KSITIGSGRAFHAI	L	Vaccine	rat()
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> Gun-1 <i>HIV component:</i> V3</p> <p>Ab type: V3 References: [McKnight (1995)]</p> <ul style="list-style-type: none"> • Dv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight (1995)] 							
410	Fv	gp160(305–320)	gp120(Gun-1v)	KSITIGSGRAFHAI	L	Vaccine	rat()
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> Gun-1 <i>HIV component:</i> V3</p> <p>Ab type: V3 References: [McKnight (1995)]</p> <ul style="list-style-type: none"> • Fv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight (1995)] 							
411	Gv	gp160(305–320)	gp120(Gun-1v)	KSITIGSGRAFHAI	L	Vaccine	rat()
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> Gun-1 <i>HIV component:</i> V3</p> <p>Ab type: V3 References: [McKnight (1995)]</p> <ul style="list-style-type: none"> • Gv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight (1995)] 							
412	Hv	gp160(305–320)	gp120(Gun-1v)	KSITIGSGRAFHAI	L	Vaccine	rat()
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> Gun-1 <i>HIV component:</i> V3</p> <p>Ab type: V3 References: [McKnight (1995)]</p>							

Table of HIV MAbs

- Hv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight (1995)]

413	50.1 (R/V3– 50.1, Fab 50.1)	gp160(306–310)	gp120(MN)	RIHIG	L	Vaccine	murine(IgG1κ)
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Vaccine: *Vector/type:* peptide *Strain:* MN *HIV component:* V3

Ab type: V3 **Donor:** Mary White-Scharf, Repligen Corporation, Cambridge, MA

References: [D’Souza (1991), White-Scharf (1993), Potts (1993), Ghiara (1993), Rini (1993), Bou-Habib (1994), VanCott (1994), Robert-Guroff (1994), Moore (1994b), VanCott (1995), Fontenot (1995), Seligman (1996), Berman (1997), LaCasse (1998), Stanfield (1999), Hoffman (1999), Park (2000), York (2001)]

- 50.1: Called R/V3–50.1 – potent neutralizing of lab strains[D’Souza (1991)]
- 50.1: Epitope defined by peptide reactivity and changes affinity with amino acid substitutions – epitope RIHIGP [White-Scharf (1993)]
- 50.1: No synergistic neutralization of MN when combined with CD4BS MAb F105 – isotype stated to be IgG2a [Potts (1993)]
- 50.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 and 50.1 Fab fragments – epitope KRIHIGP [Ghiara (1993)]
- 50.1: Crystal structure of V3 loop bound to 50.1 – light chain binds just to the left of GPG, heavy chain binds further to the left [Rini (1993)]
- 50.1: No neutralization of primary isolate JR-CSF – greater affinity for and neutralization of T cell tropic strain T-CSF, derived from JR-CSF [Bou-Habib (1994)]
- 50.1: Potent MN neutralization, slow dissociation rate [VanCott (1994)]
- 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FACS signal, Ab affinity, and viral neutralization [Robert-Guroff (1994)]
- 50.1: Shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore (1994b)]
- 50.1: Used to monitor HIV-1 Env expression in infected H9 cells [VanCott (1995)]
- 50.1: Competition ELISAs with serial deletions produced comparable estimate of epitope length to crystal structure and alanine substitution – KRIHIGP [Seligman (1996)]
- 50.1: Binds to 6/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)]
- 50.1: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by anti-V3 MAbs – the primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse (1998)]
- 50.1: The crystal structure of V3 loop peptides bound to Fabs was obtained – conformational changes in the tip of the V3 loop (GPGR) were observed when different Fabs were bound [Stanfield (1999)]
- 50.1: A CD4-independent viral variant of IIIB, IIIBx, was generated on CXCR4-expressing cells – IIIBx exhibited enhanced neutralization of by CD4i MAbs and by polyclonal human sera but not by anti-V3 MAb 50.1 [Hoffman (1999)]

- 50.1: Called R/V3–50.1 – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes – 50.1 could only neutralize the sensitive form [Park (2000)]
- 50.1: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAb alters some step after binding – the dissociation constant, K_d of 50.1 for the cell associated primary and TCLA Envs was equal, 7nM [York (2001)]
- 50.1: NIH AIDS Research and Reference Reagent Program: 1289

414	polyclonal	gp160(306–318)	gp120(NY5)	KKGIAIGPGRTLY			(IgM)
		Ab type: V3	References: [Metlas (1999b), Metlas (1999a)]				
		• Auto-Abs that react with the V3 loop of NY5 are present in the sera of HIV- individuals, and are predominantly IgM [Metlas (1999b)]					
415	BAT123 (BAT-123, CGP 47 439)	gp160(306–322)	gp120(308–322 HXB2)	RIRIQRGPGRAFVTIGK	L	Vaccine	murine(IgG1 κ)

Vaccine: *Vector/type:* inactivated virus *Strain:* IIIB *HIV component:* virus

Ab type: V3 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY

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

- BAT123: CGP 47 439 is a BAT123 chimera that has a human IgG1 Fc domain
- BAT123: Anti-idiotypic MAb, AB19–4i, stimulates anti-anti-ID which neutralizes MN and IIIB [Fung (1990)]
- BAT123: Called BAT-123 – conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120 – does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- BAT123: Passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus [Safrit (1993)]
- BAT123: Variable region sequenced – heavy chain: V 3660-SB32, D unknown, J H3 – light chain: V κ 21, J κ 2 [Pirofski (1993)]
- BAT123: Passive transfer of BAT123 to hu-PBL-SCID mice 1 hour prior to inoculation with HIV-1 LAI, or up to four hours post-exposure, could protect mice from infection – the protection, like the MAb, was specific for the viral strain LAI [Gauduin (1995)]
- BAT123: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strain [Sattentau & Moore(1995)]
- BAT123: Epitope described as RGPGRFVVTIGK – V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus (BAT123 less so than the others), mimicking sCD4, and expose the gp41 epitope for MAb 50–69, in contrast to anti-V2 MAbs [Poignard (1996a)]
- BAT123: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIV-1 LAI in hu-PBL-SCID mice, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus (1998)]

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- BAT123: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- BAT123: Post-exposure passive transfer of murine BAT123 can confer protection to hu-PBL-SCID mice challenged with HIV-1 LAI – this protection is not elicited by CGP 47 439, a BAT123 chimera that has a human IgG1 Fc domain, suggesting that the protection is mediated by complement – the protective ability of BAT123 is lost when mice were treated with cobra venom factor, which inactivates serum complement – IgG1 does not fix complement efficiently so an IgG2 MAb might perform better [Gauduin (1998)]

416	838-D (838)	gp160(307–311)	Env(RF)	KSITK	L	HIV-1 infection	human(IgG1 λ)
<p>Ab type: V3 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (1997), Nyambi (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Gorny (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 838-D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 838-D was cross-reactive with V3 peptides from clade A and C, and could bind to 5/8 B clade V3 peptides – 50% neutralization of RF was obtained [Gorny (1997)] • 838-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 838-D bound B clade virions but had limited cross-reactivity with other clades, with low levels of binding to A and D virions [Nyambi (1998)] • 838-D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to many A, B, C and F peptides, poor binding to D and E [Zolla-Pazner (1999a)] • 838-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)] • 838-D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V3 MAbs 447-52D, 838-D, and 1334 bound with a 7–10 fold preference for the oligomer [Gorny (2000)] • 838-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 838-D showed intermediate reactivity [Nyambi (2000)] 							
417	1006–15D (1006)	gp160(307–312)	gp120(RF)	KSITKG	no	HIV-1 infection	human(IgG1 λ)
<p>Ab type: V3 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 1006–15D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – was somewhat cross-reactive with V3 peptides from clade A, C and other B clade V3 peptides, but not E clade [Gorny (1997)] • 1006–15D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to several B and F peptides, one C peptide, and some reactivity with A peptides – no binding was observed with D and E peptides [Zolla-Pazner (1999a)] • 1006–15D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)] 							

- 1006–15D: A panel of 47 human MABs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MABs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 1006–15D showed strong cross-reactivity [Nyambi (2000)]

418	782-D (782)	gp160(307–312)	Env(RF)	KSITKG	L	HIV-1 infection	human(IgG1 λ)
<p>Ab type: V3 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 782-D: Five human MABs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 782-D was not cross-reactive with V3 peptides from clade A or E, but could bind to 3/8 B clade V3 peptides, and 1/2 C clade V3 peptides – 50% neutralization of RF was obtained [Gorny (1997)] • 782-D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to several B and F peptides, one C peptide, and some reactivity with A and D peptides [Zolla-Pazner (1999a)] • 782-D: MAb peptide-reactivity pattern clustered with immunological related MABs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)] • 782-D: A panel of 47 human MABs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MABs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 782-D showed intermediate reactivity [Nyambi (2000)] 							
419	908-D (908, 908–12D)	gp160(307–312)	gp120(RF)	KSITKG	L	HIV-1 infection	human(IgG1 λ)
<p>Ab type: V3 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 908-D: Five human MABs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 908-D was not cross-reactive with V3 peptides from clade E, but could bind to 6/8 B clade V3 peptides, 2/4 A clade, and 1/2 C clade – 50% neutralization of RF was obtained [Gorny (1997)] • 908-D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to several A, B, C and F peptides, and poor binding to E and D peptides [Zolla-Pazner (1999a)] • 908-D: MAb peptide-reactivity pattern clustered with immunological related MABs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)] • 908-D: A panel of 47 human MABs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MABs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 908-D showed strong cross-reactivity, but achieved only 50% neutralization on 2/5 isolates tested [Nyambi (2000)] 							
420	1027–15D (1027, 1027-D, 1027D)	gp160(307–313)	Env(RF)	KSITKGP	no	HIV-1 infection	human(IgG1 λ)
<p>Ab type: V3 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]</p>							

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

421	F19.26–4	gp160(307–319)	gp120(312–324 LAI)	IRIQRGPGRAFVT	L	Vaccine	murine(IgG2a κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: V3 References: [Boudet (1994)]</p> <ul style="list-style-type: none"> • F19.26–4: Strain specific – used to raise anti-idiotypic antibodies [Boudet (1994)] 							
422	F19.48–3	gp160(307–319)	gp120(312–324 LAI)	IRIQRGPGRAFVT	L	Vaccine	murine(IgG2a κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: V3 References: [Boudet (1994)]</p> <ul style="list-style-type: none"> • F19.48–3: Strain specific – used to raise anti-idiotypic antibodies [Boudet (1994)] 							
423	F19.57–11	gp160(307–319)	gp120(312–324 LAI)	IRIQRGPGRAFVT	L (LAI)	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: V3 References: [Boudet (1991), Boudet (1994), Boudet (1995)]</p> <ul style="list-style-type: none"> • F19.57–11: MAb F19.57–11 is strain specific for LAI – used to raise anti-idiotypic rabbit antibodies (called 57-B Ab2) [Boudet (1994)] • F19.57–11: Anti-anti-idiotypic antibodies (Ab3) were raised in BALBc mice that had greater breadth of reactivity than the original F19.57–11 (Ab3 could also recognize 1282 and SF2, with aa TRK(R or S)IYIGPGRA(WY or FH)T) [Boudet (1995)] 							
424	M77	gp160(307–320)	gp120(IIIB)	IRIQRGPGRAFVTI	L	HIV-1 infection	human(IgG)
<p>Ab type: V3 Donor: Advanced BioScience Laboratories, Rockville, MD, commercial</p> <p>References: [Pal (1992), di Marzo Veronese (1992), di Marzo Veronese (1993), Watkins (1993), Cook (1994), DeVico (1995), Denisova (1995), Watkins (1996), Denisova (2000)]</p> <ul style="list-style-type: none"> • M77: IIIB-specific MAb, immunoprecipitates deglycosylated form [di Marzo Veronese (1992)] • M77: Antibody binding to viral isolates from IIIB infected lab worker followed through time – A to T substitution resulted in the loss of neutralization and native gp120 binding, but not peptide binding [di Marzo Veronese (1993)] • M77: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer <i>in vitro</i> [Cook (1994)] 							

- M77: Reacted with both reduced and non-reduced covalently cross-linked gp120-CD4 complex [DeVico (1995)]
- M77: Conformational rearrangements upon binding of M77 to gp120 generates novel epitopes called metatopes [Denisova (1995)]
- M77: Stated to be a murine MAb – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – M77 neutralization was only slightly reduced by this mutation [Watkins (1993)]
- M77: Used M77 bound to gp120 as an immunogen – analysis of polyclonal and monoclonal (62 MAbs were generated) response suggests the M77-gp120 immunogen generated MAbs to more linear epitopes than gp120 alone or gp120 bound to CD4 [Denisova (1996)]
- M77: Native M77 is highly strain specific, and V3 binding is primarily dependent on its heavy chain – a light chain switched Fab version of M77 could recognize HIV-1 strains that had substitutions on the left side of the V3 loop – R in GPGR is likely to be critical for binding [Watkins (1996)]
- M77: M77 is highly strain specific for IIIB, but anti-idiotypic Abs directed against M77 can in turn elicit an Ab response with expanded HIV cross-reactivity – this mechanism may serve to prolong the primary response and to counter-balance viral immune evasion by mutation [Denisova (2000)]

425	SP.BAL114	gp160(308–317)	gp120(BAL)	SIHIGPGRAF	L		murine?(IgG2a κ)
		Ab type: V3		References: [Arendrup (1995)]			
							<ul style="list-style-type: none"> • Authors suggest that during <i>in vivo</i> immunoselection of escape virus, the V3 domain gains increasing resemblance to that of lab strains [Arendrup (1995)]
426	SP.SF2:104	gp160(308–317)	gp120(SF2)	SIYIGPGRAF	L	HIV-1 infection	(IgG2a κ)
		Ab type: V3		References: [Arendrup (1993), Arendrup (1995)]			
							<ul style="list-style-type: none"> • SP.SF2:104: Anti-V3 antibody that could neutralize primary virus isolated from a time point of neutralization resistance of autologous virus [Arendrup (1993)] • SP.SF2:104: Authors suggest that during <i>in vivo</i> immunoselection of escape virus, the V3 domain gains increasing resemblance to lab strains [Arendrup (1995)]
427	polyclonal	gp160(308–319)	gp120(304–318 LAI)	RIHIGPGRAFYT		HIV-1 infection	human(IgG,IgM)
		Ab type: V3		References: [Langedijk (1995)]			
							<ul style="list-style-type: none"> • Polyclonal sera from six individuals tested for reactivity against a panel of peptides based on autologous sequences provide evidence for immunological escape mutations in the tip of the V3 loop [Langedijk (1995)]
428	19b	gp160(308–320)	gp120()	-I—G—FY-T	L	HIV-1 infection	human(IgG1)
		Ab type: V3		Donor: James Robinson, University of Connecticut, Storrs			
				References: [Scott (1990), Moore (1994b), Moore (1994a), Sattentau(1995), Moore (1995b), Moore (1995a), Moore & Ho(1995), Gauduin (1996), Wu (1996), Trkola (1996a), D'Souza (1997), Binley (1997a), Fouts (1997), Ugolini (1997), Boots (1997), Parren (1997b), Mondor (1998), Parren (1998a), Trkola (1998), Binley (1999), Park (2000), Kolchinsky (2001)]			
							<ul style="list-style-type: none"> • 19b: V3 loop binding MAb that is more broadly clade cross-reactive than most (binds to 19/29 clade B and 10/12 clade E gp120s) [Moore (1994b)]

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- 19b: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore (1994a)]
- 19b: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau (1995)]
- 19b: Binds to some gp120s from clades A,B,C,E, and F – weakly neutralized some B and one C clade virus [Moore (1995b)]
- 19b: Despite broad gp120 binding reactivity, not broadly neutralizing [Moore (1995a)]
- 19b: Review: more broadly cross-reactive than anti-V3 tip MAb 447-D [Moore & Ho(1995)]
- 19b: Not as effective as IgG1b12 at neutralization *ex vivo* of virus direct from plasma of HIV-1 infected individuals [Gauduin (1996)]
- 19b: MIP-1 α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of 19b blocks this inhibition [Wu (1996)]
- 19b: Inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola (1996a)]
- 19b: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – there were four sequences with variations in the defined epitope among the 9 isolates tested [D'Souza (1997)]
- 19b: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 19b bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)]
- 19b: Viral binding inhibition by 19b was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- 19b: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – 19b has an epitope involving the tip of the V3 loop, with 5 or 6 essential amino acids distributed within a 12 amino acid stretch – the previously determined binding site was confirmed -I—G—FY-T and some tolerated variants described, the I can be I, V, or L, the Y can be Y, F, or W – probably a β -turn is required for FY or FF binding, but WY in can bind with out the context of the turn [Boots (1997)]
- 19b: Neutralizes TCLA strains but not primary isolates [Parren (1997b)]
- 19b: Used as a control in this Hx10 binding and neutralizing MAb study because 19b does not bind to Hx10 [Mondor (1998)]
- 19b: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- 19b: No detectable neutralizing activity among primary isolates with different co-receptor usage – some neutralization of TCLA strains [Trkola (1998)]
- 19b: The MAbs with the broadest neutralizing activity, 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 [Binley (1999)]
- 19b: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form but 19b was an exception and required around 950 ng/ml to neutralize either form [Park (2000)]

- 19b: Mutations in two glycosylation sites in the V2 region of HIV-1 ADA at positions 190 and 197 (187 DNTSYRLINCNTS 199) cause the virus to become CD4-independent and able to enter cells through CCR5 alone – these same mutations tended to increase the neutralization sensitivity of the virus, including to 19b [Kolchinsky (2001)]

429	4G10	gp160(308–322)	gp120(308–322 LAI)	RIQRGPGRAFVTGK		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> HBcAg fusion <i>HIV component:</i> V3</p> <p>Ab type: V3 Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany</p> <p>References: [von Brunn (1993)]</p> <ul style="list-style-type: none"> • 4G10: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn (1993)] • 4G10: NIH AIDS Research and Reference Reagent Program: 2534 							
430	5F7	gp160(308–322)	gp120(308–322 LAI)	RIQRGPGRAFVTGK		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> HBcAg fusion <i>HIV component:</i> V3</p> <p>Ab type: V3 Donor: Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany</p> <p>References: [von Brunn (1993)]</p> <ul style="list-style-type: none"> • 5F7: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn (1993)] • 5F7: NIH AIDS Research and Reference Reagent Program: 2533 							
431	G3-523	gp160(308–322)	gp120(308–322)	RIQRGPGRAFVTIGK			murine()
<p>Ab type: V3 References: [Matsushita (1988), Jagodzinski (1996)]</p> <ul style="list-style-type: none"> • G3-523: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits G3-523 binding [Jagodzinski (1996)] 							
432	MN215	gp160(308–322)	gp120(MN)	RIHIGPGRAFYTTKN	L	HIV-1 infection	human(IgG1)
<p>Ab type: V3 References: [Schutten (1995b)]</p> <ul style="list-style-type: none"> • MN215: Minimum epitope for MAB using the Dutch consensus is AFYTTGE, different than defined for MN – generated by EBV transformation of PBMC – displayed higher affinity for NSI than for SI glycoproteins – amino acids HIGP were essential for binding [Schutten (1995b)] 							
433	Nea 9301	gp160(308–323)	gp120(IIIB)	RIQRGPGRAFVTIGKI			murine()
<p>Ab type: V3 Donor: Dupont, commercial</p> <p>References: [Wagner (1996)]</p>							
434	4117C	gp160(309–315)	gp120()	IXIGPGR	L	HIV-1 infection	human(IgG1λ)
<p>Ab type: V3 References: [Tilley (1991a), Tilley (1992), di Marzo Veronese (1993), Pinter (1993a), Pinter (1993b), Alsmadi & Tilley(1998)]</p> <ul style="list-style-type: none"> • 4117C: Potent neutralizing activity against MN, SF-2, and NY-5 – synergy with CD4BS MAb 1125H [Tilley (1991a)] 							

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

435	419-D (419, 419D)	gp160(309–315)	gp120(MN)	IHIGPGR	L	HIV-1 infection	human(IgG1λ)
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- Ab type:** V3 **Donor:** Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)
- References:** [Karwowska (1992b), Gorny (1993), Spear (1993), Fontenot (1995), Nyambi (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]
- 419-D: MN, NY5 and SF2 strain specific, does not cross-react with RF, CDC4, WM52 or HXB2 [Karwowska (1992b)]
 - 419-D: Neutralizes MN – binds SF2: IYIGPGR [Gorny (1993)]
 - 419-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear (1993)]
 - 419-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 419-D bound to 3/4 B clade virions, and to D clade MAL [Nyambi (1998)]
 - 419-D: Review of clade specificity and anti-V3 HIV-1-Abs – epitope is described as KRIHIGP [Zolla-Pazner (1999a)]
 - 419-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
 - 419-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 419-D showed intermediate reactivity, and no neutralization when tested against five strains – discrepancy between the epitope as described in earlier papers and as described here, KRIHIGP [Nyambi (2000)]

436	453-D (453)	gp160(309–315)	gp120(MN)	IHIGPGR	L	HIV-1 infection	human(IgG1λ)
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- Ab type:** V3 **Donor:** Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)
- References:** [Gorny (1991), Gorny (1993), VanCott (1994), Fontenot (1995), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]
- 453-D: Neutralizes MN – binds SF2: IYIGPGR – specificity: MN, SF2, NY5, RF [Gorny (1993)]
 - 453-D: Moderate homologous neutralization, moderately slow dissociation rate [VanCott (1994)]
 - 453-D : Called 453, epitope described as KRIHIGPGR – the tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot (1995)]
 - 453-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
 - 453-D : MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group – MAb 268, with a previously defined core epitope identical to 453 (HIGPGR), was not part of this reactivity group, illustrating that context can be critical [Zolla-Pazner (1999b)]

- 453-D: A panel of 47 human MABs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MABs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 453-D showed intermediate reactivity [Nyambi (2000)]

437	504-D (504, 504-10D)	gp160(309-315)	gp120(MN)	IHIGPGR	L	HIV-1 infection	human(IgG1 κ)
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Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcr6.med.nyu) (NYU Med. Center)

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

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

438	83.1 (MAb 83.1)	gp160(309-315)	gp120(SF2)	IYIGPGR	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* peptide *Strain:* MN *HIV component:* V3

Ab type: V3 **Donor:** Mary White-Scharf, Repligen Corporation, Cambridge, MA

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

- 83.1: Neutralizes SF2 [White-Scharf (1993)]
- 83.1: Study of synergism of neutralization and binding comparing F105 and sCD4 with the V3 MABs: 50.1, 59.1, 83.1, and 58.2 – synergy was observed, and the data suggest that binding of one ligand (F105) can increase the binding of the second (*e.g.* V3 loop MABs) due to conformational changes [Potts (1993)]
- 83.1: Maternally transferred anti-V3 loop MAb selectively inhibits the anti-V3 loop Ab component of the IgG response to gp120 SF2 in 21 day old BALBc mice [Jelonek (1999)]
- 83.1: 19 day old mice injected with 83.1 have a shift in IgG1 response away from the V3 loop upon vaccination, without decreasing the total IgG anti-gp120 response, suggesting that prior treatment with a MAb can mask immunogenic sites and shift the immune response to vaccination [Keller & Arora(1999)]

Table of HIV MAbs

- 83.1: 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 [Binley (1999)]

439	5023B	gp160(309–316)	gp120(309–316 BH10)	IQRGPGra	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> BH10 <i>HIV component:</i> V3</p> <p>Ab type: V3 References: [Langedijk (1991)]</p> <ul style="list-style-type: none"> • 5023B: Generation and fine mapping of murine MAbs [Langedijk (1991)] 							
440	F58/D1 (F58)	gp160(309–316)	gp120(IIIB)	IxxGPGRA	L	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> virus derived protein <i>HIV component:</i> gp120</p> <p>Ab type: V3 References: [Akerblom (1990), Broliden (1991), Moore (1993b), Millar (1998), Jackson (1999)]</p> <ul style="list-style-type: none"> • F58/D1: Binding to native gp120 1–3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)] • F58/D1: The interaction of a 17-amino-acid neutralizing microantibody (MicroAB) based on F58 and HIV-1 env was studied by electrospray ionization mass spectrometry [Millar (1998)] • F58/D1: A 17 amino acid MicroAB was made from the third complementarity-determining region of the heavy chain of MAb – F58 neutralized 5x's more efficiently in terms of mass than the original MAb, 32-fold less on a molar basis – neutralization does not involve initial attachment, but fusion and events in early infection [Jackson (1999)] 							
441	P1/D12	gp160(309–316)	gp120()	IxxGPGRA	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> virus derived protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: V3 References: [Akerblom (1990), Moore (1993b)]</p> <ul style="list-style-type: none"> • P1/D12: Binding to native gp120 1–3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)] 							
442	P4/D10 (P4D10)	gp160(309–316)	gp120()	IxxGPGRA	L	Vaccine	murine(IgG1κ)
<p>Vaccine: <i>Vector/type:</i> virus derived protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p>							

- Ab type:** V3 **References:** [Akerblom (1990), Broliden (1990), Broliden (1991), Marks (1992), Moore (1993b), Arendrup (1993), Hinkula (1994), Jacobson(1998), Schonning (1998), Schonning (1999)]
- P4/D10: Neutralizing and ADCC activity [Broliden (1990)]
 - P4/D10: Variable domain sequenced and is identical to F58/H3 [Marks (1992)]
 - P4/D10: Binding to native gp120 3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)]
 - P4/D10: Primary isolates from different time points from one individual were not susceptible to neutralization by P4/D10 [Arendrup (1993)]
 - P4/D10: Used for passive immunotherapy in four late-stage HIV-infected patients – the serum level of p24 did not decrease in any of these four – see also MAb F58/H3 [Hinkula (1994)]
 - P4/D10: Review of passive immunotherapy, summarizing [Hinkula (1994)] in relation to other studies [Jacobson(1998)]
 - P4/D10: Called P4D10 – In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, anti-V3 MAbs were found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan more efficiently than HIV-BRU – Ab binding site was suggested to be 314–323 of BRU [Schonning (1998)]
 - P4/D10: Called P4D10 – the stoichiometry of MAb neutralization was tested and the data indicated that binding for neutralization was incremental not all or none, *i.e.*, each envelope oligomer binds a single MAb and each Env oligomer bound reduces the chances of infection – MAb BC1071 was used for virion quantitation – P4D10 binds only to Env with a glycosylation site mutation at the base of the V3 loop A308T [Schonning (1999)]

443	IIIB-13 V3 (1044–13, IIIB-V3–13)	gp160(309–317)	gp120(308–316 IIIB)	IQRGPGRAF	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* peptide *Strain:* IIIB

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

444	IIIB-34 V3 (IIIB-V3–34)	gp160(309–317)	gp120(308–316 IIIB)	IQRGPGRAF	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* peptide *Strain:* IIIB

- Ab type:** V3 **References:** [Laman (1992), Laman (1993)]
- IIIB-34 V3: Neutralizes IIIB but not MN – QXGPG are critical amino acids for binding by Pepscan analysis [Laman (1992)]

Table of HIV MAbs

- IIIB-34 V3: Called IIIB-V3-34 – IIIB strain specific neutralization – binding is reduced somewhat by DTT or SDS-DTT, enhanced by NP40, but binds to native and denatured gp120 [Laman (1993)]
- IIIB-34 V3: UK Medical Research Council AIDS reagent: ARP3047

445	A47/B1	gp160(309–318)	gp120(307–316 IIIB)	IQRGPGRAFV	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120 Ab type: V3 References: [Akerblom (1990)]</p>							
446	D59/A2	gp160(309–318)	gp120(307–316 IIIB)	IQRGPGRAFV	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120 Ab type: V3 References: [Akerblom (1990)]</p>							
447	G44/H7	gp160(309–318)	gp120(307–316 IIIB)	IQRGPGRAFV	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120 Ab type: V3 References: [Akerblom (1990)]</p>							
448	μ 5.5 (5.5, μ 5.5, R μ 5.5)	gp160(309–319)	gp120(MN)	IHIGPGRAFYT	P L		murine(IgG1 κ)
<p>Ab type: V3 References: [Maeda (1992), Okamoto (1998)]</p> <ul style="list-style-type: none"> • μ5.5: sCD4 causes loss of IIIB type-specificity for MAb 0.5β, allowing binding and neutralization of MN, in contrast to MAb μ5.5 [Maeda (1992)] • μ5.5: Rμ5.5 is a humanized antibody of mouse MAb m5.5 – neutralized primary isolates with similar V3 loops – passive transfer of MAb to SCID-hu or hu-PBL-SCID mice conferred protection [Okamoto (1998)] 							
449	loop 2 (Loop 2, IgG1 Loop 2)	gp160(309–320)	gp120()	SISGPGRAFYTG	L	HIV-1 infection	human Fab()
<p>Ab type: V3 Donor: D. Burton, Scripps Research Institute, La Jolla, CA References: [Barbas III (1993), Moore (1994b), Wu (1996), Ditzel (1997), Ugolini (1997), Parren (1997b), Parren & Burton(1997), Mondor (1998), Parren (1998a), Sullivan (1998a)]</p> <ul style="list-style-type: none"> • loop2: Also known as Loop 2, IgG1 Loop 2 was a obtained by engineering Fab loop2 into an IgG1 molecule • loop 2: Sequences of the heavy and light chain Fab variable regions were generated [Barbas III (1993)] • loop 2: Called Loop 2 – shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore (1994b)] • loop 2: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of loop 2 blocks this inhibition [Wu (1996)] • loop 2: Binds to gp120 from MN and SF2 but not LAI [Ditzel (1997)] • loop 2: Viral binding inhibition by loop 2 MAb or Fab was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)] 							

- loop 2: Epitope is suggested to be GPGRAF – binds to 10/17 US clade B monomeric gp120s – IgG1 form can neutralize MN and 2 primary isolates tested [Parren & Burton(1997)]
- loop 2: Neutralizes TCLA strains but not primary isolates [Parren (1997b)]
- loop 2: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142–10 > DA48 > L17) was markedly different than Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142–10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope – binding affinity of divalent IgG1 loop 2 is only 2-fold greater than monovalent Fab loop 2, suggesting the IgG1 form may bind with only one arm [Parren (1998a)]
- loop 2: The HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – loop 2 enhances YU2 at concentrations up to 20 $\mu\text{g/ml}$ [Sullivan (1998a)]

450	268-D (268–11-D-IV, 268D, 268, 268–11D, 268–10D, MAb 268, 268–10-D)	gp160(dis 310–315)	gp120(dis MN)	HIGPGR	L	HIV-1 infection	human(IgG1 λ)
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Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Gorny (1991), D'Souza (1991), Karwowska (1992b), Gorny (1993), Spear (1993), VanCott (1994), Stamatatos & Cheng-Mayer(1995), Zolla-Pazner (1995), Fontenot (1995), McKeating (1996), Wisniewski (1996), Stamatatos (1997), LaCasse (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Beddows (1999), Oggioni (1999), Laisney & Strosberg(1999), Hioe (2000), Nyambi (2000), Park (2000), York (2001)]

- 268-D: Called 268–11-D-IV – strain specific weakly neutralizing [D'Souza (1991)]
- 268-D: Reacts with MN, NY5, CDC4, RF and SF2, does not cross-react with WM52 or HXB2 [Karwowska (1992b)]
- 268-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny (1993)]
- 268-D: Mediated deposition of complement component C3 on HIV infected cells, but not in the presence of sCD4 [Spear (1993)]
- 268-D: Moderate dissociation rate and homologous neutralization titer [VanCott (1994)]
- 268-D: Serotyping study using flow-cytometry, if H of HIGPGR was substituted in virus, 268-D did not bind [Zolla-Pazner (1995)]
- 268-D: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V3 loop epitopes were less accessible to Ab binding on the virion surface than in the gp120 monomer, particularly for macrophage-tropic isolates SF162 and SF128a, relative to T-cell tropic SF2 – sCD4 association with gp120 did not influence the binding of 268-D to virion-associated gp120, although sCD4 binding did alter epitope exposure for other anti-V3 MAbs [Stamatatos & Cheng-Mayer(1995)]
- 268-D: Failed to neutralize HXB2 and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]

Table of HIV MAbs

- 268-D: 268-D is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisniewski (1996)]
- 268-D: Poor reactivity against HIV-1 isolates SF162 and SF128A and no neutralization, in contrast to MAbs 391/95-D and 257-D [Stamatatos (1997)]
- 268-D: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by anti-V3 MAbs – the primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse (1998)]
- 268-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 268-D: Peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group – MAb 453, with an identical core epitope to 268 based on prior experiments (HIGPGR), was not part of this reactivity group, illustrating that context can be critical [Zolla-Pazner (1999b)]
- 268-D: rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – 268-D bound rgp120 W61D but could only neutralize the W61D isolate following T-cell line adaptation [Beddows (1999)]
- 268-D: Called 268–11D – Study of a live-vector mucosal vaccine that expresses HIV-1 V3 domains using the bacterium *Streptococcus gordonii* which can express heterologous Ag and can colonize the oral cavity and vagina of mice – 268-D and 257-D recognized *S. gordonii* expressing the V3 domain of MN – the vaccine stimulated V3-specific IgG2a in mice [Oggioni (1999)]
- 268-D: Called MAb 268 – To identify potential mimotopes of V3, a hexapeptide phage library was screened with MAb 268 – two hexamers were identified, HLGPGR or KAIHRI that bind to 268 with the same binding site as the V3 loop and inhibit 268 MN gp120 – KLH conjugated hexamer KAIHRI stimulates Abs in rabbits that cross-react with ML gp120 [Laisney & Strosberg(1999)]
- 268-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – V3 MAbs 447-52-D and 268–10-D did not effect proliferation [Hioe (2000)]
- 268-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 268-D showed weak reactivity [Nyambi (2000)]
- 268-D: Called 268D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- 268-D: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding – one of the TCLA V3 viruses 320SI-C3.3 shows reduced binding with this MAb, the sequence of the epitope in 320SI is HIGPGR and in 320SI-C3.3 is RIGPGR [York (2001)]
- 268-D: UK Medical Research Council AIDS reagent: ARP3024
- 268-D: NIH AIDS Research and Reference Reagent Program: 1511

451 386-D (386, gp160(310–315) gp120(MN) HIGPGR L HIV-1 infection human(IgG1λ)
386–10D,
386D)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)

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

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

452 5042A gp160(310–315) gp120(310–315 BH10) QrGPGR L Vaccine murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **References:** [Langedijk (1991), Gorny (1991)]

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

453 5042B gp160(310–315) gp120(310–315 BH10) QRGPGGr no Vaccine murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **References:** [Langedijk (1991)]

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

454 418-D (418, gp160(310–316) gp120(MN) HIGPGR L HIV-1 infection human(IgG1κ)
418D)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)

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

- 418-D: MN strain specific, does not cross-react with SF2, NY5, RF, CDC4 WM52 or HXB2 [Karwowska (1992b)]
- 418-D: Neutralizes MN, does not bind to SF2 or HXB2 [Gorny (1993)]
- 418-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 418-D: Called 418 – MAb peptide-reactivity pattern clustered with immunological related MAbs: 391.5, 412 and 418, all selected with MN V3 peptide – the core amino acids HIGPGR tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]

Table of HIV MAbs

- 418-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 418-D showed intermediate reactivity [Nyambi (2000)]

455	5021	gp160(310–316)	gp120()	QrGPGRa	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> BH10 <i>HIV component:</i> V3</p> <p>Ab type: V3 References: [Durda (1988), Durda (1990), Langedijk (1991), Moore (1993b)]</p> <ul style="list-style-type: none"> • 5021: Generation and fine mapping of murine MAbs [Langedijk (1991)] • 5021: Binding to native gp120 100–300 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)] 							
456	5025B	gp160(310–316)	gp120(310–316 BH10)	QRGPGRa	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> BH10 <i>HIV component:</i> V3</p> <p>Ab type: V3 References: [Langedijk (1991)]</p> <ul style="list-style-type: none"> • 5025B: Generation and fine mapping of murine MAbs [Langedijk (1991)] 							
457	5042	gp160(310–316)	gp120()	QRGPGRa	L	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> peptide</p> <p>Ab type: V3 References: [Durda (1988), Durda (1990), Moore (1993b)]</p> <ul style="list-style-type: none"> • 5042: Binding to native gp120 100–300 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)] 							
458	110.3	gp160(310–317)	gp120(308–328 BRU)	QRGPGRaF	L	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> infected-cell lysate <i>Strain:</i> BRU <i>HIV component:</i> virus</p> <p>Ab type: V3 References: [Thomas (1988), Evans (1989), Langedijk (1992), Pirofski (1993), Connelly (1994)]</p> <ul style="list-style-type: none"> • 110.3: Included as a control [Evans (1989)] • 110.3: MAb variable region sequenced – heavy chain: V 7138(40), D deletion, J H4 – light chain: V κ21(47), J κ2 [Pirofski (1993)] • 110.3: An anti-idiotypic MAb generated against 110.3 both mimics and binds to V3, suggesting that the V3 loop may associated with itself [Connelly (1994)] 							
459	110.4	gp160(310–317)	gp120(308–328 BRU)	QRGPGRaF	L	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> infected-cell lysate <i>Strain:</i> BRU <i>HIV component:</i> virus</p> <p>Ab type: V3 Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas</p> <p>References: [Thomas (1988), Thali (1992b), Langedijk (1992), Thali (1993), Pirofski (1993), Arendrup (1993), Thali (1994), Boudet (1994), Connelly (1994), McDougal (1996), Valenzuela (1998), Cao (1997), Guillerm (1998)]</p>							

- 110.4: 313 P/S substitution in the V3 region disrupts binding [Thali (1992b)]
- 110.4: MAb variable region sequenced – heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J H2 – light chain: V κ 21, J κ 2 [Pirofski (1993)]
- 110.4: Primary isolates from different time points from one individual were not susceptible to neutralization by 110.4 [Arendrup (1993)]
- 110.4: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali (1994)]
- 110.4: An anti-idiotypic MAb generated against 110.3 also blocks binding of 110.4 [Connelly (1994)]
- 110.4: Neutralizes HIV-1 LAI [McDougal (1996)]
- 110.4: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11–20 is through inhibition of viral binding to the cell [Valenzuela (1998)]
- 110.4: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao (1997)]
- 110.4: Used for flow cytometry in a study of the anti-CD4, CDR3 loop MAb called 13B8.2, in a study of HIV-1 induced programmed cell death [Guillerm (1998)]

460	110.5	gp160(310–317)	gp120(308–328 BRU)	QRGPGRAF	L	Vaccine	murine(IgG1 κ)
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Vaccine: *Vector/type:* infected-cell lysate *Strain:* BRU *HIV component:* virus

Ab type: V3 **Donor:** E. Kinney-Thomas or Genetic Systems, Seattle WA

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

- 110.5: Did not induce dissociation of gp120, as sCD4 did – discrepancy with [Poignard (1996a)], that was suggested to be due to MAb interference with detection, as the gp120-MAb complex was denatured in the Poignard study [Moore (1990)]
- 110.5: Binding insensitive to gp120 reduction [Cordell (1991)]
- 110.5: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 110.5: Variable region sequenced – heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J H2 – light chain: V κ 21, J κ 2 [Pirofski (1993)]
- 110.5: Thrombin cleavage of V3 loop between R-315 and A-316 abrogates binding – can inhibit C4 region antibody which has conformational requirements (G3-299) – binding to native gp120 100–300 fold greater than to denatured [Moore (1993b)]
- 110.5: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 110.5 is not affected [Reitz (1988), Klasse (1993a)]
- 110.5: Pretreatment of HX10-infected H9 cells with sCD4 decreases signal from 110.5 at 37 degrees due to dissociation of gp120-gp41 [Sattentau (1995)]
- 110.5: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strains – neutralizes cell-free Hx10 [Sattentau & Moore(1995)]

Table of HIV MAbs

- 110.5: Reciprocal binding inhibition with other anti-V3 MAbs – enhances binding of some anti-V2 MAbs – binding enhanced by some CD4 binding site MAbs [Moore & Sodroski(1996)]
- 110.5: V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50–69, in contrast to anti-V2 MAbs [Poignard (1996a)]
- 110.5: Neutralizes HIV-1 LAI [McDougal (1996)]
- 110.5: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs (1996)]
- 110.5: Viral binding inhibition by 110.5 was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- 110.5: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

461	58.2	gp160(310–317)	gp120(MN)	HIGPGRAF	L	Vaccine	murine(IgG1 κ)
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Vaccine: Vector/type: peptide *Strain:* MN *HIV component:* V3

Ab type: V3 **Donor:** Repligen Corp.

References: [White-Scharf (1993), Potts (1993), Moore (1994b), Seligman (1996), Stanfield (1999), York (2001)]

- 58.2: Epitope defined by peptide reactivity and changes in affinity with amino acid substitutions – 4/7 primarily isolates were neutralized [White-Scharf (1993)]
- 58.2: Did not synergistically neutralize MN in combination with MAb F105 – there was synergistic neutralization when combined with sCD4 [Potts (1993)]
- 58.2: Modest cross-reactivity among B clade gp120s, little outside B clade – core epitope as I-IHIG [Moore (1994b)]
- 58.2: Competition ELISAs with serial deletions produced longer estimates of epitope length, RIHIGPGRAF_Y, than Alanine substitution, suggesting significance of non-contact residues [Seligman (1996)]
- 58.2: The crystal structure of Fab 58.2 bound to V3 loop peptides was obtained – conformational changes in the tip of the V3 loop (GPGR) were observed when different MAbs were bound – 58.2’s epitope was defined as KKKRIHIGPGRAF_Y [Stanfield (1999)]
- 58.2: 58.2’s epitope was noted to be IGPGRAF – Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding [York (2001)]

462	537-D (537)	gp160(311–315)	gp120(MN)	IGPGR	L	HIV-1 infection	human(IgG1 λ)
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Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

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

- 537-D: Reacts with MN, NY5, CDC4, RF, WM52 and SF2, but does not cross-react with HXB2 [Karwowska (1992b)]
- 537-D: MN type specific neutralization observed – binds SF2, also IGPGR [Gorny (1992), Gorny (1993)]
- 537-D: Moderate homologous neutralization, relatively rapid dissociation constant [VanCott (1994)]
- 537-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]

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

463	5020	gp160(311–316)	gp120(311–316 BH10)	RGPGRA	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> BH10 <i>HIV component:</i> V3</p> <p>Ab type: V3 References: [Langedijk (1991)]</p> <ul style="list-style-type: none"> • 5020: Generation and fine mapping of murine MAbs [Langedijk (1991)] 							
464	5023A (5023, NEA-9205, NEA 9205)	gp160(311–317)	gp120(311–317 BH10)	RgPGRAF	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> BH10 <i>HIV component:</i> V3</p> <p>Ab type: V3 Donor: Paul Durda, Du Pont de Nemours and Co</p> <p>References: [Langedijk (1991), D'Souza (1991), Back (1993), Rovinski (1995), Schonning (1998)]</p> <ul style="list-style-type: none"> • 5023A: Generation and fine mapping of murine MAbs [Langedijk (1991)] • 5023A: Called 5023 – Langedijk also has an MAb called 5023B – strong cross-reactive neutralizing MAb [D'Souza (1991)] • 5023A: Called 5023 – Langedijk also has an MAb called 5023B – gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662–675 is ELDKWANLWNWFNI [Back (1993)] • 5023A: Called 5023 in this paper – Used to precipitate gp160 in immunoblots in a study examining the feasibility of using unprocessed gp160 glycoprotein as an immunogen [Rovinski (1995)] • 5023A: Called NEA-9205 – The N306 glycan of the V3 loop makes the tip of the V3 loop inaccessible to this MAb in oligomeric Env, loss of this glycan enhances neutralization sensitivity [Schonning (1998)] 							
465	110.6	gp160(311–318)	gp120(BRU)	RGPGRAFV	L (weak)	Vaccine	murine(IgG1 λ)
<p>Vaccine: <i>Vector/type:</i> infected-cell lysate <i>Strain:</i> BRU <i>HIV component:</i> virus</p> <p>Ab type: V3 References: [Thomas (1988), Pirofski (1993), Langedijk (1992)]</p> <ul style="list-style-type: none"> • 110.6: Variable region sequenced – heavy chain: V J558–146b.1α, D closest to DSP16.2, J H3 – light chain: V λ1, J λ1 [Pirofski (1993)] 							
466	polyclonal	gp160(311–318)	gp120(MN)	IGPGRAFV	L	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> B. abortus complex <i>Strain:</i> SF2, MN <i>HIV component:</i> gp120</p> <p>Ab type: V3 References: [Golding (1995)]</p> <ul style="list-style-type: none"> • Ab is evoked even in mice depleted of CD4+ cells 							

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467 10/36e gp160(311–321) gp120(311–321) RGPGRAFVTIG L (HXB10) Vaccine rat(IgG2a)
 HXB10)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V3 **References:** [McKeating (1992a), McKeating (1993b), Peet (1998)]

- 10/36e: Binding to virion gp120 enhanced by sCD4 [McKeating (1992a)]
- 10/36e: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, but anti-V3 MAb 10/36e binding was dramatically diminished by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]

468 10/54 gp160(311–321) gp120(311–321) RGPGRAFVTIG L (HXB10) Vaccine rat(IgG1)
 (10/54ow/6i/6i) HXB10)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V3 **References:** [McKeating (1992a), McKeating (1993a), McKeating (1993b), Peet (1998)]

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

469 11/85b gp160(311–321) gp120(311–321) RGPGRAFVTIG L (HXB2) Vaccine rat(IgG2b)
 (11/85b/14I/14I) HXB10)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V3 **References:** [McKeating (1992a), McKeating (1993b)]

- 11/85b: Binding to virion gp120 enhanced by sCD4 [McKeating (1992a)]

470 polyclonal gp160(311–322) gp120(MN) IGPGRAFYTTKN L (MN ALA-1) Vaccine guinea pig()

Vaccine: *Vector/type:* human rhinovirus 14 *Strain:* MN *HIV component:* V3

Ab type: V3 **References:** [Smith (1998)]

- The tip of the MN V3 loop (IGPGRAFYTTKN) was inserted into cold-causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies – chimeric viruses elicited potent NABs against ALA-1 and MN [Smith (1998)]

471 0.5β (0.5 β, gp160(311–324) gp120(316–330) RGPGRAFVTIGKIG L (IIIB) Vaccine murine(IgG1κ)
 0.5β) HXB2)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* Env

Ab type: V3 **Donor:** Shuzo Matsushita or Toshio Hattori of Kumamoto University

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

- 0.5 β : Type-specific neutralization of IIIB – does not neutralize MN or RF [Matsushita (1988), Skinner (1988b)]
- 0.5 β : Emergence of virus resistant to MAb 0.5 β and autologous sera neutralization in IIIB infected chimps [Nara (1990)]
- 0.5 β : Potent neutralizing activity [D'Souza (1991)]
- 0.5 β : Chimeric mouse-human MAb C β 1 was constructed by combining the human Cgamma1 and Ckappa constant regions with the 0.5 β murine MAb – ADCC and neutralizing activity [Matsushita (1992)]
- 0.5 β : sCD4 causes loss of IIIB type-specificity, allowing binding and neutralization of MN, in contrast to MAb μ 5.5 [Maeda (1992)]
- 0.5 β : Monoclonal anti-idiotypic antibodies that mimic the 0.5 β epitope were generated [Sperlagh (1993)]
- 0.5 β : Neutralization of virus carrying an A to T substitution (contrast with MAb M77) [di Marzo Veronese (1993)]
- 0.5 β : Binding to native gp120 100–300 fold greater than to denatured [Moore (1993b)]
- 0.5 β : The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to some antiserum and conformationally sensitive neutralizing MAbs – neutralization efficiency of 0.5 β is not affected [Reitz (1988), Klasse (1993a)]
- 0.5 β : A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – of the MAbs tested, 0.5 β neutralization was the most profoundly affected by this mutation [Watkins (1993)]
- 0.5 β : MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook (1994)]
- 0.5 β : gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali (1994)]
- 0.5 β : Binding domain aa 310–319: RGPGRFVTIGKIG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: R306T, R309T and R313G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5 β [Okada (1994)]
- 0.5 β : Type-specific neutralization of IIIB – does not neutralize SF2 [Broder (1994)]
- 0.5 β : The interactions of the peptide RKSIRIQRGPGRAFVT 0.5 β were studied by NMR, and hydrophobic interactions between the two Is and the V form the base of a 12 amino acid loop with GPGR at the apex [Zvi (1995b)]
- 0.5 β : NMR of 0.5 β bound NNTRKSIRIQRGPGRAFVTIGKIG suggests that the bound amino acids are in the region SIRIQRGPGRAFVT [Zvi (1995a)]
- 0.5 β : The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits 0.5 β binding – 0.5 β epitope described as GPGRAFVTIG [Jagodzinski (1996)]
- 0.5 β : Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warriar (1996)]
- 0.5 β : Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs (1996)]
- 0.5 β : Relative to the native peptide, an O-linked α -galactosamine modified V3 peptide enhanced binding to 0.5 β , while an N-linked beta-glucosamine modified peptide showed reduced binding [Huang (1997)]

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

472	C β 1	gp160(311–324)	gp120(316–330 HXB2)	RGPGRAFTIGKIG	L	Vaccine	human(IgG1)
<p>Vaccine: <i>Vector/type:</i> protein <i>Strain:</i> IIIB <i>HIV component:</i> Env Ab type: V3 References: [Emini (1992)]</p> <ul style="list-style-type: none"> • Cβ1: passive transfer to chimpanzees confers protection against challenge with homologous cell-free virus – mouse 0.5β human IgG1 chimera [Emini (1992)] 							
473	NM-01	gp160(312–315)	gp120(MN)	GPGR	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> human rhinovirus 14 <i>Strain:</i> MN <i>HIV component:</i> V3 Ab type: V3 Donor: M. Terada References: [Ohno (1991), Yoshida (1997), Smith (1998)]</p> <ul style="list-style-type: none"> • NM-01: Resistance mutation selected by propagation of molecular cloned isolate in the presence of NM-01 [Yoshida (1997)] • NM-01: The tip of the MN V3 loop was inserted into cold-causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and NM-01 was among the Abs used – chimeric viruses elicited potent NAb in guinea pigs against ALA-1 and MN [Smith (1998)] 							
474	1026	gp160(312–317)	gp120(MN)	GPGRAF	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> MN <i>HIV component:</i> gp120 Ab type: V3 References: [Nakamura (1993), Bou-Habib (1994)]</p>							

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

475	1034	gp160(312–317)	gp120(MN)	GPGRAPH	L	Vaccine	murine(IgG)
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Vaccine: *Vector/type:* recombinant protein *Strain:* MN *HIV component:* gp120

Ab type: V3 **References:** [Bou-Habib (1994), Berman (1997)]

- 1034: Greater affinity for T cell tropic T-CSF, derived from JR-CSF, than to the primary isolate JR-CSF, close to GPGRAPH [Bou-Habib (1994)]
 - 1034: Binds to 5/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)]
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476	59.1 (R/V3– 59.1)	gp160(312–317)	gp120(308–313 MN)	GPGRAPH	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* peptide *Strain:* MN *HIV component:* V3

Ab type: V3 **Donor:** Mary White-Scharf and A. Profy, Repligen Corporation

References: [D'Souza (1991), White-Scharf (1993), Potts (1993), Ghiara (1993), Bou-Habib (1994), D'Souza (1994), Seligman (1996), Ghiara (1997), Smith (1998), Stanfield (1999), York (2001)]

- 59.1: Called R/V3–59.1 – potent neutralizing MAb [D'Souza (1991)]
 - 59.1: Epitope defined by peptide reactivity and binding affinity with amino acid substitutions – GPGRAPH [White-Scharf (1993)]
 - 59.1: Synergistic neutralization of MN when combined with sCD4 or the CD4BS MAb F105 [Potts (1993)]
 - 59.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 Fab fragment – contact residues IGGRAPH [Ghiara (1993)]
 - 59.1: Greater affinity for T-cell tropic strain T-CSF than the primary isolate JR-CSF, from which T-CSF was derived [Bou-Habib (1994)]
 - 59.1: Multi-lab study for antibody characterization and assay comparison – neutralizes MN and IIIB [D'Souza (1994)]
 - 59.1: Competition ELISAs with serial deletions produced longer estimate of epitope length than x-ray crystallography or Alanine substitution, RIHIGGRAPHYTT, suggesting significance of non-contact residues [Seligman (1996)]
 - 59.1: A conformationally restricted analog of the tip of the V3 loop was constructed and bound with Fab 59.1 – crystal structure shows interactions between 59.1 and an MN peptide and 59.1 and the modified peptide are similar, but NMR studies reveal that the modified peptide is more ordered in solution, retaining the Fab bound form [Ghiara (1997)]
 - 59.1: The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 59.1 was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith (1998)]
 - 59.1: The crystal structure of V3 loop peptides bound to Fabs was obtained – conformational changes in the tip of the V3 loop (GPGR) were observed when different MAbs were bound [Stanfield (1999)]
 - 59.1: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding [York (2001)]
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Table of HIV MAbs

477	polyclonal	gp160(312–317)	gp120(316–321)	GPGRAF		Vaccine	rabbit(Ig)
<p>Vaccine: <i>Vector/type:</i> polypeptide, protein <i>HIV component:</i> gp160 <i>Stimulatory Agents:</i> BSA</p> <p>Ab type: V3 References: [Lu (2000b), Lu (2000a)]</p> <ul style="list-style-type: none"> • High titer response to ELDKWA and RILAVERYLKD was observed upon vaccination with multiple-epitope vaccine CG-GPGRIFY-G-ELDKWA-G-RILAVERYLKD conjugated to BSA, a weak response to GPGRIFY – immunization with CG-(ELDKWA-GPGRIFY)_2-K was also tried, yielding a strong Ab response to ELDKWA, weak to GPGRIFY – gp160 vaccination yielded strong Ab response but not to any of the peptides studied here [Lu (2000b), Lu (2000a)] 							
478	10E3	gp160(312–318)	gp120(317–323 IIIB)	GPGRIFY		Vaccine	mouse(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide in keyhole limpet hemocyanin <i>Strain:</i> IIIB <i>HIV component:</i> V3</p> <p>Ab type: V3 References: [Tian (2001)]</p> <ul style="list-style-type: none"> • 10E3: Peptides GPGRIFY and ELDKWAG were conjugated to keyhole limpet hemocyanin and used to raise mouse monoclonal Ab – MAb hybridomas were generated with defined specificity – 10E3 binds to the peptide GPGRIFY and to rgp160 [Tian (2001)] 							
479	polyclonal	gp160(312–318)	gp120(317–323)	GPGRIFY		Vaccine	murine, rabbit()
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> V3 <i>Stimulatory Agents:</i> BSA</p> <p>Ab type: V3 References: [Yu (2000)]</p> <ul style="list-style-type: none"> • High levels of epitope-specific Abs were induced by the peptide-BSA conjugates C-(GPGRIFY)_4-BSA or C-(TRPNNNTRKSIRIQRGPGRIFYTIG KI)-BSA but not by rgp160 vaccine [Yu (2000)] 							
480	N11–20 (110-H)	gp160(312–320)	gp120(317–325)	GPGRIFYVTI	L (LAI)		murine(IgG1κ)
<p>Ab type: V3 Donor: J. C. Mazie, Hybridolab, Institut Pasteur</p> <p>References: [Valenzuela (1998)]</p> <ul style="list-style-type: none"> • N11–20: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11–20 is through inhibition of virus binding to the cell [Valenzuela (1998)] 							
481	5025A (5025)	gp160(313–317)	gp120(313–317 BH10)	pgRAF	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> BH10 <i>HIV component:</i> V3</p> <p>Ab type: V3 Donor: Paul Durda, Du Pont de Nemours and Co</p> <p>References: [Langedijk (1991), D’Souza (1991)]</p> <ul style="list-style-type: none"> • 5025A: Generation and fine mapping of murine MAbs [Langedijk (1991)] • 5025: Called 5025 – strain specific weakly neutralizing [D’Souza (1991)] 							
482	N70–1.9b	gp160(313–318)	gp120(316–322)	PGRIFY	L	HIV-1 infection	human(IgG1)
<p>Ab type: V3 References: [Robinson (1990a), Scott (1990)]</p> <ul style="list-style-type: none"> • N70–1.9b: Type specificity [Robinson (1990a)] • N70–1.9b: Type specific neutralization, ADCC directed against MN infected cells [Scott (1990)] 							

483	902	gp160(313–324)	gp120(IIIB)	PGRAFVTIGKIG	L	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> gp160</p> <p>Ab type: V3 Donor: Bruce Chesebro, Rocky Mountain National Laboratory, Montana</p> <p>References: [Chesebro & Wehrly(1988), Laman (1993), Broder (1994), Earl (1994)]</p> <ul style="list-style-type: none"> • 902: Strain specific neutralization of HIV [Chesebro & Wehrly(1988)] • 902: Epitope may be partially masked or altered in the oligomeric molecule [Broder (1994)] • 902: Used as a control in a study of the influence of oligomeric structure of Env in determining the repertoire of the Ab response [Earl (1994)] • 902: V3-BH10 peptide with loop-structure inhibits IL-2 induced T-cell proliferation, thought to be due to altering intracellular signaling, and MAb 908 can block the peptide inhibition [Sakaida1997] • 902: NIH AIDS Research and Reference Reagent Program: 522 							
484	694/98-D (694/98, 694.8, 694/98D)	gp160(dis 314– 317)	gp120(dis IIIB)	GRAF	L	HIV-1 infection	human(IgG1 λ)
<p>Ab type: V3 Donor: Drs. S. Zolla-Pazner and M. Gorny, NYU Med Center NY, NY</p> <p>References: [Gorny (1991), Gorny (1992), Gorny (1993), Cavacini (1993a), Spear (1993), Gorny (1994), Laal (1994), VanCott (1994), Cook (1994), VanCott (1995), Zolla-Pazner (1995), Forthal (1995), Li (1997), Zolla-Pazner (1997), Smith (1998), Li (1998), Andrus (1998), Nyambi (1998), Schonning (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Altmeyer (1999), Nyambi (2000), Park (2000)]</p> <ul style="list-style-type: none"> • 694/98-D: MAb first described [Skinner (1988b)] • 694/98-D: Type-specific lab isolate neutralization was observed – binds with 1–3 fold greater affinity to gp120 than to peptides [Gorny (1992)] • 694/98-D: Neutralizes MN and IIIB (GRAF) – binds SF2 (GRAF) – binding reactivity: MN, IIIB, SF2, NY5, RF, CDC4, WM52 [Gorny (1993)] • 694/98-D: Called 694-D – complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear (1993)] • 694/98-D: 50% neutralization of HIV-IIIB at a concentration of 0.15 μg/ml [Gorny (1994)] • 694/98-D: Potent neutralization of IIIB – no neutralization synergy in combination with CD4 binding domain MAbs [Laal (1994)] • 694/98-D: GRVY did not alter peptide binding – GRVI and GQAW enhanced dissociation – GQVF and GQAL did not bind [VanCott (1994)] • 694/98-D: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – V3 MAbs can inhibit gp120 binding to GalCer <i>in vitro</i> – binding of GalCer to gp120 inhibited but did not completely block MAb binding [Cook (1994)] • 694/98-D: Human HIV-1 infected sera and MAb 694/98 have high reactivity to MN and RF infected H9 cells, but Genentech rec gp120 IIIB vaccine recipients do not [VanCott (1995)] • 694/98-D: Serotyping study using flow-cytometry – bound GRAX bearing virus in 10/11 cases – somewhat conformation dependent [Zolla-Pazner (1995)] 							

Table of HIV MAbs

- 694/98-D: ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 694/98-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – could only achieve 50% neutralization alone – all Ab combinations tested showed synergistic neutralization – 694/98-D has synergistic response with MAbs F105, 15e, b12, 2F5, 17b, 2G12, and 48d, and with HIVIG [Li (1997)]
- 694/98-D: Used to study pre- and post-exposure prophylaxis Hu-PBL-SCID mice infected by an intraperitoneal injection of HIV-1 LAI – MAb half-life in plasma in mice is 9 days – 2 hours post-694/98-D mice were challenged with LAI, and at an Ab concentration of 1.32 mg/Kg, 50% of the mice were infected – one of the infected mice carried the resistant form GRTF rather than GRAF (critical amino acids for binding are GRA) – post-exposure prophylaxis was effective if delivered 15 min post-exposure, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus (1998)]
- 694/98-D: The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 694/98-D was among the Abs used – chimeric viruses elicited potent NAb in guinea pigs against ALA-1 and MN [Smith (1998)]
- 694/98-D: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) [Li (1998)]
- 694/98-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 694/98-D bound only to B and D clade virions and had limited cross reactivity [Nyambi (1998)]
- 694/98-D: In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, anti-V3 MAbs were found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan more efficiently than HIV-BRU [Schonning (1998)]
- 694/98-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 694/98-D: MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 694/98-D: A Semliki Forest virus (SFV) expression system carrying BX08 env was used to study the conformation of gp120 – intracytoplasmic gp120 was recognized by the anti-V3 MAbs K24 and F5.5, while gp120 at the plasma membrane was detected only by conformation dependent MAbs 2G12, 670-D and 694/98D and not linear V3 MAbs – expression in rat brain also showed that surface-expressed Env was recognized only by the conformation-dependent antibodies and not by anti-V3 antibodies [Altmeyer (1999)]
- 694/98-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 694/98-D showed intermediate reactivity [Nyambi (2000)]
- 694/98-D: Called 694/98D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]

485	9205 (NEA-9205)	gp160(315–317)	gp120(IIIB)	RAF (core reactivity)	L	Vaccine	murine(IgG1)
<i>Vaccine: Vector/type: peptide Strain: IIIB HIV component: V3</i>							
Ab type: V3 Donor: NEN, Boston MA, commercial							

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

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

486	110.I	gp160(316–322)	gp120(316–322)	AFVTIGK	L	Vaccine	murine()
Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> gp120							
Ab type: V3 Donor: F. Traincard, Pasteur Institute, France							
References: [Moore (1993b), Moore (1994c), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Wyatt (1997), Parren (1998a)]							
<ul style="list-style-type: none"> • 110.I: Binds to carboxy-terminal side of the V3 loop – inhibits binding of C4 region MAb G3-299 [Moore (1993b)] • 110.I: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains [Sattentau & Moore(1995)] • 110.I: Reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and enhances binding of some anti-V2 MAbs – binding enhanced by some anti-CD4 binding site MAbs [Moore & Sodroski(1996)] • 110.I: Epitope suggested to be RAFVTIGK – V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50–69, in contrast to anti-V2 MAbs [Poignard (1996a)] • 110.I: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)] • 110.I: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)] 							
487	anti-HIV-2 polyclonal	gp160(dis 315–318 + 329–331)	gp120(dis 315–318 SBL6669 HIV-2)	FHSQ...WCR		Vaccine	guinea pig(IgG)
Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> HIV-2 SBL6669-ISY <i>HIV component:</i> V3							
Ab type: HIV-2 V3 References: [Morner (1999)]							
<ul style="list-style-type: none"> • Neutralizing Abs against HIV-2 V3 are produced when peptides spanning two non-contiguous parts of the V3 loop are used for vaccination including amino acids 315–318 near the tip (FHSQ) and 329–331 (WCR) at the C-term Cys [Morner (1999)] 							
488	IIIB-V3-01	gp160(320–328)	gp120(IIIB)	IGKIGNMRQ	no	Vaccine	murine(IgG1)
Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> IIIB <i>HIV component:</i> V3							
Ab type: V3 Donor: Jon Laman							
References: [Laman (1993)]							

Table of HIV MAbs

- IIIB-V3-01: Specific for carboxy-terminal flank of the IIIB V3 loop – epitope is hidden native gp120, exposed on denaturation [Laman (1993)]
- IIIB-V3-01: UK Medical Research Council AIDS reagent: ARP3046
- IIIB-V3-01: NIH AIDS Research and Reference Reagent Program: 1726

489	D/6D1	gp160(346–377)	gp120(351–382 LAI)	ASKLREQFGNNKTIIFKQSSG-GDPEIVTHSFN	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp120</p> <p>Ab type: V4 References: [Bristow (1994)]</p> <ul style="list-style-type: none"> • D/6D1: V4 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)] 							
490	4D7/4	gp160(360–380)	gp120(361–380 LAI)	IFKQSSGGDPEIVTHSFNCGG		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: V4 Donor: S. Ranjbar, NIBSC, UK</p> <p>References: [Moore (1994c)]</p> <ul style="list-style-type: none"> • 4D7/4: C3 region – the relative affinity for denatured/native gp120 is >10 [Moore (1994c)] • 4D7/4: UK Medical Research Council AIDS reagent: ARP3051 							
491	36.1(ARP 329)	gp160(361–381)	gp120(362–381 LAI)	FKQSSGGDPEIVTHSFNCGGE		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: V4 References: [Thiriart (1989), Moore (1994c)]</p> <ul style="list-style-type: none"> • 36.1: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P impair binding [Moore (1994c)] • 36.1: UK Medical Research Council AIDS reagent: ARP329 							
492	C12	gp160(361–381)	gp120(362–381 LAI)	FKQSSGGDPEIVTHSFNCGGE		Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p> <p>Ab type: V4 Donor: George Lewis</p> <p>References: [Moore & Ho(1993), Moore (1994c), Abacioglu (1994), Moore (1994d)]</p> <ul style="list-style-type: none"> • C12: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • C12: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P, and 384 Y/E impair binding – also binds GEFFYCNSTQLFNS, gp120(380–393 LAI) [Moore (1994c)] • C12: C3 region – epitope boundaries mapped by peptide scanning, core FNCGG [Abacioglu (1994)] 							
493	110.D	gp160(380–393)	gp120(380–393 LAI)	GEFFYCNSTQLFNS	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: C3 Donor: F. Traincard, Pasteur Institute, France</p>							

Table of HIV MAbs

499	5C2E5	gp160(422–431)	gp120(406–415 IIIB)	QFINMWQEVK		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> gp120</p> <p>Ab type: C4 Donor: T. Gregory and R. Ward, Genentech, San Francisco</p> <p>References: [Lasky (1987), Cordell (1991)]</p> <ul style="list-style-type: none"> • 5C2E5: Blocks the gp120-CD4 interaction [Lasky (1987)] • 5C2E5: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell (1991)] 							
500	G3-211	gp160(423–437)	gp120(423–437 IIIB)	IINMWQKVGKAMYAP	L	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> virus derived protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: C4 References: [Sun (1989)]</p> <ul style="list-style-type: none"> • G3-211, 42, 299, 508, 519, 536, 537: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun (1989)] 							
501	G3-537	gp160(423–437)	gp120(423–437 IIIB)	IINMWQKVGKAMYAP	L	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> virus derived protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>Ab type: C4 References: [Sun (1989), Ho (1991b), McKeating (1992b)]</p> <ul style="list-style-type: none"> • G3-537, 211, 299, 508, 519, 536, 42: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun (1989)] • G3-537: Weakly neutralizing – binds to a linear binding domain of gp120, NMWQEVGKAMYAPPISG [McKeating (1992b)] 							
502	polyclonal	gp160(425–436)	gp120()	NMWQEVGKAMYA	L	Vaccine	murine(IgA)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> IIIB <i>Stimulatory Agents:</i> cholera toxin adjuvant</p> <p>Ab type: CD4BS References: [Bukawa (1995)]</p> <ul style="list-style-type: none"> • Polyclonal secretory IgA antibody raised by mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to the V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa (1995)] 							
503	1795	gp160(425–441)	gp120(425–441 IIIB)	NMWQEVGKAMYAPPISG	L	Vaccine	()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>HIV component:</i> Env</p> <p>Ab type: CD4BS References: [McKeating (1992b)]</p> <ul style="list-style-type: none"> • 1795: CD4 binding site – weakly neutralizing – binding inhibited by WQEVGKAMYA, GKAM may be involved [McKeating (1992b)] 							
504	G3-299	gp160(429–438)	gp120(429–438 BRU)	EVGKAMYAPP	L	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> virus derived protein <i>HIV component:</i> gp120</p> <p>Ab type: C4 Donor: M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY</p> <p>References: [Sun (1989), Moore (1993b), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Ditzel (1997), Wyatt (1997), Parren (1998a)]</p>							

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

505	G3-42 (G3 42)	gp160(429–438)	gp120(429–438 BRU)	EVGKAMYAPP	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY

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

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

Table of HIV MAbs

- G3-42: 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 [Binley (1999)]
- 0.5β: MAbs 0.5β and G3-42 were used to study synthesis of oligomeric and monomeric forms of Env – inhibition of glycosylation by tunicamycin results in reduction of oligomeric gp120 at the cell surface and of monomer in the cytoplasm – neither MAb recognized non-glycosylated Env precursor [Jagodzinski & Trzeciak(2000)]

506	G3-508 (G3 508)	gp160(429–438)	gp120(429–438 BRU)	EVGKAMYAPP	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **Donor:** M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY

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

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

507	G3-519	gp160(429–438)	gp120(429–438 BRU)	EVGKAMYAPP	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun (1989), Moore & Ho(1993), Moore (1993b), D’Souza (1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Wyatt (1997), Parren (1998a), Binley (1999)]

- G3-519: Best neutralization of RF in panel of 7 MAbs that bind overlapping epitope [Sun (1989)]
- G3-519: Neutralizes IIIB, is reactive with SF-2 gp120, mild inhibition of HIV-1+ sera binding to IIIB gp120 [Moore & Ho(1993)]
- G3-519: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 5 fold greater affinity than native – 433A/L, 435Y/H, 438P/R and 430V/S substitutions impaired binding [Moore (1993b)]
- G3-519: Included in a multi-lab study for antibody characterization, and binding and neutralization assay comparison, also binds IIIB: IINMWQKVGKAMYAPP [D’Souza (1994)]
- G3-519: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-519: Non-reciprocal enhanced binding in the presence of the C5 MAb 1C1 and the C1 MAb 135/9 – reciprocal enhanced binding with some V2 MAbs. Inhibited binding the presence of some C4, V3 and CD4 binding site MAbs [Moore & Sodroski(1996)]
- G3-519: Epitope described as KVGKAMYAPP – binding resulted in slight gp120 dissociation from virus but no significant exposure of the gp41 epitope for MAb 50–69 [Poignard (1996a)]
- G3-519: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- G3-519: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- G3-519: The MAbs with the broadest neutralizing activity, 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 NAb 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 [Binley (1999)]

508	G3-536	gp160(429–438)	gp120(429–438 BRU)	EVGKAMYAPP	L	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY

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

- G3-536: Weak neutralization of IIIB and RF – cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – epitope:IINMWQKVGKAMYAP [Sun (1989)]
- G3-536: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell (1991)]
- G3-536: Weakly neutralizing – binds to a linear determinant in the CD4 binding domain of gp120 [McKeating (1992b)]
- G3-536: Neutralizes IIIB, is reactive with SF-2 gp120, mild inhibition of HIV-1+ sera binding to IIIB gp120 [Moore & Ho(1993)]
- G3-536: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 15 fold greater affinity than native – 433A/L, 435Y/H, 438P/R, and 430V/S substitutions impaired binding [Moore (1993b)]

Table of HIV MAbs

- G3-536: Enhances binding of anti-V2 MAb 697-D [Gorny (1994)]
- G3-536: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-536: Inhibits binding of some V3, C4 and CD4 binding site MAbs, enhances binding of V2 region MAbs [Moore & Sodroski(1996)]
- G3-536: Epitope described as KVGKAMYAPP [Poignard (1996a)]
- G3-536: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

509	ICR38.1a (38.1a, 388/389)	gp160(429–438)	gp120(427–436 BRU)	EVGKAMYAPP	L	Vaccine	rat(IgG2b)
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Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: C4 **References:** [Cordell (1991), McKeating (1992b), McKeating (1992a), McKeating (1992c), McKeating (1993b), McKeating (1993a), Moore (1993b), Jeffs (1996), Peet (1998), Kropelin (1998)]

- ICR38.1a: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with MAbs G3-536, 5C2E5, and ICR38.8f [McKeating (1992b), Cordell (1991)]
- ICR38.1a: Unable to exert a synergistic effect in combination with V3 directed MAbs, in contrast to MAb 39.13g, that binds to a conformational epitope involved in CD4 binding [McKeating (1992a)]
- ICR38.1a: Studied in the context of a neutralization escape mutant [McKeating (1993a)]
- ICR38.1a: Unreactive with solid-phase decapeptide, competed in solution phase assay – ICR 38.1a and ICR 38.8f were initially reported to be independent MAbs, but are actually subclones of the same MAb [Moore (1993b)]
- ICR38.1a: Called 38.1a – 10 to 20 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs (1996)]
- ICR38.1a: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – ICR38.1a was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- ICR38.1a: Called 388/389 – anti-C1 region MAb 87–135/9 blocks gp120 interaction with CD4+ cells – blocking activity is additive when combined with antibodies which bind in the C4 region of gp120 (F105, 388/389, and b12) [Kropelin (1998)]
- ICR38.1a: UK Medical Research Council AIDS reagent: ARP388/ARP389

510	ICR38.8f	gp160(429–438)	gp120(429–438 BRU)	EVGKAMYAPP	L	Vaccine	rat(IgG2b)
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Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: C4 **References:** [Cordell (1991)]

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

Table of HIV MAbs

511	MO86/C3	gp160(429–443) Ab type: C4	gp120(429–443) References: [Ohlin (1992)]	EVGKAMYAPPISGQI		<i>in vitro</i> stimulation	human(IgM)
<ul style="list-style-type: none"> • MO86: Generated in response to IIIB Env 286–467 upon <i>in vitro</i> stimulation of uninfected-donor lymphocytes [Ohlin (1992)] 							
512	13H8	gp160(431–440) Vaccine: <i>Vector/type:</i> recombinant protein Ab type: C4	gp120(412–453) <i>Strain:</i> MN References: [Nakamura (1992), Nakamura (1993), Jeffs (1996)]	GKAMYAPPIS	L	Vaccine	murine(IgG)
<ul style="list-style-type: none"> • 13H8: Cross blocks 5C2 in IIIB-rsgp160 ELISA – reactive with diverse strains in rgp120 ELISA [Nakamura (1992)] • 13H8: Bound diverse strains, neutralizing activity against MN [Nakamura (1993)] • 13H8: Binds V3 and C4 peptides (J. P. Moore, per. comm.) • 13H8: 3 and 4.5 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120, respectively [Jeffs (1996)] 							
513	G45–60	gp160(431–440) Vaccine: <i>Vector/type:</i> virus derived protein Ab type: C4	gp120(429–438 BRU) <i>Strain:</i> IIIB <i>HIV component:</i> gp120 References: [Sun (1989), Moore (1993b), Gorny (1994), Moore & Sodroski(1996), Jagodzinski (1996)]	GKAMYAPPIS	L	Vaccine	murine(IgG1)
<ul style="list-style-type: none"> • G45–60: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPI, decapeptide flanking peptides also bound – bound equivalently to native and denatured gp120 – 433A/L and 435Y/H (not 430V/S) substitutions impaired binding [Moore (1993b)] • G45–60: Enhances binding of anti-V2 MAb 697-D [Gorny (1994)] • G45–60: Non-reciprocal enhancement of G45–60 binding by some C1 and C5 antibodies – reciprocal enhancement of some V2 region MAbs – reciprocal inhibition with many MAbs that bind to the V3, C4 and CD4 binding site regions [Moore & Sodroski(1996)] • G45–60: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus CRDS inhibits G45–60 binding [Jagodzinski (1996)] 							
514	polyclonal	gp160(432–451) Vaccine: <i>Vector/type:</i> vaccinia Ab type: C4	gp120(42–61 LAI) <i>HIV component:</i> Env References: [Collado (2000)]	KAMYAPPISGQIRCSSNITG	no	Vaccine	murine()
<ul style="list-style-type: none"> • Vaccinia p14 can elicit NAb and p39 tends to be immunodominant, so these two proteins were fused to regions of HIV-1 Env – reduced glycosylation was noted when p14 or p39 was placed in the N-term region of the fusion protein – chimeric proteins shifted the Env Ab response from V3 to either a C1 or C4 domain, depending on the construct – all chimeric Env proteins: 14kEnv, 39kEnv, and Env39k elicited a strong Ab response to the C1 region of gp120 (LFCASDAKAYDTEVHNVWAT), and Env39k mounted a strong response to the C4 region (KAMYAPPISGQIRCSSNITG) [Collado (2000)] 							
515	1662	gp160(433–439) Vaccine: <i>Vector/type:</i> poliovirus Ab type: C4	gp120(IIIB) <i>HIV component:</i> Env References: [McKeating (1992b)]	AMYAPPI	no	Vaccine	()
<ul style="list-style-type: none"> • 1662: Did not bind to native gp120, epitope not exposed [McKeating (1992b)] 							

Table of HIV MAbs

516	1663	gp160(433–439)	gp120(IIIB)	AMYAPPI	no Vaccine	()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>HIV component:</i> Env Ab type: C4 References: [McKeating (1992b)] • 1663: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]</p>						
517	1664	gp160(433–439)	gp120(IIIB)	AMYAPPI	no Vaccine	()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>HIV component:</i> Env Ab type: C4 References: [McKeating (1992b)] • 1664: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]</p>						
518	1697	gp160(433–439)	gp120(IIIB)	AMYAPPI	no Vaccine	()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>HIV component:</i> Env Ab type: C4 References: [McKeating (1992b)] • 1697: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]</p>						
519	1794	gp160(433–442)	gp120(IIIB)	AMYAPPISGQ	no Vaccine	()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>HIV component:</i> Env Ab type: C4 References: [McKeating (1992b)] • 1794: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]</p>						
520	1804	gp160(433–442)	gp120(IIIB)	AMYAPPISGQ	no Vaccine	()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>HIV component:</i> Env Ab type: C4 References: [McKeating (1992b)] • 1804: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]</p>						
521	1807	gp160(433–442)	gp120(IIIB)	AMYAPPISGQ	no Vaccine	()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>HIV component:</i> Env Ab type: C4 References: [McKeating (1992b)] • 1807: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]</p>						
522	1808	gp160(433–442)	gp120(IIIB)	AMYAPPISGQ	no Vaccine	()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>HIV component:</i> Env Ab type: C4 References: [McKeating (1992b)] • 1808: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]</p>						

523	polyclonal (VEI5)	gp160(454–474)	Env()	LTRDGGNNNESEIFRPGGGD	HIV-1 infection	human()
<p>Ab type: V1, V2, V3, V4, V5 References: [Carlos (1999)]</p> <ul style="list-style-type: none"> • Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTGGDIGNIRQ [Carlos (1999)] 						
524	polyclonal	gp160(460–467)	gp120(LAI)	NNNNGSEI	HIV-1 infection, Vaccine	human()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160</p> <p>Ab type: V5 References: [Loomis-Price (1997)]</p> <ul style="list-style-type: none"> • HIV-1+ positive individuals were given a gp160 vaccine as immunotherapy, and this region was the most reactive new epitope as measured by a modified Pepsan technique which improved sensitivity – 4/14 showed vaccine-induced reactivity [Loomis-Price (1997)] 						
525	CRA1(ARP 323) (CRA- 1)	gp160(461–470)	gp120(451–470 LAI)	SNNESEIFRL	no Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> Env</p> <p>Ab type: V5C5 Donor: M. Page, NIBSC, UK</p> <p>References: [Moore & Ho(1993), Moore (1994d), Moore (1994c), Moore & Sodroski(1996), Trkola (1996a)]</p> <ul style="list-style-type: none"> • CRA1: Bound preferentially to denatured IIIB and SF2 gp120 [Moore & Ho(1993)] • CRA1: Some C5 mutations abrogate binding 470 P/L or G, 475 M/S, some C2 mutations enhance binding [Moore (1994d)] • CRA1: The relative affinity for denatured/native gp120 is 24 – C5 mutations 470 P/L or G, 475 M/S impairs binding to the native gp120 – only mutation 470 P/L impairs binding to denatured [Moore (1994c)] • CRA1: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – reciprocal binding inhibition with anti-C5 antibodies 1C1 and M91 – non-reciprocal binding enhancement some C1 and V2 antibodies – non-reciprocal binding inhibition of some CD4 binding site antibodies [Moore & Sodroski(1996)] • CRA1: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)] • CRA1: UK Medical Research Council AIDS reagent: ARP323 						
526	M91	gp160(461–470)	gp120(451–470 LAI)	SNNESEIFRL	no Vaccine	rat(IgG2a)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> Env</p> <p>Ab type: V5C5 Donor: Fulvia di Marzo Veronese</p> <p>References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Ditzel (1997), Binley (1998)]</p> <ul style="list-style-type: none"> • M91: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)] 						

Table of HIV MAbs

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

527	9201	gp160(471–482) Ab type: C5 References: [McDougal (1996)]	gp120(475–486 LAI) Donor: Du Pont References: [McDougal (1996)]	GGGDMRDNRWSE	no	murine()
● 9201: Does not neutralize LAI [McDougal (1996)]						
528	1C1	gp160(471–490) Ab type: C5 References: [Moore (1994c), Moore (1994d), VanCott (1995), Moore & Sodroski(1996)]	gp120(471–490 LAI) Donor: Repligen Inc, Cambridge, MA, commercial References: [Moore (1994c), Moore (1994d), VanCott (1995), Moore & Sodroski(1996)]	GGGDMRDNRSELYKYKVVK	Vaccine	murine(IgG)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> Env						
● 1C1: The relative affinity for denatured/native gp120 is 15 [Moore (1994c)]						
● 1C1: C2 and V3 regions substitutions can influence binding [Moore (1994d)]						
● 1C1: Linear epitope not exposed on conformationally intact gp120 [VanCott (1995)]						
● 1C1: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – M91 binding was enhanced by 1C1, but 1C1 binding was inhibited by M91 – non-reciprocal binding enhancement of some C1 and V2 antibodies – non-reciprocal binding inhibition of some CD4 binding site antibodies [Moore & Sodroski(1996)]						
529	3F5	gp160(471–490) Ab type: C5 References: [Moore (1994c)]	gp120(471–490 LAI) Donor: S. Nigida, NCI, USA References: [Moore (1994c)]	GGGDMRDNRSELYKYKVVK	Vaccine	murine(IgG)
Vaccine: <i>Strain:</i> LAI <i>HIV component:</i> Env						
● 3F5: The relative affinity for denatured/native gp120 is 100 [Moore (1994c)]						
530	5F4/1	gp160(471–490) Ab type: C5 References: [Moore (1994c)]	gp120(471–490 LAI) Donor: S. Ranjbar, NIBSC, UK References: [Moore (1994c)]	GGGDMRDNRSELYKYKVVK	Vaccine	murine()
Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> HIV-2 ROD						
● 5F4/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>10 fold) – mutation 485 K/V impairs binding [Moore (1994c)]						

Table of HIV MAbs

531	660-178	gp160(471-490)	gp120(471-490 LAI)	GGGDMRDNRSELYKYKVVK	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>Strain:</i> LAI	<i>HIV component:</i> Env	
		Ab type: C5		Donor: G. Robey, Abbott Labs		
		References: [Moore (1994c), Moore (1994d)]				
		<ul style="list-style-type: none"> • 660-178: The relative affinity for denatured/native gp120 is >100 [Moore (1994c)] • 660-178: $\Delta V1/V2$ and $\Delta V1/V2/V3$ reduce binding – C2 and C5 mutations enhance binding [Moore (1994d)] 				
532	9301	gp160(471-490)	gp120(471-490 LAI)	GGGDMRDNRSELYKYKVVK	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>Strain:</i> LAI	<i>HIV component:</i> Env	
		Ab type: C5		Donor: Dupont, commercial		
		References: [Skinner (1988b), Moore & Ho(1993), Moore (1994c), Moore (1994d), Wagner (1996)]				
		<ul style="list-style-type: none"> • 9301: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • 9301: The relative affinity for denatured/native gp120 is 19 [Moore (1994d)] • 9301: Wagner <i>et al.</i> claim that Nea 9301 is anti-V3 – might they have meant MAb 9305? [Wagner (1996)] 				
533	B221 (221)	gp160(471-490)	gp120(471-490 LAI)	GGGDMRDNRSELYKYKVVK	Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>Strain:</i> NL43	<i>HIV component:</i> gp160	
		Ab type: C5		Donor: Rod Daniels		
		References: [Moore & Ho(1993), Bristow (1994), Moore (1994c)]				
		<ul style="list-style-type: none"> • B221: Called 221 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • B221: MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIIB:NL43, Micro-GenSys [Bristow (1994)] • B221: The relative affinity for denatured/native gp120 is 12 – mutation 477 D/V impairs binding [Moore (1994c)] • B221: Called 221 – C2 and V3 substitutions influence binding [Moore (1994d)] • B221: UK Medical Research Council AIDS reagent: ARP301 				
534	8C6/1	gp160(471-490)	gp120(471-490 LAI)	GGGDMRDNRSELYKYKVVK	Vaccine	murine(IgG)
	Vaccine:	<i>Strain:</i> LAI				
		Ab type: V5C5		Donor: S. Ranjbar, NIBSC, UK		
		References: [Moore (1994c)]				
		<ul style="list-style-type: none"> • 8C6/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>30 fold) – mutation 485 K/V impairs binding [Moore (1994c)] • 8C6/1: UK Medical Research Council AIDS reagent: ARP3052 				
535	H11	gp160(472-477)	gp120(472-477 HXB2)	GGDMRD		murine()
		Ab type: C5		References: [Pincus & McClure(1993), Pincus (1996)]		

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

536	W2	gp160(472–491)	gp120(472–491 LAI)	GGDMRDNRSELYKYKVVVKI	Vaccine	murine(IgG)
<p>Vaccine: Strain: LAI HIV component: Env</p> <p>Ab type: C5 Donor: D. Weiner, U. Penn., USA</p> <p>References: [Moore (1994c)]</p> <ul style="list-style-type: none"> • W2: The relative affinity for denatured/native gp120 is 30 – mutation 485 K/V impairs binding [Moore (1994c)] 						
537	1331A	gp160(dis gp160(483–508))	gp120(dis 510–516)	dwVVQREKR	HIV-1 infection	human(IgG3λ)
<p>Ab type: C5 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Nyambi (1998), Gorny (2000), Hochleitner (2000b), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 1331A: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-C5 Abs 670-D and 1331A bound to 3/4 B clade viruses (they don't bind to IIIB), and to subtype D MAL [Nyambi (1998)] • 1331A: Core epitope dwVVQREKR maps to gp120(510–516) – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – C5 MAbs 858-D, 989-D and 1331A bound with a 5–10 fold preference for the monomer[Gorny (2000)] • 1331A: The Ab binding site was studied with epitope excision (protein is bound in native conformation to immobilized MAb, then digested with proteolytic enzymes) and extraction (protein is digested then allowed to react with Ab), followed by mass spectroscopy – two non-contiguous aa in C5 were protected, E-507 and I-487, which are thought to be located on opposite sides of hydrophobic pocket involved in gp120/gp41 interaction [Hochleitner (2000b)] • 1331A: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 858-D bound only 4/26, the worst of all anti-C5 MAbs tested, while MAb 1331A, which shares the same core epitope (positions 495–516), bound to 18/26 [Nyambi (2000)] 						
538	M38	gp160(485–504)	gp120(490–508)	KYKVVKEIPLGVAPTKAKRR	no Vaccine	murine()
<p>Vaccine: Vector/type: virus Strain: IIIB HIV component: virus</p> <p>Ab type: C5 References: [Beretta (1987), Grassi (1991), Lopalco (1993), DeSantis (1994), Beretta & Dalgleish(1994)]</p> <ul style="list-style-type: none"> • M38: Binds to gp120 and to a 80 kd human protein expressed on a small fraction of mononuclear cells in the lymph nodes [Beretta (1987)] • M38: Binds to the carboxy terminus of gp120, in a gp41 binding region, and also to denatured human HLAs (antigenic homology) [Lopalco (1993)] • M38: Infected individuals have HLA class I-gp120 cross-reactive antibodies [DeSantis (1994)] 						

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539	Chim 1 (C-1)	gp160(487–493)	gp120(492–498 HXB2)	KVVKEIP		humanized chimpanzee()
<p>References: [Pincus & McClure(1993), Pincus (1996)]</p> <ul style="list-style-type: none"> • Chim 1: Binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus (1996)] 						
540	polyclonal	gp160(489–511)	gp120(495–516 BRU)	KIEPLGVAPTKAKRRVQREKR	no HIV-1 infection	human()
<p>References: [Hernandez (2000)]</p> <ul style="list-style-type: none"> • Chimeric peptide combining two peptides gp160(495–516 and 584–612) served as a specific and broadly reactive antigen for diagnostic detection of HIV-1 [Hernandez (2000)] 						
541	110.1	gp160(491–500)	gp120(491–500 LAI)	IEPLGVAPTK	no Vaccine	murine(IgG1κ)
<p>Vaccine: <i>Vector/type:</i> infected-cell lysate <i>Strain:</i> BRU <i>HIV component:</i> virus</p> <p>Ab type: C5 Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas</p> <p>References: [Gosting (1987), Linsley (1988), Thomas (1988), Pincus (1991), Moore (1994c), Cook (1994), McDougal (1996), Binley (1997a), Valenzuela (1998)]</p> <ul style="list-style-type: none"> • 110.1: There is another antibody with this ID that binds to gp120, but at aa 200–217 [Pincus (1996)] • 110.1: Referred to as 110–1 – does not inhibit CD4-gp120 binding or neutralize HIV-1 strains [Linsley (1988)] • 110.1: Difference in the epitope: mapped to aa 421–429 (KQIINMWQE), the T1 sequence – poor efficacy as an immunotoxin when linked to RAC [Pincus (1991)] • 110.1: The relative affinity for denatured/native gp120 is 0.7 [Moore (1994c)] • 110.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the carboxy-terminus of gp120 inhibit gp120 binding to GalCer but not as potently as anti-V3 MAbs – binding of GalCer to gp120 does not inhibit MAb binding [Cook (1994)] • 110.1: Does not neutralize HIV-1 LAI [McDougal (1996)] • 110.1: Does not effect LAI viral binding or entry into CEM cells [Valenzuela (1998)] 						
542	42F	gp160(491–500)	gp120(491–500 HXB2)	IEPLGVAPTK	no HIV-1 infection	human(IgG1λ)
<p>Ab type: C5 References: [Alsmadi (1997), Alsmadi & Tilley(1998)]</p> <ul style="list-style-type: none"> • 42F: 42F and 43F were isolated from a long term non-progressor by EBV transformation of PBMC – samples were taken 14 months apart – both MAbs stained diverse strains of infected cells and directed ADCC – were more potent for ADCC if the cell was infected with HIV-1, rather than just presenting absorbed gp120 [Alsmadi (1997)] • 42F: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against strains IIIB, MN, SF-2, and RF, but not a clone of MN [Alsmadi & Tilley(1998)] 						
543	43F	gp160(491–500)	gp120(491–500 HXB2)	IEPLGVAPTK	no HIV-1 infection	human(IgG1λ)

Table of HIV MAbs

		Ab type: C5	References: [Alsmadi (1997)]			
		<ul style="list-style-type: none"> • 43F: 42F and 43F were isolated from a long term non-progressor by EBV transformation of PBMC – samples were taken 14 months apart – both MAbs stained diverse strains of infected cells and directed ADCC – were more potent for ADCC if the cell was infected with HIV-1, rather than just presenting absorbed gp120 [Alsmadi (1997)] 				
544	RV110026	gp160(491–500)	gp120(491–500 LAI)	IEPLGVAPTK	Vaccine	human()
	Vaccine:	<i>Vector/type:</i> peptide		<i>Strain:</i> LAI		
		Ab type: C5	Donor: Commercial, Olympus Inc			
		References: [Moore (1994c), Moore (1994d)]				
		<ul style="list-style-type: none"> • RV110026: Preferentially binds SDS-DTT denatured gp120 (15 fold using R1/87 as capture reagent) [Moore (1994c)] 				
545	105–306	gp160(492–500)	gp120(498–505 HAM112, O group)	KPFSVAPTP	Vaccine	murine(IgG1 κ)
	Vaccine:	<i>Vector/type:</i> recombinant protein		<i>Strain:</i> HAM112 (group O)	<i>HIV component:</i> gp160	
		Ab type: C-term	References: [Scheffel (1999)]			
		<ul style="list-style-type: none"> • 105–306: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity – 105–306 bound to two overlapping peptides [Scheffel (1999)] 				
546	GV1G2	gp160(494–499)	gp120(494–499 IIIB)	LGVAPT	Vaccine	murine()
	Vaccine:	<i>Vector/type:</i> protein-Ab complex		<i>HIV component:</i> gp120 complexed with MAb M77		
		Ab type: C5	References: [Denisova (1996)]			
		<ul style="list-style-type: none"> • GV1G2: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV12F6 and GV3H1 are homologous to GV1G2 and were generated in the same experiment [Denisova (1996)] 				
547	750-D	gp160(498–504)	gp120(503–509)	PTKAKRR	no HIV-1 infection	human(IgG3 λ)
		Ab type: C-term	References: [Forthal (1995), Hioe (2000)]			
		<ul style="list-style-type: none"> • 750-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity [Forthal (1995)] • 750-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – C5 MAbs 450-D and 750-D did not effect proliferation [Hioe (2000)] 				
548	450-D (450-D-3, 450D)	gp160(498–504)	gp120(475–486 BH10)	PTKAKRR (or RRVVQRE, or MRDNWRSELYKY depending on reference)	no HIV-1 infection	human(IgG1 λ)
		Ab type: C5	Donor: Susan Zolla-Pazner (Zollas01@mrcr6.med.nyu), NYU Med Center, NY, NY			
		References: [Durda (1988), Karwowska (1992a), Karwowska (1992b), Spear (1993), Laal (1994), Gorny (1994), Cook (1994), Forthal (1995), Manca (1995), Li (1997), Hioe (2000), Hioe (2001), Verrier (2001)]				
		<ul style="list-style-type: none"> • 450-D: Bound to MN, SF-2 and IIIB, but was not neutralizing [Karwowska (1992a)] • 450-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear (1993)] 				

- 450-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal (1994)]
- 450-D: Epitope is defined as PTKAKRR [Gorny (1994)]
- 450-D: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the carboxy-terminus of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook (1994)]
- 450-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 450-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 450-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 6 $\mu\text{g/ml}$ [Li (1997)]
- 450-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – C5 MAbs 450-D and 750-D did not effect proliferation [Hioe (2000)]
- 450-D: CD4BS MAbs when complexed with gp120, inhibit proliferation of gp120-specific CD4 T-cells and IFN- γ production – 450-D does not have this effect and was used as a control in this study [Hioe (2001)]
- 450-D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 $\mu\text{g/ml}$: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]

549	670-D (670)	gp160(498–504)	gp120(503–509)	PTKAKRR	no HIV-1 infection	human(IgG1 λ)
		Ab type: C5		Donor: Susan Zolla-Pazner (Zollas01@mcr6.med.nyu), NYU, NY		
		References: [Zolla-Pazner (1995), Forthal (1995), Hill (1997), Gorny (1997), Gorny (1998), Nyambi (1998), Altmeyer (1999), Gorny & Zolla-Pazner(2000), Nyambi (2000), Verrier (2001)]				
		● 670-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner (1995)]				
		● 670-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity, numbering provided suggests epitope is RRVVQRE [Forthal (1995)]				
		● 670-D: gp120 can inhibit MIP-1 α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill (1997)]				
		● 670-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-C5 Abs 670-D and 1331A bound to 3/4 B clade viruses (they didn't bind to IIIB), and to subtype D MAL – 670-D also reacted with subtype A[Nyambi (1998)]				
		● 670-D: A Semliki Forest virus (SFV) expression system carrying BX08 env was used to study the conformation of gp120 – intracytoplasmic gp120 was recognized by the anti-V3 MAbs K24 and F5.5, while gp120 at the plasma membrane was detected only by conformation dependent MAbs 2G12, 670-D and 694/98D and not V3 MAbs – expression in rat brain also showed that surface-expressed Env was recognized only by the conformation-dependent antibodies and not by anti-V3 antibodies [Altmeyer (1999)]				
		● 670-D: A gp120 C5 MAb used as a negative control in a study of anti-gp41 MAbs [Gorny & Zolla-Pazner(2000)]				
		● 670-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 670-D bound 21/26, and was the most cross-reactive C5 MAb [Nyambi (2000)]				

Table of HIV MAbs

- 670-D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 $\mu\text{g/ml}$: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]

550	polyclonal	gp160(503–509)	gp120(471–477)	RRVVQRE	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp120</p> <p>References: [Jeyarajah (1998)]</p> <ul style="list-style-type: none"> Mice were immunized with peptide APTKAKRRRVVQREKR – epitope excision and extraction combined with mass spectrometry was used to map the fine structure of epitopes recognized by polyclonal Ab to HIV-1 Env – a major epitope was identified between positions 472 and 478 [Jeyarajah (1998)]; 						
551	722-D	gp160(503–509)	gp120(503–509)	RRVVQRE	no HIV-1 infection	human(IgG1 κ)
<p>Ab type: C-term References: [Laal (1994), Forthal (1995)]</p> <ul style="list-style-type: none"> 722-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal (1994)] 722-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)] 						
552	polyclonal	gp160(503–511)	gp120(508–516)	RRVVQREKR	HIV-1 infection	human()
<p>Ab type: C-term References: [Palker (1987), Loomis-Price (1997)]</p> <ul style="list-style-type: none"> Most HIV-1+ individuals have an antibody response to this epitope – in this study, reactivity to RRVVQREKR was used as a positive control for HIV-1+ gp160 vaccine recipients [Loomis-Price (1997)] 						
553	1131-A	gp160(505–511)	gp120(510–516 LAI)	VVQREKR	no HIV-1 infection	human(IgG3 λ)
<p>Ab type: C-term References: [Bandres (1998)]</p> <ul style="list-style-type: none"> 1131-A: A very high affinity antibody used in studies that demonstrate that CXCR4 can bind to gp120 in the absence of CD4-gp120 interactions, and that this binding can be enhanced by Env deglycosylation [Bandres (1998)] 						
554	858-D	gp160(505–511)	gp120(510–516 LAI)	VVQREKR	no HIV-1 infection	human(IgG)
<p>Ab type: C-term Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)</p> <p>References: [Zolla-Pazner (1995), Forthal (1995), Gorny (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> 858-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner (1995)] 858-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)] 858-D: The binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – C5 MAbs 858-D, 989-D and 1331A bound with a 5–10 fold preference for the monomer[Gorny (2000)] 858-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 858-D bound only 4/26, the worst of all anti-C5 MAbs tested, while MAb 1331A, which shares the same core epitope (positions 495–516), bound to 18/26 [Nyambi (2000)] 						

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555 989-D	gp160(505–511) Ab type: C-term	gp120(LAI) Donor: Susan Zolla-Pazner (Zollas01@mccrcr6.med.nyu) (NYU Med. Center)	VVQREKR	HIV-1 infection	human(IgG)
	<p>References: [Zolla-Pazner (1995), Gorny (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> ● 989-D: In serotyping study using flow-cytometry, showed B clade specificity, but only reacted with 7/11 B clade virus [Zolla-Pazner (1995)] ● 989-D: The binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – C5 MAbs 858-D, 989-D and 1331A bound with a 5–10 fold preference for the monomer[Gorny (2000)] ● 989-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 989-D bound to 6/26 [Nyambi (2000)] 				
556 1A1	gp160(525–543)	gp41(526–543 BH10)	AAGSTMGAASMTLTVQARQ	no HIV-1 infection	human(IgG1 κ)
	<p>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria</p> <p>References: [Buchacher (1994)]</p> <ul style="list-style-type: none"> ● 1A1: Human MAb generated using EBV transformation of PBL from HIV-1+ volunteers [Buchacher (1994)] 				
557 24G3	gp160(525–543)	gp41(526–543 BH10)	AAGSTMGAASMTLTVQARQ	no HIV-1 infection	human(IgG1 κ)
	<p>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria</p> <p>References: [Buchacher (1992), Buchacher (1994)]</p> <ul style="list-style-type: none"> ● 24G3: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells [Buchacher (1994)] 				
558 25C2 (IAM 41–25C2)	gp160(525–543)	gp41(526–543 BH10)	AAGSTMGAASMTLTVQARQ	no HIV-1 infection	human(IgG1 κ)
	<p>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX</p> <p>References: [Buchacher (1992), Buchacher (1994), Sattentau (1995)]</p> <ul style="list-style-type: none"> ● 25C2: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells – binds oligomeric and monomeric gp41, and gp160 [Buchacher (1994)] ● 25C2: Called IAM 41–25C2 – Binding domain overlaps sites that are critical for gp120-gp41 association – binding is enhanced by sCD4 – binding region defined as: gp41(21–38 BH10) [Sattentau (1995)] 				
559 5F3	gp160(525–543)	gp41(526–543 BH10)	AAGSTMGAASMTLTVQARQ	no HIV-1 infection	human(IgG1 κ)
	<p>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria</p> <p>References: [Buchacher (1994)]</p> <ul style="list-style-type: none"> ● 5F3: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells [Buchacher (1994)] 				
560 α (566–586)	gp160(561–581)	gp41(566–586 BRU)	AQQHLLQLTVWGIKQLQARIL	HIV-1 infection	human()
	<p>References: [Poumbourios (1992)]</p>				

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561	PC5009 <i>Vaccine:</i>	gp160(572–591) <i>Vector/type:</i> recombinant protein	gp41(577–596 BRU) <i>HIV component:</i> gp160	GIKQLQARILAVERYLKDQQ	Vaccine	murine()
<p>References: [Poumbourios (1992)]</p> <ul style="list-style-type: none"> • PC5009: Recognized only monomeric gp41 [Poumbourios (1992)] 						
562	polyclonal α (577–596)	gp160(572–591)	gp41(577–596 BRU)	GIKQLQARILAVERYLKDQQ	HIV-1 infection	human plasma()
<p>References: [Poumbourios (1992)]</p> <ul style="list-style-type: none"> • α(577–596): Affinity purified from HIV-1+ plasma – preferentially bind oligomer [Poumbourios (1992)] 						
563	polyclonal	gp160(576–592)	gp41(583–599)	LQARILAVERYLKDQQL	HIV-1 infection	human sera()
<p>References: [Klasse (1993b)]</p> <ul style="list-style-type: none"> • 42 HIV-1 positive human sera were tested against wildtype peptide, and peptide with substitution 589 A to T: 11/42 reacted strongly with wildtype, weakly with A589T – 31 reacted weakly with parental, even more weakly with substituted [Klasse (1993b)] 						
564	1F11	gp160(578–612)	gp41(579–613 BH10)	ARILAVERYLKDQQLLGIWGC- SGKLICTTAVPWNA	no HIV-1 infection	human(IgG1 κ)
<p>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria</p> <p>References: [Buchacher (1992), Buchacher (1994)]</p> <ul style="list-style-type: none"> • 1F11: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)] 						
565	1H5	gp160(578–612)	gp41(579–613 BH10)	ARILAVERYLKDQQLLGIWGC- SGKLICTTAVPWNA	no HIV-1 infection	human(IgG1 κ)
<p>References: [Buchacher (1992), Buchacher (1994)]</p> <ul style="list-style-type: none"> • 1H5: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)] 						
566	3D9	gp160(578–612)	gp41(579–613 BH10)	ARILAVERYLKDQQLLGIWGC- SGKLICTTAVPWNA	no HIV-1 infection	human(IgG1 κ)
<p>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria</p> <p>References: [Buchacher (1992), Buchacher (1994)]</p> <ul style="list-style-type: none"> • 3D9: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)] 						
567	4B3	gp160(578–612)	gp41(579–613 BH10)	ARILAVERYLKDQQLLGIWGC- SGKLICTTAVPWNA	no HIV-1 infection	human(IgG1 λ)
<p>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria</p> <p>References: [Buchacher (1992), Buchacher (1994), Chen (1994b)]</p> <ul style="list-style-type: none"> • 4B3: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)] 						
568	4D4	gp160(578–612)	gp41(579–613 BH10)	ARILAVERYLKDQQLLGIWGC- SGKLICTTAVPWNA	no HIV-1 infection	human(IgG1 λ)

<p>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX References: [Buchacher (1992), Buchacher (1994), Chen (1994b), Sattentau (1995), Binley (1999)]</p> <ul style="list-style-type: none"> • 4D4: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)] • 4D4: 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 [Binley (1999)] 						
569	4G2	gp160(578–612)	gp41(579–613 BH10)	ARILAVERYLKDQQLLGIWGC- SGKLICTTAVPWNA	no HIV-1 infection	human(IgG1 κ)
<p>Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria References: [Buchacher (1992), Buchacher (1994)]</p> <ul style="list-style-type: none"> • 4G2: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)] 						
570	polyclonal	gp160(579–589)	gp41(586–596 IIIB)	RILAVERYLKD	Vaccine	mouse, rabbit()
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41 <i>Stimulatory Agents:</i> BSA Ab type: C-domain References: [Xiao (2000b)]</p> <ul style="list-style-type: none"> • Strong epitope-specific neutralizing antibody responses were induced using the peptide C(RILAVERYLKD)_2-BSA, but not full gp160 [Xiao (2000b)] 						
571	polyclonal	gp160(579–589)	gp41(586–596)	RILAVERYLKD	Vaccine	rabbit(Ig)
<p>Vaccine: <i>Vector/type:</i> polyepitope, protein <i>HIV component:</i> gp160 <i>Stimulatory Agents:</i> BSA Ab type: N-term References: [Lu (2000b), Lu (2000a)]</p> <ul style="list-style-type: none"> • High titer response to ELDKWA and RILAVERYLKD was observed upon vaccination with multiple-epitope vaccine CG-GPGRAPHY-G-ELDKWA-G-RILAVERYLKD conjugated to BSA, a weak response to GPGRAPHY – immunization with CG-(ELDKWA-GPGRAPHY)_2-K was also tried, yielding a strong Ab response to both ELDKWA and GPGRAPHY – gp160 vaccination yielded strong Ab response but not to any of the peptides studied here [Lu (2000b), Lu (2000a)] 						
572	polyclonal	gp160(579–599)	gp41(583–604)	RILAVERYLKDQQLLGIWGCS	no Vaccine	rabbit sera()
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> desialylated gp160 References: [Benjouad (1993)]</p> <ul style="list-style-type: none"> • MAbs raised against desialylated HIV-1 gp160 cross-react with HIV-2 gp140 due to immunodominant conserved epitope in gp41 [Benjouad (1993)] 						

Table of HIV MAbs

573	2A2/26	gp160(579–601)	gp41(584–606 BRU)	RILAVERYLKDQQLLGIWGCS-GK	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> gp41</p> <p>References: [Poumbourios (1992), Poumbourios (1995)]</p> <ul style="list-style-type: none"> • 2A2/26: Immunodominant region, binds both oligomer and monomer [Poumbourios (1992)] • 2A2/26: Δ 550–561 (Δ LLRAIEAQQHLL), a region important for oligomer formation diminishes binding, Δ (550–561 +571–581) abrogates binding [Poumbourios (1995)] 						
574	50–69 (SZ-50.69)	gp160(dis 579–613)	gp41(dis 579–613 BH10)	RILAVERYLKDQQLLGIWGCS-GKLI	no HIV-1 infection	human(IgG2κ)
<p>Ab type: cluster I Donor: Susan Zolla-Pazner (Zollas01@mrcrcr6.med.nyu), NYU, NY</p> <p>References: [Till (1989), Pinter (1989), Gorny (1989), Xu (1991), Robinson (1991), Sattentau & Moore(1991), Eddleston (1993), Spear (1993), Laal (1994), Chen (1995), Sattentau (1995), Manca (1995), McDougal (1996), Poignard (1996a), Binley (1996), Klasse & Sattentau(1996), Stamatatos (1997), Boots (1997), Mitchell (1998), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000), Zwick (2001b), Verrier (2001)]</p> <ul style="list-style-type: none"> • 50–69: Combined with deglycosylated A chain of ricin is toxic to lines of HIV-infected T cells (H9) and monocytes (U937) [Till (1989)] • 50–69: Reacts preferentially with gp160 oligomer, compared to gp41 monomer [Pinter (1989)] • 50–69: Kills HIV-infected cells when coupled to deglycosylated ricin A chain [Gorny (1989)] • 50–69: The epitope is affected by the conformation conferred by the two cysteines at amino acids 598 and 604 [Xu (1991)] • 50–69: Enhances HIV-1 infection <i>in vitro</i> – synergizes with huMAb 120–16 <i>in vitro</i> to enhance HIV-1 infection to level approaching that found in polyclonal anti-HIV serum [Robinson (1991)] • 50–69: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)] • 50–69: Called SZ-50.69 – binds to an epitope within aa 579–613 [Eddleston (1993)] • 50–69: Did not mediate deposition of complement component C3 on HIV infected cells unless cells were pre-incubated with sCD4 – complement mediated virolysis of MN and IIIB in the presence of sCD4 [Spear (1993)] • 50–69: Epitope described as cluster I, 601–604, conformational – does not neutralize IIIB or synergize neutralization by anti-V3 MAb 447-52D or by CD4 BS MAbs [Laal (1994)] • 50–69: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen (1995)] • 50–69: Preferentially binds oligomer – binding increased after pretreatment of infected cells with sCD4 – binding domain overlaps site that is critical for gp120-gp41 association [Sattentau (1995)] • 50–69: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)] • 50–69: Does not neutralize HIV-1 LAI [McDougal (1996)] • 50–69: Prebinding of anti-V3, and CD4i MAbs 48d and 17b, but not anti-V2 neutralizing MAbs, expose the 50–69 epitope [Poignard (1996a)] • 50–69: Binds to a linear epitope located in the cluster I region – binding of 50–69 and 240-D inhibited by Fabs A1, A4, M8B, M26B, M12B and T2 [Binley (1996)] 						

- 50–69: Used to test exposure of gp41 upon sCD4 binding [Klasse & Sattentau(1996)]
- 50–69: Binding of anti-gp120 MAbs IgG1b12 or 654–30D does not mediate significant exposure of the gp41 epitopes for MAbs 2F5 and 50–69 [Stamatatos (1997)]
- 50–69: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – 50–69 maps to an immunodominant domain in gp41 – three groups of peptides were selected, one which seems most closely related to gp41 sequence peptide consensus is WGCxx(RK)(x n)LxC – the analogous gp41 sequence WGCSGKLIC is present in most M group clades, except D with a common L to H substitution [Boots (1997)]
- 50–69: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50–69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 – identifies non-contiguous W596-G597-C598 and C604-T605 as minimal epitope [Mitchell (1998)]
- 50–69: This antibody binds to a cluster I epitope in gp41, 567–647, and recognizes a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41 – this MAb doesn't react with either of the peptides N51 or C43 individually – MAbs 50–69 and 1367 had similar properties – MAb 50–69 bound the fusogenic form of the protein in liquid phase [Gorny & Zolla-Pazner(2000)]
- 50–69: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)]
- 50–69: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 50–69 bound the majority of isolates although binding was moderate to weak – specifies discontinuous binding site range as aa 579–613 [Nyambi (2000)]
- 50–69: This paper primarily concerns 4E10 and Z13, MAbs that both bind proximally to the 2F5 binding site to a conserved epitope, and that neutralize some primary isolates from clades B, C, and E – MAb 50–69 binding to infected cells is enhanced by sCD4, while 4E10 and Z13 binding is essentially unaltered [Zwick (2001b)]
- 50–69: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 µg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 50–69: NIH AIDS Research and Reference Reagent Program: 531

575 9–11	gp160(579–604)	gp41(584–609)	RILAVERYLKDQQLLGIWGCS-GKLIC	Vaccine	murine(IgG1)
Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> gp160					
References: [Mani (1994)]					
● 9–11: required the C-C disulfide bridge and loop formation, can bind simultaneously with 41–1 [Mani (1994)]					
576 98–43	gp160(579–604)	gp41(579–604 HXB2)	RILAVERYLKDQQLLGIWGCS-GKLIC	no HIV-1 infection	human(IgG2κ)
References: [Pinter (1989), Gorny (1989), Tyler (1990), Xu (1991)]					

Table of HIV MAbs

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

577	41-1 (41.1)	gp160(579-608)	gp41(584-609)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	Vaccine	murine(IgG1κ)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> gp160</p> <p>References: [Gosting (1987), Dalglish (1988), Pincus (1991), Pincus & McClure(1993), Mani (1994), Pincus (1996), Pincus (1998)]</p> <ul style="list-style-type: none"> ● 41-1: This antibody to gp41(584-609) [Mani (1994)] seems to have been named the same as a different MAb to gp41(735-752 IIIB) [Dalglish (1988)] ● 41-1: Also called 41.1, although possibly not, the literature is confusing because two gp41 MAbs that bind to this region with similar names (dash versus period) are listed as murine and human ● 41-1: Broadly reactive [Gosting (1987)] ● 41-1: This antibody seems to have been named the same as a different MAb to gp41(735-752) [Dalglish (1988)] ● 41-1: Efficacious as an immunotoxin when coupled to RAC – gave linear epitope as gp160 579-603 [Pincus (1991)] ● 41-1: Called 41.1, and described as a human MAb – cross-competes with 41.4 – sCD4 enhances the efficacy of immunotoxins <i>in vitro</i> 30-fold – MAb was coupled to ricin A chain (RAC) [Pincus & McClure(1993)] ● 41-1: Did not require the C-C disulfide bridge and loop formation, can bind simultaneously with 9-11 [Mani (1994)] ● 41-1: Called 41.1, and described as a human MAb, binding 579-604 – a panel of immunotoxins was generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)] 						
578	41.4	gp160(579-608)	gp41(584-609)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV		()
<p>Donor: Jan McClure, Bristol-Myers Squibb Pharmaceutical Res Inst, Seattle, WA</p> <p>References: [Pincus & McClure(1993)]</p> <ul style="list-style-type: none"> ● 41.4: Binds to peptide weakly, but to gp160 with higher affinity than 41.1, and cross-competes with 41.1 – probably conformational – MAb was coupled to ricin A chain (RAC) – sCD4 enhances the efficacy of immunotoxins <i>in vitro</i> 30-fold [Pincus & McClure(1993)] 						
579	Fab A1	gp160(579-608)	gp41(584-609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1κ)
<p>References: [Binley (1996)]</p> <ul style="list-style-type: none"> ● Fab A1: Binds to cluster I region – competes with MAbs 240-D and 50-69 – conformation sensitive – variable regions sequenced [Binley (1996)] 						
580	Fab A4	gp160(579-608)	gp41(584-609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1κ)
<p>References: [Binley (1996)]</p>						

					<ul style="list-style-type: none"> • Fab A4: Binds to cluster I region – competes with MABs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)] 	
581	Fab M12B	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1 κ)
					<p>References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab M12B: Binds to cluster I region – competes with MABs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)] 	
582	Fab M26B	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1 κ)
					<p>References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab M26B: Binds to cluster I region – competes with MABs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)] 	
583	Fab M8B	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1 κ)
					<p>References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab M8B: Binds to cluster I region – competes with MABs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)] 	
584	Fab T2	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1 κ)
					<p>References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab T2: Binds to cluster I region – competes with MABs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)] 	
585	86 (No. 86)	gp160(579–613)	gp41(586–620 IIIB)	RILAVERYLKDQQLLGIWGCS-GKLICTTAVPWNAS	no HIV-1 infection	human(IgG1 κ)
					<p>Donor: Evan Hersh and Yoh-Ichi Matsumoto</p> <p>References: [Sugano (1988), Robinson (1990b), Robinson (1990c), Pincus (1991), Moran (1993), Wisnewski (1996), Mitchell (1998)]</p> <ul style="list-style-type: none"> • 86: Reacts with gp41 and also reacted weakly with gp120 [Sugano (1988)] • 86: Antibody dependent enhancement (ADE) of HIV-1 IIIB infectivity in the presence of complement [Robinson (1990b)] • 86: Peptide 586–620 blocks complement mediated ADE [Robinson (1990c)] • 86: Poor immunotoxin activity when coupled to RAC – peptide binding stated to be aa 579–603 [Pincus (1991)] • 86: Heavy (V H1) and light (V κI) chain sequenced – enhancing activity – similar germline sequence to MAb S1–1, but very different activity [Moran (1993)] • 86: 86 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)] 	

Table of HIV MAbs

- 86: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50–69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 [Mitchell (1998)]
- 86: NIH AIDS Research and Reference Reagent Program: 380

586	polyclonal	gp160(580–597) References: [Petrov (1990)]	gp41(584–602)	ILAVERYLKDQQLGIWG	no HIV-1 infection	human sera()
<ul style="list-style-type: none"> • Immunodominant and broadly reactive peptide [Petrov (1990)] 						
587	V10–9	gp160(580–613) References: [Robinson (1990b), Robinson (1990c)]	gp41(586–620 IIIB)	ILAVERYLKDQQLGIWGCSG- KLICTTAVPWNAS	no HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • V10–9: Antibody dependent enhancement (ADE) of HIV-1 IIIB infectivity, synergistically enhanced by MAb 120–16 [Robinson (1990b)] • V10–9: Peptide 586–620 blocks complement mediated ADE [Robinson (1990c)] 						
588	polyclonal	gp160(582–589) References: [Klasse (1991)]	gp41(589–596)	AVERYLKD	HIV-1 infection	human sera()
<ul style="list-style-type: none"> • Substitutions and deletions in peptide 583–599 were systematically studied – alterations in AVERYLKD abrogated the antigenicity of peptides with most of 14 human sera [Klasse (1991)] 						
589	polyclonal	gp160(584–604) References: [Shafferman (1989)]	gp41(74–94)	ERYLKDQLLGIWGCSGKLIC	HIV-1 infection	human()
<ul style="list-style-type: none"> • Immunogenic domain useful for diagnostics [Shafferman (1989)] 						
590	polyclonal	gp160(584–612) References: [Hernandez (2000)]	gp41(587–617 BRU)	ERYLKDQQLLGIWGCSGKLIC- TTAVPWNA	no HIV-1 infection	human()
<ul style="list-style-type: none"> • Chimeric peptide combining two peptides gp160(495–516 and 584–612) served as a specific and broadly reactive antigen for diagnostic detection of HIV-1 [Hernandez (2000)] 						
591	2F11	gp160(589–600) References: [Eaton (1994)]	gp41(589–600 HXB2)	DQQLGIWGCSG	no HIV-1 infection	human(IgG1)
<ul style="list-style-type: none"> • 2F11: Enhances infectivity even in the absence of complement – does not mediate ADCC or neutralize virus [Eaton (1994)] 						
592	246-D (SZ-246.D)	gp160(590–597) Ab type: cluster I References: [Xu (1991), Robinson (1991), Spear (1993), Eddleston (1993), Forthal (1995), Manca (1995), Saarloos (1995), Earl (1997), Gorny & Zolla-Pazner(2000), Nyambi (2000), Verrier (2001)]	gp41(579–604 HXB2) Donor: Susan Zolla-Pazner (Zollas01@mrcr6.med.nyu), NYU Med Center, NY, NY	qqLLGIWg	no HIV-1 infection	human(IgG1 κ)

- 246-D: Fine mapping indicates core is LLGI [Xu (1991)]
- 246-D: Did not mediate deposition of complement component C3 on HIV infected cells unless cells were pre-incubated with sCD4 [Spear (1993)]
- 246-D: No neutralizing activity, some enhancing activity [Robinson (1991)]
- 246-D: Called SZ-246.D [Eddleston (1993)]
- 246-D: No neutralizing activity, both ADCC and viral enhancing activity [Forthal (1995)]
- 246-D: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 246-D: Ab-mediated activation of complement on HIV+ cells is higher than Ab independent activation – what has been termed “Ab independent” in fact results in part from IgM in normal human serum that is HIV-cross-reactive [Saarloos (1995)]
- 246-D: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50–69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 [Mitchell (1998)]
- 246-D: This antibody, along with murine MAb D61, can be blocked by any of a group of 8 conformational MAbs (M10, D41, D54, T4, T6, T9, T10 and T35) [Earl (1997)]
- 246-D: Core epitope aa 591 to 597, a cluster I epitope that does not bind to either a peptide complex that approximates the core of the fusogenic form of gp41 or the individual peptides N51 and C43 that form this structure – MAbs 181-D and 246-D had similar properties [Gorny & Zolla-Pazner(2000)]
- 246-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 246-D bound strongly or moderately to all 26 HIV-1 group M clades viruses tested and showed the strongest binding of all anti-Env MAbs tested, including the V3 and C5 region MAbs – notes core epitope as LLGI – no neutralizing activity was observed when 246-D was tested with five isolates [Nyambi (2000)]
- 246-D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 $\mu\text{g/ml}$: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 246-D: NIH AIDS Research and Reference Reagent Program: 1245

593	9G5A	gp160(591–594)	gp41(596–599 IIIB)	QLLG	anti-idiotypic	murine(IgM)
<p>References: [Lopalco (1993), Beretta & Dalgleish(1994)]</p> <p>● 9G5A: Anti-idiotypic to gp120 C terminus (C5 region) MAb M38 [Lopalco (1993)]</p>						
594	181-D (SZ-181.D)	gp160(591–597)	gp41(591–597 HXB2)	qLLGIWg	no HIV-1 infection	human(IgG2 κ)
<p>Ab type: cluster I Donor: Susan Zolla-Pazner (Zollas01@mrcr6.med.nyu), NYU, NY</p> <p>References: [Xu (1991), Robinson (1991), Eddleston (1993), Forthal (1995), Fontenot (1995), Gorny & Zolla-Pazner(2000), Nyambi (2000)]</p> <p>● 181-D: Fine mapping indicates core is LLGIW [Xu (1991)]</p> <p>● 181-D: No enhancing or neutralization activity [Robinson (1991)]</p> <p>● 181-D: Called SZ-181.D [Eddleston (1993)]</p>						

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

595	240-D (F240:)	gp160(592–600)	gp41(592–600 HXB2)	LLGIWGCSG	no HIV-1 infection	human()
<p>Ab type: cluster I Donor: Susan Zolla-Pazner (Zollas01@mrcr6.med.nyu), NYU, NY</p> <p>References: [Xu (1991), Robinson (1991), Spear (1993), Binley (1996), Wisnewski (1995), Wisnewski (1996), Mitchell (1998), Nyambi (2000)]</p> <ul style="list-style-type: none"> ● 240-D: Fine mapping indicates core is IWG [Xu (1991)] ● 240-D: No neutralizing activity, some enhancing activity [Robinson (1991)] ● 240-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear (1993)] ● 240-D: Binds to a linear epitope located in the cluster I region – binding of 50–69 and 240-D inhibited by Fabs A1, A4, M8B, M26B, M12B and T2 [Binley (1996)] ● 240-D: Called F240: F240 in V H3 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)] ● 240-D: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50–69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 [Mitchell (1998)] ● 240-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 246-D bound strongly or moderately to 24/26 HIV-1 group M clades viruses tested [Nyambi (2000)] ● 240-D: NIH AIDS Research and Reference Reagent Program: 1242 						
596	F240	gp160(592–606)	gp41(592–606 BH10)	LLGIWGCSGKLICTT	no HIV-1 infection	human(IgG1κ)
<p>Ab type: cluster I Donor: L. Cavacina or M. Posner, Dept. of Med. Harvard Med. School, Boston MA, USA</p> <p>References: [Cavacini (1998a), York (2001)]</p> <ul style="list-style-type: none"> ● F240: Seems to be distinct from MAb 240-D, an antibody with a similar epitope in the immunodominant region of gp41 – dose-dependent reactivity with HIV isolates RF, SF2, IIIB, and MN was observed – F240 had no neutralizing activity and enhances infection in the presence of complement – reactivity of F240 is enhanced by preincubation of cells with sCD4 or anti-CD4BS MAb F105 – heavy and light chain variable domains were sequenced, and a strong homology to hu MAb 3D6 was observed, as 3D6 binds to the same epitope, these MAbs may define a human Ab clonotype [Cavacini (1998a)] 						

- F240: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559-64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NABs alters some step after binding [York (2001)]

597 D49	gp160(592-608)	gp41(597-613)	LLGIWGCSGKLICTTAV	Vaccine	murine()
Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> dimeric Env				
	Ab type: cluster I References: [Earl (1994), Earl (1997)]				
	<ul style="list-style-type: none"> • D49: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues [Earl (1997)] 				
598 D61	gp160(592-608)	gp41(592-608 HXB2)	LLGIWGCSGKLICTTAV	Vaccine	murine()
Vaccine:	<i>Vector/type:</i> protein <i>HIV component:</i> dimeric Env				
	Ab type: cluster I References: [Earl (1994), Richardson (1996), Weissenhorn (1996), Earl (1997)]				
	<ul style="list-style-type: none"> • D61: Linear gp41 epitope in the cluster I region – human sera blocked binding in oligomeric ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 [Richardson (1996)] • D61: Does not precipitate gp41(21-166), but due to a structural difference in the disulfide bonding region near the two cysteines – the authors propose that this region may change conformation during the activation of the membrane fusion state of the HIV-1 glycoprotein [Weissenhorn (1996)] • D61: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues – this antibody, along with human MAb 246-D, can be blocked by any of a group of 8 conformational MAbs (M10, D41, D54, T4, T6, T9, T10 and T35) – members of this competition group are blocked by sera from HIV-1+ individuals [Earl (1997)] 				
599 T32	gp160(592-608)	gp41(597-613)	LLGIWGCSGKLICTTAV	Vaccine	murine()
Vaccine:	<i>Vector/type:</i> tetrameric Env <i>HIV component:</i> Env				
	Ab type: cluster I References: [Earl (1994), Earl (1997)]				
	<ul style="list-style-type: none"> • T32: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues [Earl (1997)] 				
600 T34	gp160(592-608)	gp41(597-613)	LLGIWGCSGKLICTTAV	Vaccine	murine()
Vaccine:	<i>Vector/type:</i> tetrameric Env <i>HIV component:</i> Env				
	Ab type: cluster I References: [Earl (1994), Earl (1997)]				
	<ul style="list-style-type: none"> • T34: Binding maps to region 597-613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues [Earl (1997)] 				

Table of HIV MAbs

601	115.8	gp160(593–604)	gp41(598–609)	LGLIWGCSGKLIC	Vaccine	murine(IgM)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Oldstone (1991)]</p> <ul style="list-style-type: none"> • 115.8: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – poor reactivity with CSGKLIC – reacts well with longer HIV-2 peptide NSWGCAFRQVC as well as CAFRQVC – disulfide bond between cysteines required [Oldstone (1991)] 						
602	M-1	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1,IgG2b)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-1: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)] 						
603	M-11	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-11: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)] 						
604	M-13	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-13: Reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)] 						
605	M-2	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-2: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)] 						
606	M-22	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-22: Strongest reaction of 12 anti-HIV-1 gp41 MAbs to a cellular 43-kd protein found in rat and human astrocytes [Yamada (1991)] 						
607	M-24	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-24: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)] 						

Table of HIV MAbs

608	M-25	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-25: Reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)] 						
609	M-28	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-28: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)] 						
610	M-29	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-29: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)] 						
611	M-36	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-36: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)] 						
612	M-4	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-4: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)] 						
613	M-6	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Yamada (1991)]</p> <ul style="list-style-type: none"> • M-6: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)] 						
614	polyclonal α (598–609)	gp160(594–601)	gp41(598–609)	GIWGCSGK	HIV-1 infection	human()
<p>References: [Poumbourios (1992)]</p> <ul style="list-style-type: none"> • α(598–609): Affinity purified from HIV-1+ plasma – immunodominant region, binds oligomer and monomer [Poumbourios (1992)] 						

Table of HIV MAbs

615	1B8.env	gp160(594–604)	gp41(594–605 HXB2)	GIWGCSGKLIC	no	HIV-1 infection	human(IgG2λ)
<p>References: [Banapour (1987)]</p> <ul style="list-style-type: none"> • 1B8.env: Highly conserved epitope recognized by the majority of HIV-1 infected people [Banapour (1987)] 							
616	polyclonal	gp160(594–609)	gp41(601–616)	GIWGCSGKLICTTAVP	no	HIV-1 infection	human sera()
<p>References: [Petrov (1990)]</p> <ul style="list-style-type: none"> • Immunodominant and broadly reactive peptide [Petrov (1990)] 							
617	clone 3	gp160(597–606)	gp41(597–606)	GCSGKLICTT	L	HIV-1 infection	human(IgG1)
<p>References: [Cotropia (1992), Cotropia (1996)]</p> <ul style="list-style-type: none"> • clone 3: Core binding domain gcsgkLIC – lack of serological activity to this region correlates with rapid progression in infants ([Broliden1989]) [Cotropia (1992)] • clone 3: Inhibits replication of three diverse HIV-1 laboratory strains, as well as an AZT-resistant isolate [Cotropia (1996)] 							
618	4	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Oldstone (1991)]</p> <ul style="list-style-type: none"> • 4: There is another MAb with this ID that reacts with integrase [Oldstone (1991), Bizub-Bender (1994)] • 4: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – poor cross-reactivity with HIV-2 peptide CAFRQVC – slightly more reactive with longer HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)] 							
619	41–6	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	murine(IgG2b)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Oldstone (1991)]</p> <ul style="list-style-type: none"> • 41–6: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – poor cross-reactivity with HIV-2 peptide CAFRQVC – slightly more reactive with LGLIWGCSGKLIC and HIV-2 form NSWGCAFRQVC – disulfide bond between cysteines required [Oldstone (1991)] 							
620	41–7	gp160(598–604)	gp41(605–611)	CSGKLIC	no	HIV-1 infection	human(IgG1κ)
<p>References: [Bugge (1990)]</p> <ul style="list-style-type: none"> • 41–7: Sera from 6/6 HIV-1 positive, but no HIV-2 positive individuals, interfered with 41–7 binding [Bugge (1990)] 							
621	68.1	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	murine(IgM)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Oldstone (1991)]</p> <ul style="list-style-type: none"> • 68.1: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – cross-reactive with HIV-2 peptide CAFRQVC – more reactive with longer HIV-1 peptide LGLIWGCSGKLIC and HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)] 							

Table of HIV MAbs

622	68.11	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	murine(IgM)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Oldstone (1991)]</p> <ul style="list-style-type: none"> • 68.11: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – cross-reactive with HIV-2 peptide CAFRQVC – more reactive with longer HIV-1 peptide LGLIWGCSGKLIC and HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)] 							
623	75	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	rat(IgG)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>References: [Oldstone (1991)]</p> <ul style="list-style-type: none"> • 75: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – poor cross-reactivity with HIV-2 peptide CAFRQVC – more reactive with longer HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)] 							
624	105–732	gp160(599–606)	gp41(601–608 HAM112, O group)	KGRLLICYT		Vaccine	murine(IgG2b κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HAM112 (group O) <i>HIV component:</i> gp160</p> <p>References: [Scheffel (1999)]</p> <ul style="list-style-type: none"> • 105–732: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity – MAb 105–732 bound to two overlapping peptides [Scheffel (1999)] 							
625	3D6 (IAM 41–3D6)	gp160(599–613)	gp41(604–617 BH10)	SGKLICTTAVPWNAS	no	HIV-1 infection	human(IgG1 κ)
<p>Ab type: immunodominant region Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX</p> <p>References: [Felgenhauer (1990), He (1992), Chen (1994b), Sattentau (1995), Stigler (1995), Wisnewski (1996), Kunert (1998), Cavacini (1998b), Cavacini (1998a), Cavacini (1999)]</p> <ul style="list-style-type: none"> • 3D6: Sequence of cDNA encoding V- regions [Felgenhauer (1990)] • 3D6: Fab fragment crystal structure [He (1992)] • 3D6: This MAb binds to HIV gp41, and to a 43 kd protein found in human T, B and monocyte cell lines, proposed molecular mimicry [Chen (1994b)] • 3D6: Called IAM 41–3D6: binding increased after pretreatment of infected cells with sCD4 – binding domain overlaps site that is critical for gp120-gp41 association [Sattentau (1995)] • 3D6: Optimum peptide for binding 3D6 Fab was CSGKLICTTAVPW [Stigler (1995)] • 3D6: 3D6 is V H3 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)] • 3D6: The complete V, J and D(H) domain was sequenced – in contrast the sequences of five neutralizing MAbs, 3D6 had very little somatic mutation, with homologies of 97–98% relative to germline genes [Kunert (1998)] • 3D6: Binds to the immunodominant region of gp41 – a strong homology between heavy variable domains of hu MAb 3D6 and MAb F20 was observed, these MAbs may define a human Ab clonotype [Cavacini (1998a)] 							

Table of HIV MAbs

- 3D6: Cavacini *et al.* note that both MAbs F223 and 3D6 are anti-HIV-1 Env MAbs that have an autoimmune response and that both uses VH3 germline genes [Cavacini (1999)]

626	F172-D8 (F172-D8, scFvD8)	gp160(604–615)	gp41(609–620)	CTTAVPWNASWS?		human()
<p>References: [Legastelois & Desgranges(2000)]</p> <ul style="list-style-type: none"> • F172-D8: As an approach to intercellular immunization using a single-chain variable fragment, scFvD8 was constructed based on the MAb F172-D8, directed at a loop in gp41 between the two heptad repeat regions – intracellular scFvD8 expression decreased gp160 expression and a scFvD8 transfected cell line did not support infection by HIV-1 Ba-L or primary isolates [Legastelois & Desgranges(2000)] 						
627	D50	gp160(632–655)	gp41(642–665)		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> dimeric Env</p> <p>Ab type: cluster II References: [Earl (1994), Binley (1996), Richardson (1996), Earl (1997), Srivastava (2002)]</p> <ul style="list-style-type: none"> • D50: Thought to be a discontinuous epitope recognizing residues between 649–668 – designated cluster II – Fabs D5, D11, G1, T3, M12, M15, S6, S8, S9, S10 block binding [Binley (1996)] • D50: Richardson suggests this is a linear gp41 epitope [Richardson (1996)] • D50: Found to bind to a linear peptide, between Env amino acids 642–655 – can be blocked by the conformation dependent MAbs D16, D17, D31, D36, D37, D40, D44, D55, D59, T37, and T45 – the region is in the immunogenic cluster two region – reactive with 9/10 HIV-1 strains tested, all except HIV-1 ADA, in which the change E659D and E662A may result in the loss of binding (ELLE to DLLA) [Earl (1997)] • D50: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – D50 was used to capture the o-gp140 for ELISA to test the antigenicity of o-gp140 using a panel of well characterized MAbs [Srivastava (2002)] 						
628	5–21-3	gp160(642–665)	gp41(642–665 HXB2)	IHSLIEESQNQQEKNEQELLE- LDK	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> gp41</p> <p>References: [Hunt (1990), Scheffel (1999)]</p> <ul style="list-style-type: none"> • 5–21-3: Recognizes a contiguous, conformation-dependent epitope in a hydrophilic region [Hunt (1990)] • 5–21-3: Binds group M gp41, used as a control in a study of group O MAbs [Scheffel (1999)] 						
629	120–16 (SZ- 120.16)	gp160(644–663)	gp41(644–663 HXB2)	SLIEESQNQQEKNEQELLE	no	HIV-1 infection human(IgG2κ)
<p>References: [Andris (1992), Robinson (1990b), Tyler (1990), Xu (1991), Robinson (1991), Eddleston (1993), Forthal (1995), Wisnewski (1996)]</p> <ul style="list-style-type: none"> • 120–16: Antibody dependent enhancement (ADE) of HIV-1 IIIB infectivity, synergistically enhanced by MAb V10–9 [Robinson (1990b)] • 120–16: Potent ADCC (in contrast to MAb 98–43, gp41(579–604)) [Tyler (1990)] 						

- 120–16: Less reactive region than AVERY region – most Abs involving this region bound conformational epitopes, this was the only linear one [Xu (1991)]
- 120–16: Synergizes with huMAb 50–69 *in vitro* to enhance HIV-1 infection [Robinson (1991)]
- 120–16: Called SZ-120.16 [Eddleston (1993)]
- 120–16: No neutralizing activity, both ADCC and viral enhancing activity [Forthal (1995)]
- 120–16: 120–16 is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisniewski (1996)]

630	98–6 (SZ-98.6)	gp160(dis 644–663)	gp41(dis 644–663 HXB2)	SLIEESQNQQEKNEQELLEL	no	HIV-1 infection	human(IgG2 κ)
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Ab type: α -helical bundle **Donor:** Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu), NYU, NY

References: [Pinter (1989), Gorny (1989), Till (1989), Robinson (1990b), Tyler (1990), Andris (1992), Sattentau & Moore(1991), Robinson (1991), Xu (1991), Eddleston (1993), Spear (1993), Tani (1994), Laal (1994), Chen (1995), Forthal (1995), Manca (1995), Sattentau (1995), Wisniewski (1996), Nyambi (1998), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000), Taniguchi (2000), Verrier (2001)]

- 98–6: Reacts preferentially with gp160 oligomer, compared to gp41 monomer [Pinter (1989)]
- 98–6: Kills HIV-infected cells when coupled to deglycosylated ricin A chain [Gorny (1989)]
- 98–6: Toxic to HIV-infected T cells (H9) and monocytes (U937) when coupled to deglycosylated A chain of ricin [Till (1989)]
- 98–6: No neutralizing or enhancing activity for HIV-1 IIIB [Robinson (1990b)]
- 98–6: Serves as target for antibody-dependent cellular cytotoxicity, ADCC [Tyler (1990)]
- 98–6: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 98–6: No neutralizing or enhancing activity [Robinson (1991)]
- 98–6: Appeared to be specific for a conformational or discontinuous epitope [Xu (1991)]
- 98–6: Called SZ-98.6 – binds to a conformational domain within aa 644–663 of gp41, and reacts with astrocytes, as do 167–7 and ND-15G1 [Eddleston (1993)]
- 98–6: Did not mediate deposition of complement component C3 on HIV infected cells, binding enhanced by sCD4 [Spear (1993)]
- 98–6: This MAb was expressed as a surface anti-gp41 monoclonal antibody receptor for gp41 on a CD4-negative B-cell line. Transfected cells could bind HIV Envelope, but could not be infected by HIV-1. When CD4 delivered by retroviral constructs was expressed on these cells, they acquired the ability to replicate HIV-1, and sIg/gp41 specifically enhanced viral replication [Tani (1994)]
- 98–6: Epitope described as cluster II, 644–663, conformational – does not neutralize IIIB or synergize neutralization by anti-V3 MAb 447-52D or by CD4 BS MAbs [Laal (1994)]
- 98–6: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen (1995)]
- 98–6: No neutralizing activity, positive ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 98–6: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 98–6: Preferentially recognizes oligomeric form of gp41 – enhanced binding to HIV-1 infected cells at 37 degrees relative to 4 degrees – addition of sCD4 enhances binding [Sattentau (1995)]
- 98–6: 98–6 is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisniewski (1996)]

Table of HIV MAbs

- 98–6: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-gp41 Abs 98–6, 1367 and 1342 were not able to bind detectably with any of the viruses from any clade [Nyambi (1998)]
- 98–6: 98–6 and 2F5 both bind to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, and to C43 alone but not to N51 alone – 98–6 and 2F5 have comparable affinities for C43, but 98–6 has a higher affinity for the complex and the binding of 98–6 is not inhibited by N51 [Gorny & Zolla-Pazner(2000)]
- 98–6: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)]
- 98–6: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistently to cluster II MAbs – no neutralizing activity was observed when tested against 5 isolates, but 98–6 did not bind to these isolates [Nyambi (2000)]
- 98–6: The fusogenic form of gp41 is recognized by 98–6, and the epitope is a conformational epitope formed by the interaction of two regions of gp41 which form an α -helical bundle [Taniguchi (2000)]
- 98–6: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 $\mu\text{g/ml}$: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 98–6: NIH AIDS Research and Reference Reagent Program: 1240

631	167–7 (SZ-167.7)	gp160(644–663)	gp41(644–663)	SLIEESQNQQEKNEQELLEL		HIV-1 infection	human(IgG2 λ)
		Ab type: cluster II		References: [Xu (1991), Eddleston (1993)]			
		<ul style="list-style-type: none"> ● 167–7: Specific for a conformational epitope [Xu (1991)] ● 167–7: Called SZ-167.7 – binds to a conformational domain within aa 644–663 of gp41, and reacts with astrocytes, as do 98–6 and ND-15G1 [Eddleston (1993)] 					
632	167-D	gp160(644–663)	gp41(644–663 HXB2)	SLIEESQNQQEKNEQELLEL	no	HIV-1 infection	human(IgG1 λ)
		Ab type: cluster II		Donor: Susan Zolla-Pazner (Zollas01@mcr6.med.nyu), NYU, NY			
		References: [Spear (1993), Forthal (1995), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000)]					
		<ul style="list-style-type: none"> ● 167-D: Did not mediate deposition of complement component C3 on HIV infected cells – complement mediated virolysis of MN and IIIB in the presence of sCD4 [Spear (1993)] ● 167-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)] ● 167-D: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)] ● 167-D: This cluster II MAb binds to a conformational epitope in the region 644–663 – like most cluster II MAbs (126-6, 167-D, 1281, 1342, and 1379 all reacted similarly) it binds to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, but not to C43 nor to N51 alone [Gorny & Zolla-Pazner(2000)] 					

- 167-D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)]
- 167-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistently to cluster II MAbs [Nyambi (2000)]

633	ND-15G1	gp160(644–663)	gp41(644–663 HXB2)	SLIEESQNQEQEKNEQELLEL	L P	HIV-1 infection	human(IgG1 κ)
		Ab type: cluster II		References: [Eddleston (1993)]			
		• ND-15G1: Mapped to the conformational epitope within aa 644–663, and reacts with astrocytes, as do 98–6 and 167–7 [Eddleston (1993)]					

634	2F5 (IAM 2F5, IAM- 41–2F5, IAM2F5, c2F5)	gp160(dis 656– 671)	gp41(dis 662–667 BH10)	NEQELLELDKWASLWN	L P	HIV-1 infection	human(IgG3 κ)
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Ab type: adjacent to cluster II **Donor:** Hermann Katinger, U. of Bodenkultur, or Polymun Scientific Inc., Vienna, Austria, or Viral Testing Systems Corp., Houston TX

References: [Buchacher (1992), Muster (1993), Allaway (1993), Klasse (1993a), Purtscher (1994), Laal (1994), Buchacher (1994), D’Souza (1994), Conley (1994b), Thali (1994), Chen (1994b), Muster (1994), Beretta & Dalglish(1994), D’Souza (1995), Trkola (1995), Sattentau (1995), Moore & Ho(1995), Neurath (1995), Kessler 2nd (1995), Calarota (1996), McKeating(1996), Poignard (1996b), Sattentau(1996), Conley (1996), Pincus (1996), McKeating (1996), Stoiber (1996), Purtscher (1996), Schutten (1997), D’Souza (1997), Mo (1997), Li (1997), Kessler II (1997), Moore & Trkola(1997), Mascola (1997), Stamatatos (1997), Turbica (1997), Ugolini (1997), Burton & Montefiori(1997), Earl (1997), Gorny (1997), Andrus (1998), Mondor (1998), Connor (1998), Parren (1998a), Yang (1998), Trkola (1998), Fouts (1998), Ernst (1998), Takefman (1998), Li (1998), Jiang (1998), Parren (1998b), Geffin (1998), Kunert (1998), Frankel (1998), Montefiori & Evans(1999), Poignard (1999), Beddows (1999), Muhlbacher (1999), Parren (1999), Mascola (1999), Mascola (2000), Baba (2000), Gorny & Zolla-Pazner(2000), Kunert (2000), Liao (2000), Lu (2000b), Lu (2000a), Nyambi (2000), Park (2000), Xiao (2000c), Dong (2001), Kolchinsky (2001), Tumanova (2001), York (2001), Zwick (2001b), Zwick (2001c), Mascola & Nabel(2001), Barnett (2001), Moore (2001), Zeder-Lutz (2001), Parker (2001), Spenlehauer (2001), Verrier (2001), Stiegler (2001), Hofmann-Lehmann (2001), Xu (2001), Sanhadji (2000), Coeffier (2000), Armbruster (2002), Srivastava (2002)]

- 2F5: DKWA defined as the core sequence – highly conserved epitope neutralizing MAb [Buchacher (1992), Muster (1993)]
- 2F5: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway (1993)]
- 2F5: Called IAM-41–2F5 – reports MAb to be IgG1 – the gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 2F5 is not affected [Klasse (1993a)]

Table of HIV MAbs

- 2F5: Broadly reactive neutralizing activity, core epitope, ELDKWA, is relatively conserved – neutralized 2 primary isolates [Purtscher (1994)]
- 2F5: Failed to show synergy with anti-CD4 binding site IIIB neutralizing antibodies [Laal (1994)]
- 2F5: MAb generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]
- 2F5: Included in a multi-lab study for antibody characterization binding and neutralization assay comparison [D'Souza (1994)]
- 2F5: Called IAM-41–2F5 – neutralized lab and primary isolates – $t_{1/2}$ dissociation 122 min for the peptide, and 156 min for gp41 – core D(K/R)W – Ab resistant isolate had the sequence KLDNWA [Conley (1994b)]
- 2F5: gp41 mutation (582 A/T) that reduces neutralization of anti-CD4 binding site MAbs does not alter 2F5's ability to neutralize [Thali (1994)]
- 2F5: 2F5 core epitope ELDKWA inserted into an immunogenic loop in influenza virus hemagglutinin can elicit IIIB, MN and RF neutralizing sera in immunized mice [Muster (1994)]
- 2F5: Found to neutralize MN, JRCSF, and two B subtype primary isolates, but not a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs [D'Souza (1995)]
- 2F5: Cross-clade primary virus neutralizing activity – LDKW defined as the core epitope [Trkola (1995)]
- 2F5: Called IAM 41–2F5 – exposed in the presence of gp120 on the cell surface, while most of gp41 is masked – binds proximal to transmembrane region [Sattentau (1995)]
- 2F5: Review: binds to the only generally accepted strong neutralizing epitope outside of gp120, one of only 3 MAbs with strong broad activity against primary viruses, the others are 2G12 and IgG1b12 – unique member of epitope cluster [Moore & Ho(1995)] and John Moore, per comm 1996
- 2F5: MAb binding decreases the accessibility or alters the conformation of the gp41 fusion domain and of gp120 domains, including the binding site for the CD4 cell receptor [Neurath (1995)]
- 2F5: Broad cross-clade neutralization of primary isolates – additive neutralization in combination with anti-CD4BS MAb IgG1b12 (Called BM12) [Kessler 2nd (1995)]
- 2F5: Only 4/20 Argentinian and 3/43 Swedish HIV+ sera reacted with LLELDKWASL – sera reacting with peptides that contained ELDKWA tended to have high neutralization titers – the region carboxyl terminal to EDLKWA was found to be more important for polyclonal sera AB binding, 670–675 WNWFDI – 2F5 bound most strongly to the peptide QELLELDKWA [Calarota (1996)]
- 2F5: ELDKWAS is in a gp41 binding region for the negative regulator of complement factor H (CFH) – Abs to HIV generally do not cause efficient complement-mediated lysis, but binding of 2F5 can interfere with CHF binding, facilitating HIV destruction by complement [Stoiber (1996)]
- 2F5: Primary isolates from clade A, B, and E are neutralized by 2F5 – neutralization requires the LDKW motif – neutralization resistant isolates or 2F5 selected variants all had substitutions in the D or K [Purtscher (1996)]
- 2F5: Neutralizes HXB2, primary isolates, and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]
- 2F5: Review: one of three MAbs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard (1996b)]
- 2F5: Review: only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]

- 2F5: 2F5 was infused into two chimpanzees which were then given an intravenous challenge with a primary HIV-1 isolate – both became infected, but with delayed detection and prolonged decrease in viral load relative to controls, indicating that preexisting, neutralizing antibodies (passively administered or actively elicited) affect the course of acute-phase virus replication and can be influential after the Ab can no longer be detected in the peripheral circulation [Conley (1996)]
- 2F5: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)]
- 2F5: Called IAM 2F5 – antibody mediated enhancement or inhibition seemed to be determined by isolate rather than antibody specificity – in this study, only 2F5 inhibited the entry of all the viruses studied, irrespective of their phenotype, and directly proportional to its affinity to monomeric HIV-1 gp160 [Schutten (1997)]
- 2F5: Of three neutralizing MAbs (257-D, IgG1b12, and 2F5), 2F5 was the only one to inhibit the entry of all viruses studied, both SI and NSI, with a potency proportional to its affinity for monomeric gp126 [Schutten (1997)]
- 2F5: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 μg per ml for 90% viral inhibition – the isolates with no 2F5 neutralizing susceptibility had the sequences ALGQWA or ELDTWA instead of EDLKWA – 7/9 primary isolates were neutralized, and ALDKWQ and ALDKWA were susceptible to neutralization [D'Souza (1997)]
- 2F5: A JRCSF variant that was selected for IgG1b12 resistance remained sensitive to MAbs 2G12 and 2F5, for combination therapy [Mo (1997)]
- 2F5: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB Env – strong neutralizer of SHIV-vpu+ – all Ab combinations tested showed synergistic neutralization – 2F5 has synergistic response with MAbs 694/98-D (anti-V3), 2G12, b12, and F105 [Li (1997)]
- 2F5: IgG1b12 was more potent with greater breadth than MAb 2F5 in an infection reduction assay including 35 primary isolates [Kessler II (1997)]
- 2F5: Review: MAbs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MAbs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs' epitopes [Moore & Trkola(1997)]
- 2F5: Binding of anti-gp120 MAbs IgG1b12 or 654–30D does not mediate significant exposure of the gp41 epitopes for MAbs 2F5 and 50–69 [Stamatatos (1997)]
- 2F5: Using concentrations of Abs achievable *in vivo*, the triple combination of 2F5, 2G12 and HIVIG was found to be synergistic to have the greatest breadth and magnitude of response against 15 clade B primary isolates [Mascola (1997)]
- 2F5: Used to standardize polyclonal response to CD4 BS [Turbica (1997)]
- 2F5: The only MAb out of a large panel to show no correlation between Viral binding inhibition and neutralization [Ugolini (1997)]
- 2F5: This review summarizes results about 2F5: it binds extracellularly, near the transmembrane domain, it is the only gp41 MAb that is neutralizing, it reacts with many non-B clade viruses and has a paradoxically weak binding to virus, given the neutralizing titers [Burton & Montefiori(1997)]
- 2F5: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIV-1 LAI in hu-PBL-SCID mice, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus (1998)]

Table of HIV MAbs

- 2F5: This MAb and the results of [Ugolini (1997)] are discussed – the authors propose that an Ab bound to gp41 would typically project less from the surface of the virion and so be unable to interfere with attachment [Parren (1998a)]
- 2F5: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D [Connor (1998)]
- 2F5: A neutralization assay was developed based on hemi-nested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang (1998)]
- 2F5: A wide range of neutralizing titers was observed that was independent of co-receptor usage – 2F5 was the most potent of the MAbs tested [Trkola (1998)]
- 2F5: Points out that 2G12 and 2F5, potent neutralizing antibodies, were identified by screening for cell surface (oligomeric Envelope) reactivity [Fouts (1998)]
- 2F5: The ELDKWA core epitope was inserted into the antigenic site B of influenza hemagglutinin and expressed on baculovirus infected insect cells, flanked by 3 additional random amino acids, xELDKWaxx – FACS was used to isolate the clone that displayed the epitope with the most markedly increased binding capacity for 2F5, to identify particularly specific immunogenic constructs – PELDKWAPP was a high affinity form selected by FACS [Ernst (1998)]
- 2F5: Induces complement-mediated lysis in MN but not primary isolates – primary isolates are refractive to CML [Takefman (1998)]
- 2F5: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) [Li (1998)]
- 2F5: Used as a control in the study of anti-gp41 MAb NC-1 – 2F5 does not react with HIV-2 gp41 or gp160 [Jiang (1998)]
- 2F5: MAbs 2G12, 2F5 and b12 are broadly neutralizing, as are some human polyconal sera, but this paper describes a set of primary isolates that are resistant to all three MAbs and 2 broadly neutralizing sera – results indicate that resistance levels of pediatric isolates might be higher than adult isolates – resistance in general did not seem to be conferred by a loss of binding affinity for gp120 or gp41, rather by a more global perturbation of oligomeric Envelope [Parren (1998b)]
- 2F5: The natural immune response to the core epitope of 2F5, ELDKWA, was studied in perinatally infected children and levels of reactivity to this epitope were correlated with absolute CD4 numbers over time and health status – 3/10 children who had no antibody reactivity to ELDKWA had substitutions in the epitope (ALDKWA, ELDQWA, and KLDKWA) – 2F5 competed with the ELDKWA-reactive sera depending on the serum titer [Geffin (1998)]
- 2F5: The complete V, J and D(H) domain was sequenced – unlike non-neutralizing anti-gp41 MAb 3D6, five neutralizing MAbs (2F5, 2G12, 1B1, 1F7, and 3D5) showed extensive somatic mutations giving evidence of persistent antigenic pressure over long periods – in contrast to Geffin98, where multiple pediatric sera were found to compete with 2F5, cross-competition was noted to be very rare in sera from HIV+ adults – Kunert *et al.* propose that because there is a binding site of human complement factor H which overlaps the 2F5 binding site, it may generally be masked from the immune system – 2F5 also has a remarkably long CDR3 loop of 22 amino acids, and this region could not be readily assigned to any described D(H) fragment, leading to the suggestion of recombination of two fragments from novel regions [Kunert (1998)]
- 2F5: Infection of dendritic cells cultured from CD14+ blood cells or from cadaveric human skin was blocked by neutralizing MAbs IgG1b12, or 2F5 and 2G12 delivered together, but not by control non-neutralizing anti-gp120 MAb 4.8D, indicating that NAbs could interrupt early mucosal transmission events [Frankel (1998)]

- 2F5: rgp120 derived from a R5X4 subtype B virus was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs [Beddows (1999)]
- 2F5: A meeting summary presented results regarding neutralization –MAbs 2G12 and 2F5 tested for their ability to neutralize primary isolate infection of genetically engineered cell lines (cMAGI and others, presented by T. Matthews, A. Trkola, J. Bradac) – an advantage of such cell lines over PBMCs is that markers (X-Gal) can be added for staining to simplify the assay – the consensus of the meeting was that these engineered cell lines did not improve the sensitivity of detection of primary isolate neutralization – D. Burton and J. Mascola presented results concerning passive immunization and protection of hu-PBL-SCID mice and macaques, respectively, and both found combinations of MAbs that were able to achieve 99% neutralization *in vitro* corresponded to efficacy *in vivo* [Montefiori & Evans(1999)]
- 2F5: Hu-PBL-SCID mice were infected with HIV-1s JRCSF and SF162 to study the effect of NAb on an established infection – no significant differences in the initial rate of decrease in viral load or the plateau levels of viral RNA between the b12 treated and control mice were seen – in most of the Ab treated mice b12 escape mutants were observed with varying patterns of mutations – a combination of b12, 2G12 and 2F5 protected 1/3 mice, and an isolate from one of the other two was resistant to neutralization by all three MAbs [Poignard (1999)]
- 2F5: In a study of 116 HIV-1+ individuals, Ab reactivity to a peptide encompassing the ELDKWA peptide decreased in CDC stage C patients compared with stage A patients, and longitudinal studies showed a decline in 6/8 patients, while overall Ab reactivity to rec soluble gp160 stayed constant [Muhlbacher (1999)]
- 2F5: Review of the neutralizing Ab response to HIV-1 [Parren (1999)]
- 2F5: Combinations of HIVIG, 2F5, 2G12 were administered in passive-transfer experiments 24 hours prior to challenge with pathogenic SHIV 89.6PD – 3/6 animals given HIVIG/2F5/2G12 were completely protected, the others had reduced viremia and normal CD4 counts – 1/3 monkeys given 2F5/2G12 showed transient infection, the other two had reduced viral load – all monkeys that received HIVIG, 2F5, or 2G12 alone became infected and developed high-level plasma viremia, although animals that got HIVIG or 2G12 had a less profound CD4 T cell decline [Mascola (1999)]
- 2F5: Because HIV-1 is most often transmitted across mucosal surfaces, the ability of passive transfer of infused HIVIG/2F5/2G12 to protect against mucosal exposure of macaques to pathogenic SHIV 89.6PD was studied – HIVIG/2F5/2G12 protected 4/5 animals against vaginal challenge, 2F5/2G12 combined protected 2/5 animals, and 2G12 alone protected 2/4 animals – in contrast, Mascola and co-workers had previously shown single MAbs could not protect against intravenous challenge – Ab treated animals that got infected through vaginal inoculation had low viral loads and only modest declines in CD4 counts – the infused Abs were detected in the nasal, vaginal, and oral mucosa [Mascola (2000)]
- 2F5: Paper uses IgG1 form of 2F5 – a triple combination of 2F5, F105 and 2G12 effectively neutralized perinatal infection of macaque infants when challenged with SHIV-vpu+ – the plasma half-life was 4.2 ± 0.8 days [Baba (2000)]
- 2F5: MAbs 98–6 and 2F5 both bind to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, and to C43 alone but not to N51 alone – 98–6 and 2F5 have comparable affinities for C43, but 98–6 has a higher affinity for the complex and 2F5 may bind to an epitope of C43 that is directly involved with complex formation –and IgG1 rec form of the Ab was used in this study [Gorny & Zolla-Pazner(2000)]
- 2F5: 2F5 is a candidate for immunotherapy, but generally IgG1 has a longer half-life in humans than IgG3, so the isotype was switched – rec CHO-derived MAb 2F5 IgG1kappa and hybridoma-derived MAb 2F5 IgG3kappa displayed identical specificity, *in vitro* function, and epitope (ELDKWA) – it remains to be determined if isotype switching will prolong β -clearance [Kunert (2000)]

Table of HIV MAbs

- 2F5: Low levels of anti-ELDKWA antibodies are observed in HIV-1+ individuals, so a C-domain P2 peptide linked to a carrier was used to immunize mice and rabbits, and stimulated a high-level anti-ELDKWA response [Liao (2000)]
- 2F5: ELDKWA peptide vaccine study [Lu (2000b)]
- 2F5: ELDKWA peptide vaccine study [Lu (2000a)]
- 2F5: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98-6 and 1342 had poor cross reactivity – Clade D isolates bound most consistently to cluster II MAbs [Nyambi (2000)]
- 2F5: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i gp120 specific MAbs are 20–100 fold more efficient at neutralizing the sensitive form – gp41 MAbs bind less, and 2F5 behaves the opposite of gp120 MAbs in that it neutralizes the “sensitive” form less efficiently [Park (2000)]
- 2F5: Mutations in two glycosylation sites in the V2 region of HIV-1 ADA at positions 190 and 197 (187 DNTSYRLINCNTS 199) cause the virus to become CD4-independent and able to enter cells through CCR5 alone – these same mutations tended to increase the neutralization sensitivity of the virus, including to antibody 2F5 [Kolchinsky (2001)]
- 2F5: A peptide called 5-Helix was designed that binds to the C-peptide region of gp41 – 5-Helix is a potent inhibitor of HIV-1 entry that binds immediately COOH-terminal to the C-peptide region targeted by 5-Helix – the conformation of the bound 2F5 epitope is a hairpin turn [Root2001a]
- 2F5: A phage peptide library was screened with MAb 2F5, and from the peptides that bound the amino acids DKW were found to be most critical for binding – the mimetic peptide RDWSFDRWSLSEFWL elicited a cross-reactive Ab response to gp41 when used to immunize rabbits [Tumanova (2001)]
- 2F5: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding [York (2001)]
- 2F5: This paper primarily concerns 4E10 and Z13, MAbs that both bind proximally to the 2F5 binding site to a conserved epitope, and that neutralize some primary isolates from clades B, C, and E – the minimal 2F5 epitope is determined to be EQELLELDKWASLW, based on screening a gp160 fragment expression library, longer than previous studies – broadly neutralizing MAbs 2F5, IgG1b12, and 4E10 and Z13 fail to neutralize different subsets of viruses [Zwick (2001b)]
- 2F5: Neutralization synergy between anti-HIV NAbs b12, 2G12, 2F5, and 4E10 was studied – a classic fixed-ratio method was used, as well as a method where one Ab was fixed at a low neutralization titer and the other was varied – using primary isolates, a two to four fold enhancement of neutralization was observed with MAb pairs, and a ten-fold enhancement with a quadruple Ab combination – no synergy was observed with any MAb pair in the neutralization of TCLA strain HXB2 [Zwick (2001c)]
- 2F5: Review of studies in macaques that have shown immune control of pathogenic SHIV viremia, improved clinical outcome, and protection, and the implications of the observations for HIV vaccines [Mascola & Nabel(2001)]

- 2F5: SF162ΔV2 is a virus that has a 30 amino acid deletion in the V2 loop that does not abrogate its infectivity but renders it highly susceptible to neutralization – when incorporated into a codon-optimized DNA vaccine with a CMV promoter and delivered by gene gun, SF162ΔV2 gave higher neutralizing Ab titers against SF162 than did SF162 itself, and Abs that cross-neutralized non-homologous primary isolates were obtained only when SF162ΔV2, but not intact SF162, was used as the immunogen – Control MAbs 2F5 and 2G12 could neutralize all of the following primary isolates: 91US056(R5), 92US714(R5), 92US660(R5), 92HT593(R5X4), and BZ167(R5X4), while after the first protein boost, the sera from two SF162ΔV2 immunized macaques could neutralize 91US056(R5), 92US714(R5), 92US660(R5) and ADA(R5), but not 92HT593(R5X4) or 92US657(R5) – the pattern of cross-recognition shifted after the second boost [Barnett (2001)]
- 2F5: Moore and colleagues review the data concerning the lack of a clear relationship between genetic subtype and serotype – 2F5 is considered in some detail, as it represents a rare vulnerability from the neutralizing antibody perspective, although while it is apparently linear, attempts to present the peptide to the immune system have failed to elicit neutralizing Abs [Moore (2001)]
- 2F5: Neutralizing synergy between MAbs 1b12, 2G12 and 2F5 was studied using surface plasmon resonance to determine the binding kinetics for these three mAbs with respect to monomeric and oligomeric env protein gp160 III_B – the 2G12 epitope is highly accessible on both monomeric and oligomeric Envs, 1b12 is highly accessible on monomers but not oligomers, and 2F5 on neither form – binding of 2G12 exposes the 2F5 epitope on gp160 oligomers [Zeder-Lutz (2001)]
- 2F5: Matrix-assisted laser desorption ionization mass spectrometry (MALDI-MS) in combination with proteolytic protection was used to identify the functional epitope for MAb 2F5, NEQELLELDKWASLWN, in the disulfide bond associated gp120/gp41 protein SOS-gp140 (JRFL) – this minimal epitope is much larger than the ELDKWA core epitope previously defined by peptide ELISA, and this could help explain why ELDKWA-peptides are poor immunogens in terms of eliciting a 2F5-like antibody response [Parker (2001)]
- 2F5: A luciferase-reporter gene-expressing T-cell line was developed to facilitate neutralization and drug-sensitivity assays – luciferase and p24 antigen neutralization titer end points were found comparable using NAb from sera from HIV+ donors, and MAbs 2F5, 2G12 and IgG1b12 [Spencehauer (2001)]
- 2F5: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 μg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 2F5: 4E10 binds proximal to 2F5 and neutralizes primary isolates of clades A, B, C, D, and E – viruses that were resistant to 2F5 were neutralized by 4E10 and vice versa [Stiegler (2001)]
- 2F5: A combination of MAbs IgG1b12, 2F5, and 2G12 was given postnatally to four neonate macaques that were then challenged with highly pathogenic SHIV89.6P – one of the four infants remained uninfected after oral challenge, two infants had no or a delayed CD4(+) T-cell decline [Hofmann-Lehmann (2001)]
- 2F5: Twenty HIV clade C isolates from five different countries were susceptible to neutralization by anti-clade B MAbs in a synergistic quadruple combination of mAbs IgG1b12, 2G12, 2F5, and 4E10 [Xu (2001)]
- 2F5: 2F5 or sCD4-IgG chimeric immunoadhesin were transferred into 3T3 cells, incorporated into a collagen structure called the neo-organ, and transplanted into SCIDhu mice that were then challenged with MN or LAI – the continuous production of the therapeutic molecules in this context resulted in dramatic reduction of viral load [Sanhadji (2000)]

Table of HIV MAbs

- 2F5: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – 2F5 recognized o-gp140 [Srivastava (2002)]
- 2F5: UK Medical Research Council AIDS reagent: ARP3063
- 2F5: NIH AIDS Research and Reference Reagent Program: 1475

635	polyclonal	gp160(662–667)	gp41(662–667)	ELDKWA;	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> E. coli MalE protein <i>HIV component:</i> gp41 peptide</p> <p>References: [Coeffier (2000)]</p> <ul style="list-style-type: none"> • The antigenicity of ELDKWA inserted in MalE protein was estimated from 2F5 binding analysis using BIAcore(R) and its immunogenicity in mice was measured – specific but non-neutralizing MAbs were raised [Coeffier (2000)] 							
636	polyclonal	gp160(662–667)	gp41()	ELDKWA	L P	Vaccine	rabbit()
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41</p> <p>Ab type: C-domain References: [Liao (2000)]</p> <ul style="list-style-type: none"> • Low levels of anti-ELDKWA antibodies are observed in HIV-1+ individuals, so a C-domain P2 peptide linked to a carrier was used to immunize mice and rabbits, and stimulated a high-level anti-ELDKWA response in mice and rabbits – vaccine was C-TSLIHSLIEESQNQQEKNEQELLELDKWA linked to carrier peptide K/G [(KGGG)_7-K] [Liao (2000)] 							
637	polyclonal	gp160(662–667)	gp41(669–674)			Vaccine	mouse, rabbit()
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> Env <i>Stimulatory Agents:</i> BSA</p> <p>Ab type: C-domain References: [Xiao (2000b)]</p> <ul style="list-style-type: none"> • Strong epitope-specific neutralizing antibody responses were induced using a Env peptide bound to BSA, C(ELDKWAG)_4-BSA, but not full gp160 [Xiao (2000b)] 							
638	polyclonal	gp160(662–667)	gp41(662–667 BH10)	ELDKWA	L	Vaccine	murine(IgG,IgA)
<p>Vaccine: <i>Vector/type:</i> influenza virus <i>Strain:</i> BH10 <i>HIV component:</i> gp41 peptide</p> <p>Ab type: C-domain References: [Muster (1994), Muster (1995)]</p> <ul style="list-style-type: none"> • Sustained ELDKWA specific IgA response in mucosa of immunized mice [Muster (1995)] 							
639	polyclonal	gp160(662–667)	gp120(669–674)	ELDKWA		Vaccine	rabbit(Ig)
<p>Vaccine: <i>Vector/type:</i> polypeptide, protein <i>HIV component:</i> gp160 <i>Stimulatory Agents:</i> BSA</p> <p>Ab type: C-domain References: [Lu (2000b), Lu (2000a)]</p> <ul style="list-style-type: none"> • High titer response to ELDKWA and RILAVEYLKD was observed upon vaccination with multiple-epitope vaccine CG-GPGRIFY-G-ELDKWA-G-RILAVEYLKD conjugated to BSA, with a weak response to GPGRIFY – immunization with CG-(ELDKWA-GPGRIFY)_2-K was also tried, yielding a strong Ab response to both ELDKWA and GPGRIFY – gp160 vaccination yielded strong Ab response but not to any of the peptides studied here [Lu (2000b), Lu (2000a)] 							

640	TH-Ab1	gp160(662–667)	gp41(669–674)	ELNKWA	L and P	Vaccine	rabbit(IgG1)
	Vaccine:	<i>Vector/type:</i> peptide		<i>Strain:</i> B clade TH936705	<i>HIV component:</i> gp41	<i>Stimulatory Agents:</i> Freund's adjuvant	
		Ab type: C-domain		References: [Xiao (2000a), Dong (2001)]			
		<ul style="list-style-type: none"> • TH-Ab1: ELNKWA is an escape variant not recognized by the broadly neutralizing MAb 2F5, which recognizes the core epitope ELDKWA – Abs were raised against the peptide escape variant CGELNKWAGELNKWA linked to keyhole limpet carrier protein – these polyclonal antibodies, like the monoclonal antibody TH-Ab1 also raised to ELNKWA, could recognize ELDKWA and escape mutant peptide epitopes ELEKWA and ELDEWA [Dong (2001)] 					
641	5B2	gp160(662–668)	Env(669–674 IIIB)	ELDKWA		Vaccine	mouse(IgG)
	Vaccine:	<i>Vector/type:</i> peptide in keyhole limpet hemocyanin		<i>Strain:</i> IIIB	<i>HIV component:</i> gp41		
		Ab type: C-domain		References: [Tian (2001)]			
		<ul style="list-style-type: none"> • 5B2: There is an RT specific Ab [Szilvay (1992)] and a gp41 specific Ab [Tian (2001)] both called 5B2 • 5B2: Peptides GPGRAFY and ELDKWA were conjugated to keyhole limpet hemocyanin and used to raise mouse MAbs – MAb hybridomas were generated with defined specificity – 5B2 and 9G11 bind to the peptide and to rgp41 [Tian (2001)] 					
642	9G11	gp160(662–668)	Env(669–674 IIIB)	ELDKWA		Vaccine	mouse(IgG)
	Vaccine:	<i>Vector/type:</i> peptide in keyhole limpet hemocyanin		<i>Strain:</i> IIIB	<i>HIV component:</i> gp41		
		Ab type: C-domain		References: [Tian (2001)]			
		<ul style="list-style-type: none"> • 9G11: Peptides GPGRAFY and ELDKWA were conjugated to keyhole limpet hemocyanin and used to raise mouse monoclonal Ab – MAb hybridomas were generated with defined specificity – 5B2 and 9G11 bind to the peptide and to rgp41 [Tian (2001)] 					
643	4E10	gp160(671–676)	gp160(671–676 MN)	NWFDIT	P	HIV-1 infection	human(IgG3 κ)
		Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, Vienna, Austria					
		References: [Buchacher (1992), Buchacher (1994), D'Souza (1994), Stiegler (2001), Zwick (2001b), Zwick (2001c), Xu (2001)]					
		<ul style="list-style-type: none"> • 4E10: MAbs generated by hybridoma, electrofusion of PBL from HIV-1+ volunteers with CB-F7 heteromyeloma cells – also binds to MHC class II proteins – anti-class II Abs are only found in HIV-1 positive people – this paper maps 4E10's binding site to AEGTDRV, gp160(823–829), but the later Zwick <i>et al.</i> study in 2001 revised the epitope location [Buchacher (1994)] • 4E10: Included in a multi-lab study for antibody characterization, binding and neutralization assay comparison [D'Souza (1994)] • 4E10: 4E10 binds proximal to 2F5 and neutralizes primary isolates of clades A, B, C, D, and E – viruses that were resistant to 2F5 were neutralized by 4E10 and vice versa [Stiegler (2001)] • 4E10: MAbs 4E10 and Z13 both bind proximally to 2F5 to a conserved linear epitope that has some conformational aspects – both bind to MN virions, bind weakly to infected cells in a manner that is not disrupted by sCD4 and neutralize some primary isolates from clades B, C, and E – maps minimal 4E10 epitope to NWFDIT, contrary to an earlier report – different strains were refractive to neutralization by broadly neutralizing Abs IgG1b12, 2F5, Z13 and 4E10 [Zwick (2001b)] 					

Table of HIV MAbs

- 4E10: Neutralization synergy between anti-HIV NAb b12, 2G12, 2F5, and 4E10 was studied – a classic fixed-ratio method was used, as well as a method where one Ab was fixed at a low neutralization titer and the other was varied – using primary isolates, a two-four fold enhancement of neutralization was observed with MAb pairs, and a ten-fold enhancement with a quadruple Ab combination – no synergy was observed with any MAb pair in the neutralization of TCLA strain HXB2 [Zwick (2001c)]
- 4E10: Twenty HIV clade C isolates from five different countries were susceptible to neutralization by anti-clade B MAbs in a synergistic quadruple combination of mAbs IgG1b12, 2G12, 2F5, and 4E10 [Xu (2001)]

644	Z13	gp160(671–676) Ab type: C-term	gp41(671–676 MN) References: [Zwick (2001b)]	NWFDIT	P	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Z13: MAbs 4E10 and Z13 both bind proximally to 2F5 to a relatively conserved linear epitope that has some conformational aspects – both bind to MN virions, bind weakly to infected cells in a manner that is not disrupted by sCD4 and can neutralize some primary isolates from clades B, C, and E – Z13 was selected using an antibody phage display library with the MN gp41 peptide LLELDKWASLWNWFDITNWSW from an HIV infected donor who had an exceptionally broad NAb response – different strains were refractive to neutralization by broadly neutralizing Abs IgG1b12, 2F5, Z13 and 4E10 – epitope location noted here is by analogy to MAb 4E10 [Zwick (2001b)]; _____ 							
645	B30	gp160(720–734)	gp41(720–734 BH10)	HLPIPRGPDPRPEGIE		Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> LAI <i>HIV component:</i> gp160 Donor: George Lewis References: [Abacioglu (1994)]</p> <ul style="list-style-type: none"> • B30: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)] 							
646	polyclonal	gp160(724–745)	gp41(731–752)	PRGPDRPEGIEEEGGGERDRDRS		Vaccine	murine(IgA,IgG2a)
<p>Vaccine: <i>Vector/type:</i> Cowpea mosaic virus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide References: [Durrani (1998)]</p> <ul style="list-style-type: none"> • Comparison of intranasal and oral immunization of HIV-1 peptide expressed in a plant viral vector – intranasal gave the better response [Durrani (1998)] 							
647	41S-2	gp160(725–745)	gp160(732–750)	RGPDRPEGIEEEGGGERDRDRS	yes	Vaccine	murine(IgG2b κ)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> gp41 <i>Stimulatory Agents:</i> keyhole limpet hemocyanin References: [Hifumi (2000)]</p> <ul style="list-style-type: none"> • 41S-2: BALBc mice were immunized with gp41 peptide and a MAb specific for the peptide was generated – isolated MAb light chains displayed proteolytic activity towards the peptide epitope which may be due to a catalytic triad on light chain (Asp73, Ser76, and His79) – no catalytic activity was observed for the whole antibody [Hifumi (2000)] 							

648 447-52D gp160(312–315) gp120(MN) GPXR L HIV-1 infection human(IgG3λ)
 (447/52-DII, 447-52-D, 447d, 447-52-D, 447-D, 447, 447D)

Ab type: V3 **Donor:** Dr. Susan Zolla-Pazner, NYU Med Center NY, NY, or Cellular Products Inc, Buffalo, NY, USA

References: [Gorny (1992), Buchbinder (1992), Karwowska (1992b), Gorny (1993), Keller (1993), Cavacini (1993a), Spear (1993), Conley (1994a), Laal (1994), VanCott (1994), Gorny (1994), Moore (1994a), Sattentau(1995), Fontenot (1995), Saarloos (1995), Zolla-Pazner (1995), Zolla-Pazner & Sharpe(1995), Moore (1995a), Moore & Ho(1995), Forthal (1995), Jagodzinski (1996), Trkola (1996a), Sattentau(1996), D'Souza (1997), Binley (1997a), Fouts (1997), Hioe (1997), Boots (1997), Parren (1997b), Hill (1997), Gorny (1997), Inouye (1998), Mondor (1998), Smith (1998), Parren (1998a), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Connor (1998), Gorny (1998), Nyambi (1998), Hioe (1999), Beddows (1999), Gorny (2000), Grovit-Ferbas (2000), Hioe (2000), Ly & Stamatatos(2000), Nyambi (2000), Park (2000), York (2001), Verrier (2001), Srivastava (2002)]

- 447-52D: Requires GPXR at the tip of the V3 loop – neutralizes a broad array of B clade lab isolates [Gorny (1992)]
- 447-52D: 60-fold increase in neutralization potency when combined 1:1 with human MAb 588-D [Buchbinder (1992)]
- 447-52D: Reacts with MN, NY5, CDC4, SF2, RF, WM52, and HXB2 [Karwowska (1992b)]
- 447-52D: Neutralizes MN and IIIB: GPGR, and binds SF2: GPGR [Gorny (1993)]
- 447-52D: Peptide phage library showed that any of the residues ADGLMNQRS in the X position tolerated in peptides that react well with the antibody [Keller (1993)]
- 447-52D: Additive neutralization of MN and SF2 when combined with CD4 binding site MAb F105 – supra-additive neutralization of RF [Cavacini (1993a)]
- 447-52D: Complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear (1993)]
- 447-52D: Requires GPxR at the tip of the V3 loop, common in B clade – neutralized primary isolates [Conley (1994a)]
- 447-52D: Neutralization synergy in combination with CD4 binding domain MAbs [Laal (1994)]
- 447-52D: GPGQ in MAL resulted in enhanced dissociation – GPGQ in CM234 or K14T did not bind – binding affected by identity of amino acids flanking GPGR core [VanCott (1994)]
- 447-52D: Mild oxidation of carbohydrate moieties does not alter binding [Gorny (1994)]
- 447-52D: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore (1994a)]
- 447-52D: Called 447d – Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau (1995)]
- 447-52D: Called 447 – The tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot (1995)]

Table of HIV MAbs

- 447-52D: Ab-mediated activation of complement on HIV+ cells is higher than Ab independent activation – what has been termed “Ab independent” in fact results in part from IgM in normal human serum that is HIV-cross-reactive [Saarloos (1995)]
- 447-52D: Serotyping study using flow-cytometry – bound only to GPGR V3 loop tips [Zolla-Pazner (1995)]
- 447-52D: Neutralization of primary and prototype laboratory HIV-1 isolates using a resting cell assay enhances sensitivity [Zolla-Pazner & Sharpe(1995)]
- 447-52D: Binding affected by identity of amino acids flanking GPGR core – poor breadth of primary virus neutralization [Moore (1995a)]
- 447-52D: Review: the V3 loop motif GPGR is not common outside subtype B isolates, MAb 19b is more cross-reactive [Moore & Ho(1995)]
- 447-52D: Neutralizing (- complement), no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 447-52D: Called 447-52-D – The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits binding [Jagodzinski (1996)]
- 447-52D: Neutralizes JR-FL – strongly inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola (1996a)]
- 447-52D: Review: called 447-52-D – only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]
- 447-52D: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – many of these isolates had the GPGR motif at the apex of the V3 loop [D’Souza (1997)]
- 447-52D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 447-52D bound monomer, oligomer, and neutralized JRFL [Fouts (1997)]
- 447-52D: Tested using a resting cell neutralization assay [Hioe (1997)]
- 447-52D: Viral binding inhibition by 447-D was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- 447-52D: Neutralizes TCLA strains but not primary isolates [Parren (1997b)]
- 447-52D: Called 447 – gp120 can inhibit MIP-1 α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill (1997)]
- 447-52D: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – 447-52D has an epitope involving the tip of the V3 loop, that was previously studied with this method [Keller (1993)] – in Keller *et al.*, with no competition, LxGPxR was the most common six-mer, 38% of the peptides – after competition with a gp120 IIIB ligand (QRGPGR)i, RGPxR was the most common and one peptide had the sequence QRGPGR, showing type specific mimotyopes can be enriched by strain specific ligand competition protocols [Boots (1997)]
- 447-52D: Used as a control for comparison to five V3 RF selected antibodies – 447-52D was reactive with A, B, and C clade peptides, but not E [Gorny (1997)]
- 447-52D: Called 447-D – 447-D resistance took longer to acquire in virus with the M184V substituted RT, and had the form (AAC N to TAC Y) at position 5 of the V3 loop, rather than the GPGR to GPGR resistance found with wildtype RT [Inouye (1998)]
- 447-52D: Inhibits binding of Hx10 to both CD4 positive and negative HeLa cells [Mondor (1998)]
- 447-52D: Called 447-52-D – The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 447-52D was among the Abs used – chimeric viruses elicited potent NABs in guinea pigs against ALA-1 and MN [Smith (1998)]

- 447-52D: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- 447-52D: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D [Connor (1998)]
- 447-52D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 1324E was comparable to 447-52D [Gorny (1998)]
- 447-52D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 447-52D was the most potent and cross-reactive of 18 human MAbs tested and was the only MAb which bound to virions from isolates CA20 (subtype F), CA13 (subtype H), and VI526 (subtype G) [Nyambi (1998)]
- 447-52D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 447-52D: MAb peptide-reactivity pattern clustered with the immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group – 447 reacted with peptides containing GPGR, but also with many lacking this sequence (GPGQ, for example), and it failed to react with 2/14 peptides containing GPGR, illustrating the importance of context [Zolla-Pazner (1999b)]
- 447-52D: The presence of leukocyte function-associated molecule 1 (LFA-1) promotes virus infectivity and hinders neutralization, and anti-LFA-1 MAbs can enhance the neutralizing effect of anti-HIV V3 MAb 447-52D and anti-HIV CD4BS MAb IgG1b12 – non-neutralizing anti-HIV CD4BS MAb 654-D did not become neutralizing in the presence of anti-LFA-1 MAbs [Hioe (1999)]
- 447-52D: rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – TCLA strains showed enhanced 447-52D neutralization sensitivity relative to PBMC-adapted lines (32X increase between HIV-1(M2424/PBMC(p0)) and HIV-1(M2424/H9(p9)) and a >128X increase between HIV-1(W61D/PBMC) and HIV-1(W61D/SupT1) isolates) [Beddows (1999)]
- 447-52D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V3 MAbs 447-52D, 838-D, and 1334 bound with a 7–10 fold preference for the oligomer [Gorny (2000)]
- 447-52D: To determine the antigenicity of virus killed by thermal and chemical inactivation, retention of conformation-dependent neutralization epitopes was examined, and exposure of CD4BS epitopes was found to be enhanced (MAbs IgG1b12, 205-46-9, and 205-43-1) – binding to 2G12 and 447-52D epitopes was essentially unaltered – the 17b CD4i epitope was also exposed [Grovit-Ferbas (2000)]
- 447-52D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – V3 MAbs 447-52D and 268–10-D did not effect proliferation [Hioe (2000)]
- 447-52D: Called 447D – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447D and 391–95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]

Table of HIV MAbs

- 447-52D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 447-52D showed the highest cross-reactivity, bound to 24/26 viruses tested, but achieved 90% neutralization only against MN, 50% against CA5, and no neutralization was observed for 3 other isolates tested [Nyambi (2000)]
- 447-52D: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- 447-52D: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding – the dissociation constant, Kd of 447-52D for the cell associated primary and TCLA Envs was equal, 3nM [York (2001)]
- 447-52D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 µg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 447-52D: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – 447-D recognized the gp120 monomer much more readily than the o-gp140, suggesting the V3 loop is less exposed on o-gp140 as it is on the intact virions [Srivastava (2002)]

649	C8	gp160(727–732)	gp41(727–732 BH10)	PDRPEG	no	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

References: [Pincus & McClure(1993), Pincus (1993), Abacioglu (1994), McLain (2001)]

- C8: Immunotoxin of C8 coupled to ricin-A does not mediate cells killing, and is not affected by sCD4 [Pincus & McClure(1993)]
- C8: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – C8 was used as a control – the dominant response among vaccinees was to this mid-gp41 region, but not among the infected lab workers – Abs binding this region do not neutralize, bind to infected cells, nor serve as immunotoxins [Pincus (1993)]
- C8: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]
- C8: The substitution R725G (P[R→G]GPD RPEGIEEGGERDRDRS) alters the antigenic exposure of this region on the virion resulting in the loss of the downstream neutralizing epitope ERDRD, increased exposure of the epitope GPD RPEG in the virion, while the epitope IE EE remains unchanged [McLain (2001)]

650	B31	gp160(727–734)	gp41(727–734 BH10)	PDRPEGIE		Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

<p>References: [Abacioglu (1994)]</p> <ul style="list-style-type: none"> • B31: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)] 							
651	B33	gp160(727–734)	gp41(727–734 BH10)	PDRPEGIE	no	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> NL43 <i>HIV component:</i> gp160</p> <p>References: [Abacioglu (1994), Bristow (1994)]</p> <ul style="list-style-type: none"> • B33: There are two MAbs in the literature named B33, see also gp120, positions 123–142 – MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys [Bristow (1994)] • B33: Epitope boundaries mapped by peptide scanning IgG1 [Abacioglu (1994)] 							
652	1576	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Vella (1993)]</p> <ul style="list-style-type: none"> • 1576: Not neutralizing [Vella (1993)] 							
653	1578	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Evans (1989), Vella (1993)]</p> <ul style="list-style-type: none"> • 1578: No neutralizing activity – epitope may be formed by regions from both poliovirus and HIV [Evans (1989)] • 1578: Core epitope: IEEE – in this study, neutralized IIIB, but not RF or MN [Vella (1993)] 							
654	1579	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Vella (1993)]</p> <ul style="list-style-type: none"> • 1579: Core epitope: IEEE – neutralized IIIB, but not RF or MN [Vella (1993)] 							
655	1583	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Evans (1989), Vella (1993), Sattentau (1995)]</p> <ul style="list-style-type: none"> • 1583: Neutralizing activity, less broad than 1577 [Evans (1989)] • 1583: Core epitope: ERDRD – Could neutralize HIV IIIB but not HIV RF [Vella (1993)] • 1583: Cytoplasmic domain, epitope not exposed at the surface of HIV-1 infected cells [Sattentau (1995)] 							
656	1899	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Vella (1993)]</p> <ul style="list-style-type: none"> • 1899: Could neutralize HIV IIIB and HIV RF [Vella (1993)] 							

Table of HIV MAbs

657	1907	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Vella (1993)]</p> <ul style="list-style-type: none"> • 1907: Could not neutralize HIV IIIB, RF or MN [Vella (1993)] 							
658	1908	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Evans (1989), Vella (1993), Sattentau (1995)]</p> <ul style="list-style-type: none"> • 1908: Neutralized IIIB, but not RF or MN [Vella (1993)] • 1908: Cytoplasmic domain, epitope not exposed at the surface of HIV-1 infected cells [Sattentau (1995)] 							
659	1909	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> poliovirus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Vella (1993)]</p> <ul style="list-style-type: none"> • 1909: Neutralized HIV IIIB but not HIV RF [Vella (1993)] 							
660	41–1	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine(IgMκ)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Mani (1994), Dalgleish (1988)]</p> <ul style="list-style-type: none"> • 41–1: This antibody gp41(735–752 IIIB) [Dalgleish (1988)] seems to have been named the same as a different MAb to gp41(584–609) [Mani (1994)] • 41–1: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish (1988)] 							
661	41–2	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine(IgMκ)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Dalgleish (1988)]</p> <ul style="list-style-type: none"> • 41–2: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish (1988)] 							
662	41–3	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine(IgMκ)
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide</p> <p>References: [Dalgleish (1988)]</p> <ul style="list-style-type: none"> • 41–3: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish (1988)] 							
663	ED6	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no		murine(IgM)
<p>References: [Evans (1989)]</p>							

Table of HIV MAbs

664	LA9 (121–134)	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no		murine(IgM)
References: [Evans (1989)]							
665	1575	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGGERDRDRS	no	Vaccine	murine()
Vaccine: <i>Vector/type:</i> poliovirus <i>Strain:</i> IIIB <i>HIV component:</i> gp41 peptide							
Ab type: C-term Donor: C. Vella, NIBSC, Potters Bar UK							
References: [Evans (1989), Vella (1993), Buratti (1997), Cleveland (2000a)]							
<ul style="list-style-type: none"> • 1575: Neutralizing activity, less broad than 1577 [Evans (1989)] • 1575: Core epitope: IEEE – neutralized IIIB, but not RF or MN [Vella (1993)] • 1575: Study shows that MAb 1575 can recognize the IEEE sequence in both gp41, and in the HPG30 region of the p17 protein – motif is conserved in both regions in different HIV-1 clades [Buratti (1997)] • 1575: Ab binding to IEEE suppresses neutralizing Ab binding to adjacent epitope ERDRD [Cleveland (2000a)] 							
666	88–158/02	gp160(732–747)	gp41(732–752 IIIB)	GIEEEGGGERDRDRSIR		Vaccine	murine(IgG2b)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> gp41							
References: [Niedrig (1992a)]							
<ul style="list-style-type: none"> • 88–158/02: Mild inhibition of <i>in vitro</i> activity at high MAb concentrations – profound enhancing activity at low concentrations – significant reactivity to virion – domain non-immunogenic in humans [Niedrig (1992a)] 							
667	88–158/022	gp160(732–747)	gp41(732–752 IIIB)	GIEEEGGGERDRDRSIR		Vaccine	murine(IgG2b)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> gp41							
References: [Niedrig (1992a)]							
<ul style="list-style-type: none"> • 88–158/022: Mild inhibition of <i>in vitro</i> activity at high MAb concentrations – profound enhancing activity at low concentrations – significant reactivity to virion – domain non-immunogenic in humans [Niedrig (1992a)] 							
668	88–158/079	gp160(732–747)	gp41(732–752 IIIB)	GIEEEGGGERDRDRSIR		Vaccine	murine(IgG1)
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> gp41							
References: [Niedrig (1992a)]							
<ul style="list-style-type: none"> • 88–158/079: Mild inhibition of HIV <i>in vitro</i> at high MAb concentrations – profound enhancing activity at low concentrations – weak binding to virion – domain non-immunogenic in humans [Niedrig (1992a)] 							
669	polyclonal	gp160(dis 733–736)	gp41(dis 735–752 IIIB)	IEEE	L	Vaccine	murine(IgG)
Vaccine: <i>Vector/type:</i> Cowpea mosaic virus <i>HIV component:</i> gp41 peptide							
Ab type: C-term References: [Cleveland (2000b), McLain (2001)]							
<ul style="list-style-type: none"> • When PRGPDRPEGIEEEGGGERDRDRS was used as antigen an immunodominant, non-neutralizing response to IEEE was observed, but immunization GERDRDR shifts the response to ERDRD [Cleveland (2000b)] 							

Table of HIV MAbs

- The substitution 725 RG (P[R->G]GPDRPEGIEEEGGGERDRDRS) alters the antigenic exposure of this region on the virion resulting in the loss of the downstream neutralizing epitope ERDRD, increased exposure of the epitope GPDRPEG in the virion, while the epitope IEEE remains unchanged [McLain (2001)]

670	polyclonal	gp160(dis 733–736)	gp41(dis 735–752 NL43)	IEEE	L	Vaccine	murine(IgG)
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Vaccine: *Vector/type:* Cowpea mosaic virus *HIV component:* gp41 peptide

Ab type: C-term **References:** [McLain (2001)]

- The substitution 725 RG (P[R->G]GPDRPEGIEEEGGGERDRDRS) alters the antigenic exposure of this region on the virion resulting in the loss of the downstream neutralizing epitope ERDRD, increased exposure of the epitope GPDRPEG in the virion, while the epitope IEEE remains unchanged [McLain (2001)]

671	B8	gp160(733–741)	gp41(733–741 BH10)	IEEEGGGERD	no	Vaccine	murine(IgG1)
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Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

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

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

672	1577	gp160(739–743)	gp41(735–752 IIIB)	ERDRD	no	Vaccine	murine()
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Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

Ab type: C-term **Donor:** C. Vella or Morag Ferguson (NIBSC, Potters Bar UK)

References: [Evans (1989), D’Souza (1991), Vella (1993), Cleveland (2000a)]

- 1577: Raised against IIIB peptide chimera – neutralized African and American HIV-1 lab strains [Evans (1989)]
- 1577: Non-neutralizing in this multi-lab study [D’Souza (1991)]
- 1577: Core epitope: ERDRD – could neutralize HIV IIIB and HIV RF [Vella (1993)]
- 1577: Ab binding to IEEE suppresses neutralizing Ab binding to adjacent epitope ERDRD [Cleveland (2000a)]
- 1577: UK Medical Research Council AIDS reagent: ARP317
- 1577: NIH AIDS Research and Reference Reagent Program: 1172

673	polyclonal	gp160(dis 739–743)	gp41(dis 735–752 IIIB)	ERDRD	L	Vaccine	murine(IgG)
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Vaccine: *Vector/type:* Cowpea mosaic virus *HIV component:* gp41 peptide

Ab type: C-term **References:** [Cleveland (2000b), McLain (2001)]

- ERDRD-specific IgG recognizes an externalized loop of the gp41 C-terminal tail with high affinity – neutralized HIV-1 B clade strains IIIIB, NL-4.3, RF, MN and D clade virus CBL-4, but HXB-2D (clade B) was not recognized – when PRGPDRPEGIEEEGGGERDRDRS was used as antigen an immunodominant, non-neutralizing response to IEEE was observed, but immunization GERDRDR shifts the response to ERDRD – NAb does not inhibit attachment of free virus, but does inhibit an event that precedes fusion-entry [Cleveland (2000b)]
- The substitution 725 RG (P[R->G]GPDRPEGIEEEGGGERDRDRS) alters the antigenic exposure of this region on the virion resulting in the loss of the downstream neutralizing epitope ERDRD, increased exposure of the epitope GPDRPEG in the virion, while the epitope IEEE remains unchanged [McLain (2001)]

674 DZ	gp160(822–855)	gp41(827–860 BRU)	VAEGTDRVIEVVQGACRAIRH- IPRRIRQGLERIL	L	Vaccine	human(IgG1 λ)
	Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIIB <i>HIV component:</i> gp60					
	References: [Boyer (1991)]					
	<ul style="list-style-type: none"> • DZ: Weakly neutralizing IIIIB – binds to peptides 827–843 and 846–860 of BRU – reacted specifically with IIIIB and RF [Boyer (1991)] 					
675 IVI-4G6	gp160()	gp41()			Vaccine	murine(IgG2b)
	Donor: K. Miyakoshi (Feji-Rebio Co, Tokyo, Japan)					
	References: [Yin (2001)]					
	<ul style="list-style-type: none"> • IVI-4G6: A bi-specific Ab (BFA) was made by combining Fab fragments of gp41-specific MAb IVI-4G6 and CD3-specific Mab UCHT1 – the BFA suppressed HIV-1 propagation culture and eliminated latently infected cells [Yin (2001)] 					
676 polyclonal	gp160()	gp120()		no	Vaccine	mouse()
	Vaccine: <i>Vector/type:</i> recombinant protein, virus-like particle <i>Strain:</i> LAI <i>HIV component:</i> V3, CD4BS, p55					
	Ab type: CD4BS References: [Truong (1996)]					
	<ul style="list-style-type: none"> • Antibodies raised against recombinant anti-p55 virus-like particles with the p24 region 196–226 deleted, bearing inserts of either the V3 or the CD4BS regions of gp120 were studied – no neutralizing responses, weak Env and strong Gag responses were elicited – the major homology region (MHR) and proximal regions were found to be required for capsid assembly [Truong (1996)] 					
677 polyclonal	gp160()	gp120()		no	Vaccine	mouse()
	Vaccine: <i>Vector/type:</i> recombinant protein, virus-like particle <i>Strain:</i> LAI <i>HIV component:</i> V3, CD4BS, p55					
	Ab type: V3 References: [Truong (1996)]					
	<ul style="list-style-type: none"> • Antibodies raised against recombinant anti-p55 virus-like particles with the p24 region 196–226 deleted, bearing inserts of either the V3 or the CD4BS regions of gp120 were studied – no neutralizing responses, weak Env, and strong Gag responses were elicited – the major homology region (MHR) and proximal regions were found to be required for capsid assembly [Truong (1996)] 					

Table of HIV MAbs

678	polyclonal	gp160()	gp140(SF162)	KSITIGPGRAFYATGD	yes	Vaccine	rabbit, Rhesus macaque(IgG)
Vaccine:		<i>Vector/type:</i> DNA, CMV promotor elements		<i>Strain:</i> SF162, SF162ΔV2	<i>HIV component:</i> gp140		<i>Stimula-</i>
		<i>tory Agents:</i> MF-59C					
		Ab type: V3		References: [Barnett (2001)]			
		<ul style="list-style-type: none"> • SF162ΔV2 is a virus that has a 30 amino acid deletion in the V2 loop that does not abrogate its infectivity but renders it highly susceptible to neutralization – when incorporated into a codon-optimized DNA vaccine with a CMV promoter, delivered by gene gun, SF162ΔV2 gave higher neutralizing Ab titers against SF162 than did SF162 itself, and Abs that cross-neutralized non-homologous primary isolates were obtained only when SF162ΔV2, but not intact SF162, was used as the immunogen – NAbs titers specific for SF162 increased with multiple immunizations, while titers for non-homologous isolates decreased, but anti-V3 peptide binding Abs were not likely the source of this distinction because anti-V3 titers were much lower than those against the entire envelope, and the second booster immunization did not increase the titer of anti-V3 loop Abs [Barnett (2001)] 					

Table 13: **Env**

MAB ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
679 101-342	Env()	gp120(476-505 HAM112, O group)			Vaccine	murine(IgG2a κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HAM112 (group O) <i>HIV component:</i> gp160 Ab type: C-term References: [Scheffel (1999)] <ul style="list-style-type: none"> • 101-342: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity [Scheffel (1999)] </p>						
680 101-451	Env()	gp120(498-527 HAM112, O group)			Vaccine	murine(IgG2b κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HAM112 (group O) <i>HIV component:</i> gp160 Ab type: C-term References: [Scheffel (1999)] <ul style="list-style-type: none"> • 101-451: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity [Scheffel (1999)] </p>						
681 120-1	Env()	gp120(503-532)		no	Vaccine	murine(IgM κ)
<p>Vaccine: <i>Vector/type:</i> peptide Ab type: C-term References: [Chanh (1986), Dalgleish (1988)]</p>						
682 23A (2.3A)	Env(dis)	gp120(dis)		no		()
<p>Ab type: C-term Donor: J. Robinson, Tulane University, LA References: [Thali (1992a), Thali (1993), Wu (1996), Trkola (1996a), Fouts (1997), Binley (1999)] <ul style="list-style-type: none"> • 23A: Called 2.3A – Did not block ability of gp120-sCD4 complexes to inhibit MIP-1α binding – binds to gp41-binding domain of gp120 [Wu (1996)] • 23A: C5 binding MAb – does not inhibit gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)] • 23A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – 23A bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)] • 23A: 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 NAb 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 [Binley (1999)] </p>						

Table of HIV MAbs

683	D7324	Env()	gp120()		Vaccine	sheep()
<p>Vaccine: <i>HIV component:</i> gp120</p> <p>Ab type: C-term Donor: Aalto BioReagents Ltd, Dublin, Ireland</p> <p>References: [Moore(1990), Sattentau & Moore(1991), Moore (1993a), Moore (1993b), Wyatt (1995), Trkola (1996a), Ditzel (1997), Ugolini (1997), Mondor (1998), Binley (1998)]</p> <ul style="list-style-type: none"> • D7324: Binding unaltered by gp120 binding to sCD4, in contrast to 110.5, 9284, 50–69 and 98–6 [Sattentau & Moore(1991)] • D7324: Binds to the last 15 amino acids in gp120 – used for antigen capture ELISA [Wyatt (1995)] • D7324: Epitope in C5 – Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)] • D7324: Used to capture gp120 onto solid phase for epitope mapping [Moore (1993a)] • D7324: Used to capture gp120 onto solid phase for epitope mapping [Moore (1993b)] • D7324: Used to capture gp120 onto solid phase for epitope mapping [Ditzel (1997)] • D7324: Used to capture gp120 onto solid phase for epitope mapping [Binley (1998)] 						
684	212A	Env(dis)	gp120(dis)	no	HIV-1 infection	human()
<p>Ab type: C1 Donor: J. Robinson, Tulane University, LA</p> <p>References: [Robinson (1992), Moore (1994d), Moore & Sodroski(1996), Binley (1997a), Fouts (1997), Ditzel (1997), Wyatt (1997), Parren (1997b), Sullivan (1998b), Binley (1998)]</p> <ul style="list-style-type: none"> • 212A: Mutations that inhibit binding: C1 (45 W/S) and V5 (463 N/D) – and enhance binding: V2 (179/180 LD/DL) and C5 (495 G/K) [Moore (1994d)] • 212A: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore & Sodroski(1996)] • 212A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 212A bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)] • 212A: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids are deleted [Wyatt (1997)] • 212A: Does not neutralize TCLA strains or primary isolates [Parren (1997b)] • 212A: Does not compete with binding of MAb CG10 generated in response to gp120-CD4 complex [Sullivan (1998b)] • 212A: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)] 						
685	522–149	Env(dis)	gp120(dis)	no	Vaccine	()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Env</p> <p>Ab type: C1 Donor: G. Robey, Abbott Inc.</p> <p>References: [Moore & Sodroski(1996), Trkola (1996a), Binley (1998)]</p> <ul style="list-style-type: none"> • 522–149: Binding is enhanced by C5 antibodies M91 and 1C1 – mutual binding-inhibition with anti-C1 antibody 133/290 – binding is destroyed by a W/L (position 61, LAI) gp120 amino acid substitution – other C1 antibodies enhance binding to gp120 [Moore & Sodroski(1996)] 						

- 522–149: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola (1996a)]
- 522–149: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

686	L19	Env(dis) Ab type: C1	gp120(dis HXBc2) References: [Ditzel (1997)]		HIV-1 infection	human Fab(IgG1)
<ul style="list-style-type: none"> • L19: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for the selection of Fabs – six N-term Fabs, L19 L34, L35, L52, L59, and L69, were obtained that have a similar epitope to Fab p7 [Ditzel (1997)] 						
687	M90	Env(dis) Vaccine: <i>Vector/type:</i> protein	gp120(dis) <i>HIV component:</i> Env	no	Vaccine	(IgG1)
<p>Ab type: C1 Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese (1992), DeVico (1995), Moore & Sodroski(1996), Ditzel (1997), Wyatt (1997), Binley (1998), Binley (1999)]</p> <ul style="list-style-type: none"> • M90: Reactive only with native gp120, so binds to a discontinuous epitope – reacts with multiple strains [di Marzo Veronese (1992)] • M90: Reacted with both non-reduced (but not denatured) covalently cross-linked gp120-CD4 complex [DeVico (1995)] • M90: Reciprocal inhibition of binding of other anti-C1 MAbs – inhibits CD4 binding site MAbs – enhances binding of V2 MAbs G3-4 and SC258 [Moore & Sodroski(1996)] • M90: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31–82, are deleted [Wyatt (1997)] • M90: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)] • M90: 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 [Binley (1999)] 						
688	MAG 104	Env(dis) Vaccine: <i>Vector/type:</i> sCD4-gp120 complex	gp120(dis) <i>Strain:</i> HXB2 <i>HIV component:</i> gp120	no	Vaccine	murine()
<p>Ab type: C1 Donor: C. Y. Kang, IDEC Inc References: [Kang (1994)]</p>						

Table of HIV MAbs

- MAG 104: Only observed amino acid substitutions that reduce binding: 88 N/P and 106 E/A – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang (1994)]

689	MAG 45 (#45)	Env(dis)	gp120(dis)	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120</p> <p>Ab type: C1 Donor: C. Y. Kang, IDEC Inc</p> <p>References: [Kang (1994), Moore & Sodroski(1996), Wyatt (1997)]</p> <ul style="list-style-type: none"> • MAG 45: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang (1994)] • MAG 45: Reciprocal binding inhibition with anti-C1-C5 and anti-C1-C4 discontinuous MAbs – binding enhanced by anti-V3 5G11 – inhibits binding of anti-CD4 binding site MAbs [Moore & Sodroski(1996)] • MAG 45: Called #45 – binds to efficiently sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31–50, are deleted [Wyatt (1997)] 						
690	MAG 95	Env(dis)	gp120(dis)	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120</p> <p>Ab type: C1 Donor: C. Y. Kang, IDEC Inc</p> <p>References: [Kang (1994)]</p> <ul style="list-style-type: none"> • MAG 95: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang (1994)] 						
691	MAG 97	Env(dis)	gp120(dis)	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120</p> <p>Ab type: C1 Donor: C. Y. Kang, IDEC Inc</p> <p>References: [Kang (1994)]</p> <ul style="list-style-type: none"> • MAG 97: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang (1994)] 						
692	p7	Env(dis)	gp120(dis HXBc2)	no	HIV-1 infection	human Fab(IgG1)
<p>Ab type: C1 References: [Ditzel (1997), Parren (1997b)]</p> <ul style="list-style-type: none"> • p7: gp120 immobilized on solid phase by capture with sCD4 was used for selection of Fabs – three novel N-term Fabs were obtained that bind to similar epitopes, p7, p20, and p35 – a C1 W/S substitution at position 45 abolished binding, a Y/D at position 45 reduced binding, and C5 region substitutions 475 M/S and 493 P/K enhanced binding – compete with MAbs M85, M90 and 212A, but not M91 and G3-299 [Ditzel (1997)] • p7: Does not neutralize TCLA strains or primary isolates [Parren (1997b)] 						

693	L100	Env(dis) Ab type: C1-C2	gp120(dis HXBc2) References: [Ditzel (1997), Parren (1997b), Parren & Burton(1997)]	HIV-1 infection	human Fab(IgG1)
<ul style="list-style-type: none"> • L100: Does not neutralize TCLA strains or primary isolates [Parren (1997b)] • L100: gp120 immobilized on solid phase by capture with sCD4 and then masked with Fab p7 allowed selection of a new Fab, L100, with a novel specificity for C1 and C2 – gp120 C1 substitutions 69 W/L and 76 P/Y abolish L100 binding, and C2 substitutions 252 R/W, 256 S/Y, 262 N/T and 267 E/L abolish or strongly inhibit L100 binding – inhibits binding of MAbs M90 and G3-299, but not M85, 212A, and M91 [Ditzel (1997), Parren & Burton(1997)] 					
694	2/11c (211c, 2.11c, 211/c, 2–11c)	Env(dis)	gp120(dis)	L (weak) HIV-1 infection	human()
<p>Ab type: C1-C4 Donor: J. Robinson, Tulane University, LA References: [Moore & Sodroski(1996), Trkola (1996a), Binley (1997a), Fouts (1997), Li (1997), Wyatt (1997), Binley (1998)]</p> <ul style="list-style-type: none"> • 2/11c: Inhibits binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of some anti-V2 and CD4i MAbs (48d and 17b) – similar reactivity pattern to A32, but less cross-reactive and lower affinity – A32 and 211/c are unique among known human and rodent MAbs [Moore & Sodroski(1996)] • 2/11c: Called 211c – does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)] • 2/11c: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – 2/11c bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)] • 2/11c: Called 2.11c – One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 67 μg/ml [Li (1997)] • 2/11c: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31–74, are deleted [Wyatt (1997)] • 2/11c: Called 211/c – a panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)] 					
695	C11 (c11)	Env(dis)	gp120(dis)	no HIV-1 infection	human()
<p>Ab type: C1-C5 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)]</p> <ul style="list-style-type: none"> • 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) – and ΔV1/V2/V3 [Moore (1994d)] • C11: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore & Sodroski(1996)] • C11: Did not block ability of gp120-sCD4 complexes to inhibit MIP-1α binding – binds to gp41-binding domain [Wu (1996)] • C11: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)] 					

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- C11: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – C11 bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)]
- C11: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – partial reexposure if sCD4 was bound – does not bind to HXBc2 gp120 if the 19 C-term amino acids are deleted [Wyatt (1997)]
- C11: Does not neutralize TCLA strains or primary isolates [Parren (1997b)]
- C11: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 [Sullivan (1998b)]
- C11: 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 [Binley (1999)]

696	L81	Env(dis) Ab type: C1-C5	gp120(dis) References: [Ditzel (1997), Parren (1997b)]	no	HIV-1 infection	human(IgG1)
<ul style="list-style-type: none"> • L81: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – L81 binding is abolished by C1 substitution 45 W/S, C5 substitution 491 I/F, and C3 substitution L/A [Ditzel (1997)] • L81: Does not neutralize TCLA strains or primary isolates [Parren (1997b)] 						
697	2F19C	Env() Vaccine: <i>Vector/type:</i> peptide	gp120(HIV2ROD) APGK <i>Strain:</i> HIV-2 ROD	no	Vaccine	murine()
<ul style="list-style-type: none"> • 2F19C: Binds in WB, but binds poorly to Env on the cell surface, APGK is the core binding region [Matsushita (1995)] 						
698	B2C	Env() Vaccine: <i>Vector/type:</i> peptide	gp120(HIV2ROD) HYQ(core) <i>Strain:</i> HIV-2 ROD	L	Vaccine	murine()
<ul style="list-style-type: none"> • B2C: Viral neutralization was type-specific for HIV-2 ROD [Matsushita (1995)] 						
699	1024	Env() Ab type: C4	gp120() References: [Berman (1997)]			()
<ul style="list-style-type: none"> • 1024: Binds to 1/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)] 						
700	10/46c	Env(dis) Vaccine: <i>Vector/type:</i> recombinant protein	gp120(dis) <i>HIV component:</i> gp120		Vaccine	rat()
<ul style="list-style-type: none"> • Ab type: CD4BS References: [Cordell (1991), Jeffs (1996), Peet (1998)] 						

- 10/46c: Increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs (1996)]
- 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 wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]

701	1027–30-D	Env(dis)	Env(dis)			human(IgG1 κ)
		Ab type: CD4BS Donor: Susan Zolla-Pazner (Zollas01@mrcr6.med.nyu) (NYU Med. Center) References: [Hioe (2000)]				
		<ul style="list-style-type: none"> • 1027–30-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – anti-CD4 binding site MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – CD4BS MAbs 654-D, 559/64-D, 588-D, 830-D, 1027–30-D, and 1202–30D strongly diminished proliferation [Hioe (2000)] 				
702	1125H (1125h)	Env(dis)	gp120(dis)	L (MN)	HIV-1 infection	human(IgG1 κ)
		Ab type: CD4BS Donor: Shermaine Tilley, Public Health Research Institute, USA References: [Tilley (1991b), Tilley (1991a), Thali (1992a), Wyatt (1992), Pinter (1993b), D’Souza (1995), Warriar (1996), Pincus (1996), Wyatt (1998), Alsmadi & Tilley(1998), Yang (1998)]				
		<ul style="list-style-type: none"> • 1125H: Binding to gp120 inhibited by CD4 – epitope is destroyed by reduction, but not by removal of N-linked sugars – potent neutralization of MN, RF, SF-2 and IIIB – neutralization synergy with anti-V3 MAb 4117C [Tilley (1991a)] • 1125H: Amino acid substitutions in HXB2 that strongly inhibit binding: 88, 102, 117, 113, 257, 368, 370, 421, 427, 457, 470, 480 [Thali (1992a)] • 1125H: Binding to soluble gp120 enhanced by the presence of an anti-V3 HuMAb, 41148D [Pinter (1993b)] • 1125H: Precipitation of Δ 297–329 env glycoprotein, with has a deleted V3 loop, is much more efficient that precipitation of wild type [Wyatt (1992)] • 1125H: Neutralization was MN specific – failed to neutralize JRCSF, and 2 B subtype and 1 D subtype primary isolates in a multi-laboratory study involving 11 labs [D’Souza (1995)] • 1125H: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warriar (1996)] • 1125H: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)] • 1125H: Called 1125h – summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt (1998)] • 1125H: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against all four strains [Alsmadi & Tilley(1998)] • 1125H: A neutralization assay was developed based on hemi-nested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang (1998)] 				

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703	120-1B1	Env(dis) Ab type: CD4BS	gp120(dis) Donor: Virus Testing Systems Corp., Houston, TX	L	human()
<p>References: [Watkins (1993)]</p> <ul style="list-style-type: none"> • 120-1B1: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – 120-1B1 was not affected by this mutation [Watkins (1993)] 					
704	1202-D (1202-30-D)	Env(dis)	Env(dis)		human(IgG1κ)
<p>Ab type: CD4BS Donor: Susan Zolla-Pazner (Zollas01@mcr6.med.nyu) (NYU Med. Center)</p> <p>References: [Nyambi (1998), Hioe (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 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 [Nyambi (1998)] • 1202-D: Called 1202-30D – Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – anti-CD4 binding site MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – CD4BS MAbs 654-D, 559/64-D, 588-D, 830-D, 1027-30-D, and 1202-30D strongly diminished proliferation [Hioe (2000)] • 1202-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 CD4BS MAbs – CD4BS MAbs bound consistently to most isolates of clade D, but poorly to isolates of other clades with the exception of broadly reactive MAb IgG1b12[Nyambi (2000)] 					
705	1331E	Env(dis)	gp120(dis IIIB)	HIV-1 infection	human(IgG1κ)
<p>Ab type: CD4BS Donor: Susan Zolla-Pazner (Zollas01@mcr6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny (2000)]</p> <ul style="list-style-type: none"> • 1331E: Inhibits sCD4 binding to rec gp120 LAI – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – CD4BS MAbs 559/64-D, 654-D, 729-D, 9CL and 1331E bound with a 5-13 fold preference for the oligomer [Gorny (2000)] 					
706	1570 (1570A, 1570C, and 1570D)	Env(dis)	Env(dis PR12, BH10 core)	HIV-1 infection	human()
<p>Ab type: CD4BS References: [Jeffs (2001)]</p> <ul style="list-style-type: none"> • 1570: BH10 was mutated to form the PR12 protein with the first 74 C-terminal amino acids and the V1, V2 and V3 hypervariable loops deleted and a very well exposed CD4 binding domain (CD4bd) – this proteins was used to select three new human CD4BS MAbs 1570, 1595 and 1599 – three MAbs were isolated from one individual, 1570A, C and D but all were determined to have the same V(H)3 region – 1570 was able to bind to a panel of recombinant proteins from the A, B, C, D, and E subtypes [Jeffs (2001)] 					

707	1595	Env(dis)	Env(dis PR12, BH10 core)		HIV-1 infection	human()
<p>Ab type: CD4BS References: [Jeffer (2001)]</p> <ul style="list-style-type: none"> • 1595: BH10 was mutated to form the PR12 protein with the first 74 C-terminal amino acids and the V1, V2 and V3 hypervariable loops deleted and a very well exposed CD4 binding domain (CD4bd) – this proteins was used to select three new human CD4BS MAbs 1570, 1595 and 1599 – 1595 was able to bind gp120 from the A, B, and D clades from a panel of recombinant proteins from the A, B, C, D, and E subtypes [Jeffer (2001)] 						
708	1599	Env(dis)	Env(dis PR12, BH10 core)		HIV-1 infection	human()
<p>Ab type: CD4BS References: [Jeffer (2001)]</p> <ul style="list-style-type: none"> • 1599: BH10 was mutated to form the PR12 protein with the first 74 C-terminal amino acids and the V1, V2 and V3 hypervariable loops deleted and a very well exposed CD4 binding domain (CD4bd) – this proteins was used to select three new human CD4BS MAbs 1570, 1595 and 1599 – 1599 was able to bind gp120 only from the B clade from a panel of recombinant proteins from the A, B, C, D, and E subtypes [Jeffer (2001)] 						
709	15e (1.5e, 1.5E, 15E)	Env(dis)	gp120(dis)	L	HIV-1 infection	human(IgG1κ)
<p>Ab type: CD4BS Donor: J. Robinson, Tulane University, LA, and David Ho, ADARC, NY, NY</p> <p>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), Pognard (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), Park (2000), Kolchinsky (2001)]</p> <ul style="list-style-type: none"> • 15e: Broadly neutralizing, binds multiple strains, competes with CD4 for gp120 binding, DTT reduction of env abrogates binding – more potent blocking of gp120-sCD4 binding than MAbs G3-536 and G3-537 [Ho (1991b)] • 15e: Cross-competes with MAbs ICR 39.13g and ICR 39.3b [Cordell (1991)] • 15e: Binds to gp120 of HIV-1 IIIB, but not RF – mediates ADCC – deletion of the V3 loop from gp120 does not alter ADCC activity [Koup (1991)] • 15e: gp120 mutants that affect 15e epitope binding: 113, 257, 368, 370, 421, 427, 475 – four of these coincide with amino acids important for the CD4 binding domain [Ho (1992)] • 15e: Precipitation of Δ 297–329 env glycoprotein, with a deleted V3 loop, is much more efficient that precipitation of wild type [Wyatt (1992)] • 15e: Amino acid substitutions in HXB2 that strongly inhibit binding, similar to [Ho (1992)], some additional, 88, 102, 117, 113, 257, 368, 370, 421, 427, 457, 470, 480 [Thali (1992a)] • 15e: Called N70–1.5e – does not enhance infection of HIV-1 IIIB and MN [Thali (1992a)] • 15e: Conformational, does not bind denatured gp120 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)] 						

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- 15e: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is greater than binding to wildtype gp120 [Wyatt (1993)]
- 15e: Called 15E – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – 15E neutralization was not affected by this mutation [Watkins (1993)]
- 15e: Heavy chain is V HIV, V2-1 – light chain is V_kappaI, Hum01/012. Compared to 21h and F105 [Bagley (1994)]
- 15e: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 21h and 17b) [Thali (1994)]
- 15e: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – anti-CD4 MAbs moderately inhibit gp120 binding to GalCer, possibly through steric hindrance – binding of GalCer to gp120 inhibited but did not completely block 15e binding [Cook (1994)]
- 15e: Cross-reactive with gp120 proteins from clades B and D, less so with A and C, and not reactive with clade E and F [Moore (1994b)]
- 15e: Binds with higher affinity to monomer than to oligomer, moderate association rate [Sattentau & Moore(1995)]
- 15e: The V4 and V5 domains are essential for 1.5e binding, in contrast to the V1, V2, and V3 loops [Lee (1995)]
- 15e: Called 1.5e – Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]
- 15e: gp120 binding enhanced by anti-V3 MAb 5G11 and anti-V2 MAb G3-136 – binding inhibited by other CD4 binding site MAbs, antibodies that bind to gp120 only when CD4 is bound, and CD4-IgG [Moore & Sodroski(1996)]
- 15e: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50–69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Poignard (1996a)]
- 15e: Inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola (1996a)]
- 15e: Neutralizes HIV-1 LAI less potently than V3 specific MAbs [McDougal (1996)]
- 15e: 15e is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisniewski (1996)]
- 15e: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 15e bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)]
- 15e: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 15e could only achieve 50% neutralization, but could act synergistically with anti-V3 MAb 694/98-D to achieve 90% [Li (1997)]
- 15e: Does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31–93, are deleted [Wyatt (1997)]
- 15e: Called 1.5E – Binds to 7/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)]
- 15e: Neutralizes TCLA strains, but not primary isolates [Parren (1997b)]
- 15e: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt (1998)]
- 15e: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

- 15e: A panel of MABs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MABs 15e, F91 and IgG1b12 bound better to the deleted protein than to wild type [Binley (1998)]
- 15e: Competes with CG-10 binding, a MAB raised against a gp120 CD4 complex, this was probably due to the disruption of CD4-gp120 by 15e [Sullivan (1998b)]
- 15e: No detectable neutralizing activity among primary isolates with different co-receptor usage – some neutralization of TCLA strains [Trkola (1998)]
- 15e: CD4BS MABs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer [Fouts (1998)]
- 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 μ g/ml [Sullivan (1998a)]
- 15e: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MABs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MABs against gp120 by causing conformational changes [Park (2000)]
- 15e: Mutations in two glycosylation sites in the V2 region of HIV-1 ADA at positions 190 and 197 (187 DNTSYRLINCNTS 199) cause the virus to become CD4-independent and able to enter cells through CCR5 alone – these same mutations tended to increase the neutralization sensitivity of the virus, including to 15e [Kolchinsky (2001)]
- 15e: UK Medical Research Council AIDS reagent: ARP3016

710	205-43-1	Env(dis)	gp120(dis)	no	HIV-1 infection	human()
		Ab type: CD4BS References: [Fouts (1998), Grovit-Ferbas (2000)]				
		<ul style="list-style-type: none"> • 205-43-1: Rank order of CD4BS antibodies oligomer binding is IgG1b12 = 2G6 = 205-46-9 > 205-43-1 = 205-42-15 > 15e = 21h = F91, and the only thing notably distinguishing about neutralizing IgG1b12 is that it depends on residues in V2 [Fouts (1998)] • 205-43-1: To determine the antigenicity of virus killed by thermal and chemical inactivation, retention of conformation-dependent neutralization epitopes was examined, and exposure of CD4BS epitopes was found to be enhanced (MABs IgG1b12, 205-46-9, and 205-43-1) – binding to 2G12 and 447-52D epitopes was essentially unaltered – the 17b CD4i epitope was also exposed [Grovit-Ferbas (2000)] 				
711	205-46-9	Env(dis)	gp120(dis)	no	HIV-1 infection	human()
		Ab type: CD4BS References: [Fouts (1998), Grovit-Ferbas (2000)]				
		<ul style="list-style-type: none"> • 205-46-9: Binds JRSF oligomer with high affinity as does IgG1b12, but IgG1b12 is neutralizing, 205-46-9 is 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 [Fouts (1998)] 				

Table of HIV MAbs

- 205-46-9: To determine the antigenicity of virus killed by thermal and chemical inactivation, retention of conformation-dependent neutralization epitopes was examined, and exposure of CD4BS epitopes was found to be enhanced (MAbs IgG1b12, 205-46-9, and 205-43-1) – binding to 2G12 and 447-52D epitopes was essentially unaltered – the 17b CD4i epitope was also exposed [Grovit-Ferbas (2000)]

712	21h (2.1H)	Env(dis) gp120(dis)	L	HIV-1 infection	human(IgG1)
<p>Ab type: CD4BS Donor: J. Robinson, Tulane University, LA</p> <p>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), Pognard (1996a), Wisnewski (1996), McKeating (1996), Binley (1997a), Fouts (1997), Li (1997), Ugolini (1997), Wyatt (1997), Parren (1997b), Wyatt (1998), Parren (1998a), Fouts (1998)]</p> <ul style="list-style-type: none"> ● 21h: Amino acid substitutions in HXB2 that inhibit binding, some shared with CD4 binding inhibition, 88, 113, 257, 368, 370, 421, 470, 480 [Thali (1992a)] ● 21h: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is greater than binding to wildtype gp120 [Wyatt (1993)] ● 21h: Conformational, does not bind denatured gp120 – neutralizes IIIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIIB gp120 [Moore & Ho(1993)] ● 21h: Has strong cross-reactivity with gp120 monomers from most subtypes, A-F, with the least reactivity to clade E [Moore (1994b)] ● 21h: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore (1994a)] ● 21h: Heavy chain is V HIII, VDP-35 – light chain is V_λIIIa, Hum318. Compared to 15e and F105 [Bagley (1994)] ● 21h: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 15e and 17b) [Thali (1994)] ● 21h: Binds with higher affinity to monomer than to oligomer, moderate association rate [Sattentau & Moore(1995)] ● 21h: Anti-CD4 binding site MAb – reciprocal inhibition by anti-C1, -C4 and other anti-CD4 binding site antibodies – enhanced by some anti-V2 MAbs and anti-V3 MAb 5G11 – enhances binding of some anti-V3 and -V2 MAbs [Moore & Sodroski(1996)] ● 21h: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50–69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Pognard (1996a)] ● 21h: 21h is V H3 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)] ● 21h: Called 2.1H – Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)] ● 21h: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 21h bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)] ● 21h: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIIB env – 50% neutralization could not be achieved at a maximal concentration of 67 μg/ml [Li (1997)] ● 21h: Viral binding inhibition by 21h strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)] 					

- 21h: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding – major deletions in C1 and C5 and deletions of the V1V2 and V3 loops do not diminish binding [Wyatt (1997)]
- 21h: Neutralizes TCLA strains, but not primary isolates [Parren (1997b)]
- 21h: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt (1998)]
- 21h: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- 21h: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer – conclusions of this paper contrast with [Parren (1998a)] [Fouts (1998)]
- 21h: UK Medical Research Council AIDS reagent: ARP3017

713	2G6	Env(dis)	gp120(dis)			()
<p>Ab type: CD4BS Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, or Polymun Scientific Inc., Vienna, Austria</p> <p>References: [Fouts (1998)]</p> <ul style="list-style-type: none"> • 2G6: Binds to JRFL oligomer with an affinity comparable to IgG1b12, but does not neutralize the virus, so binding of oligomer is not always predictive of neutralization – conclusions of this paper contrast with [Parren (1998a)] – authors propose a model where 205-46-9 and 2G6 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts the neutralizing effect [Fouts (1998)] 						
714	428	Env(dis)	gp120(dis)		HIV-1 infection	human()
<p>Ab type: CD4BS References: [Karwowska (1992a), Jeffs (1996)]</p> <ul style="list-style-type: none"> • 428: Slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs (1996)] 						
715	448-D (448D)	Env(dis)	gp120(dis)	L	HIV-1 infection	human(IgG1λ)
<p>Ab type: CD4BS Donor: Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU Med Center, NY, NY</p> <p>References: [Karwowska (1992a), McKeating (1992c), Spear (1993), Laal (1994), Forthal (1995), Manca (1995), Li (1997), Wyatt (1998), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 448-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska (1992a)] • 448-D: Called 448D – blocks gp120-CD4 binding – substitutions at gp120 residues 88, 113, 117, 257, 368 and 370 reduce binding – epitope similar to rat MAbs 39.13g and 39.3b [McKeating (1992c)] • 448-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear (1993)] • 448-D: Dissociation constant gp120 IIIB 0.029 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D [Laal (1994)] • 448-D: Neutralizing activity, positive ADCC activity, and no viral enhancing activity [Forthal (1995)] • 448-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)] • 448-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env [Li (1997)] 						

Table of HIV MAbs

- 448-D: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt (1998)]
- 448-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 CD4BS MAbs – CD4BS MAbs bound consistently to most isolates of clade D, but poorly to isolates of other clades with the exception of broadly reactive MAb IgG1b12[Nyambi (2000)]

716 48–16	Env(dis) Ab type: CD4BS	gp120(dis) References: [Fevrier (1995)]	no	HIV-1 infection	human(IgG κ)
	<ul style="list-style-type: none"> ● 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 x 10⁻⁹ M [Fevrier (1995)] 				
717 50–61A	Env(dis) Ab type: CD4BS	gp120(dis) References: [Fevrier (1995)]	L	HIV-1 infection	human(IgG κ)
	<ul style="list-style-type: none"> ● 50–61A: Neutralizes lab strains LAI and SF2 – competes with sera from 45 seropositive subjects – binding affinity 2.4 x 10⁻¹⁰ M [Fevrier (1995)] 				
718 5145A	Env(dis) Ab type: CD4BS	gp120(dis) References: [Pinter (1993a), Warriar (1996), Pincus (1996), Alsmadi & Tilley(1998)]	L	HIV-1 infection	human(IgG1)
	<ul style="list-style-type: none"> ● 5145A: Potent and broadly cross-reactive neutralization of lab strains [Pinter (1993a)] ● 5145A: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warriar (1996)] ● 5145A: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)] ● 5145A: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against all four strains [Alsmadi & Tilley(1998)] 				
719 558-D	Env(dis) Ab type: CD4BS	gp120(dis) Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu), NYU Med Center, NY, NY References: [McKeating (1992c), Nyambi (1998)]	L	HIV-1 infection	human()
	<ul style="list-style-type: none"> ● 558-D: Blocks gp120-CD4 binding – binds a panel of mutants all except for 256 S/Y and 262 N/T, which are probably conformationally disruptive [McKeating (1992c)] ● 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 [Nyambi (1998)] 				

720 559/64-D Env(dis) gp120(dis LAI) L HIV-1 infection human(IgG1 κ)
(559 559–64D)

Ab type: CD4BS **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU Med Center, NY, NY

References: [Karwowska (1992a), McKeating (1992c), Spear (1993), Stamatatos & Cheng-Mayer(1995), Forthal (1995), Jeffs (1996), Hioe (1997), Nyambi (1998), Gorny (2000), Hioe (2000), Nyambi (2000), Hioe (2001), York (2001)]

- 559/64-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska (1992a)]
- 559/64-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear (1993)]
- 559/64-D: Called 559–64D – The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – CD4BS loop epitopes are somewhat occluded in the oligomeric gp120 epitopes on the virion surface relative to the gp120 monomer as indicated by an increase in the half-maximal binding values to macrophage-tropic isolates SF162 and SF128a and to T-cell tropic SF2 – binding of anti-CD4BS MAbs to SF2 resulted in a significant amount of dissociation of gp120 from virion surface [Stamatatos & Cheng-Mayer(1995)]
- 559/64-D: Neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 559/64-D: Called 559 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs (1996)]
- 559/64-D: Used in the development of resting cell neutralization assay [Hioe (1997)]
- 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 [Nyambi (1998)]
- 559/64-D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – CD4BS MAbs 559/64-D, 654-D, 729-D, 9CL and 1331E bound with a 5–13 fold preference for the oligomer [Gorny (2000)]
- 559/64-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – anti-CD4 binding site MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – CD4BS MAbs 654-D, 559/64-D, 588-D, 830-D, 1027–30-D, and 1202–30D strongly diminished proliferation [Hioe (2000)]
- 559/64-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 CD4BS MAbs – CD4BS MAbs bound consistently to most isolates of clade D, but poorly to isolates of other clades with the exception of broadly reactive MAb IgG1b12[Nyambi (2000)]
- 559/64-D: CD4BS MAbs when complexed with gp120, inhibit proliferation of gp120-specific CD4 T-cells and IFN- γ production – anti-CD4BS MAbs inhibit gp120 presentation by altering the uptake and/or processing of gp120 by the APCs, not by blocking of gp120 attachment to CD4 on the surface of APCs [Hioe (2001)]
- 559/64-D: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4 induced or CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAb alters some step after binding [York (2001)]

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721	588-D (588)	Env(dis) Ab type: CD4BS	gp120(dis) Donor: Susan Zolla-Pazner (Zollas01@mrcrcr6.med.nyu), NYU Med Center, NY, NY	L HIV-1 infection	human(IgG1 κ)
<p>References: [Karwowska (1992a), Buchbinder (1992), Moore & Ho(1993), Jeffs (1996), Nyambi (1998), Hioe (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 588-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska (1992a)] • 588-D: 4-fold increase in neutralization potency for 588-D when combined 1:1 with human MAb 447-D [Buchbinder (1992)] • 588-D: Weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)] • 588-D: Called 588 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs (1996)] • 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 [Nyambi (1998)] • 588-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – anti-CD4 binding site MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – CD4BS MAbs 654-D, 559/64-D, 588-D, 830-D, 1027–30-D, and 1202–30D strongly diminished proliferation [Hioe (2000)] • 588-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 CD4BS MAbs – CD4BS MAbs bound consistently to most isolates of clade D, but poorly to isolates of other clades with the exception of broadly reactive MAb IgG1b12[Nyambi (2000)] 					
722	654-D (654–30D, 654/30D, 654-D100, 654.30D)	Env(dis)	gp120(dis LAI)	L HIV-1 infection	human(IgG κ)
<p>Ab type: CD4BS Donor: Susan Zolla-Pazner (Zollas01@mrcrcr6.med.nyu), NYU Med Center, NY, NY</p> <p>References: [Karwowska (1993), Laal (1994), Gorny (1994), Stamatatos & Cheng-Mayer(1995), Li (1997), Stamatatos (1997), Gorny (1997), Gorny (1998), Schonning (1998), Nyambi (1998), Stamatatos & Cheng-Mayer(1998), Hioe (1999), Gorny (2000), Hioe (2000), Hioe (2001), Nyambi (2000), Verrier (2001)]</p> <ul style="list-style-type: none"> • 654-D: Dissociation constant gp120 IIIB 0.008 – neutralizes IIIB, acts synergistically with anti-V3 MAb 447-52D – reported to be human(IgG1λ) [Laal (1994)] • 654-D: Mild oxidation of carbohydrate moieties inhibits binding [Gorny (1994)] • 654-D: Called 654–30D – The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – CD4BS loop epitopes are somewhat occluded in the oligomeric gp120 epitopes on the virion surface relative to the gp120 monomer as indicated by an increase in the half-maximal binding values to macrophage-tropic isolates SF162 and SF128a and to T-cell tropic SF2 – binding of anti-CD4BS MAbs to SF2 resulted in a significant amount of dissociation of gp120 from virion surface [Stamatatos & Cheng-Mayer(1995)] • 654-D: Called 654–30D – One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env [Li (1997)] 					

- 654-D: Anti-CD4 BS MAb 654-30D and IgG1b12 have comparable binding affinities, neither mediates gp120-virion dissociation, but IgG1b12 can neutralize SF128A and SF162 and 654-D cannot – 654-D actually enhances infection by both viruses in primary macrophages [Stamatatos (1997)]
- 654-D: Called 654-D100 – 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 [Schonning (1998)]
- 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 [Nyambi (1998)]
- 654-D: Called 654.30D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V2 but not V1 slightly allowed neutralization by CD4BS MAb 654.30D [Stamatatos & Cheng-Mayer(1998)]
- 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 IgG1b12 – non-neutralizing anti-HIV CD4BS MAb 654-D did not become neutralizing in the presence of anti-LFA-1 MAbs [Hioe (1999)]
- 654-D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – CD4BS MAbs 559/64-D, 654-D, 729-D, 9CL and 1331E bound with a 5–13 fold preference for the oligomer [Gorny (2000)]
- 654-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – MAb 654-D strongly diminished proliferation – there is a discrepancy in isotyping this antibody, previous reports indicated IgG1kappa, while Hioe suggests it is IgG1lambda [Hioe (2000)]
- 654-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 CD4BS MAbs – CD4BS MAbs bound consistently to most isolates of clade D, but poorly to isolates of other clades with the exception of broadly reactive MAb IgG1b12 – 654-D had the weakest binding among CD4BS MAbs, binding to only 4/26 isolates [Nyambi (2000)]
- 654-D: CD4BS MAbs when complexed with gp120, inhibit proliferation of gp120-specific CD4 T-cells and IFN- γ production – anti-CD4BS MAbs inhibit gp120 presentation by altering the uptake and/or processing of gp120 by the APCs, not by blocking of gp120 attachment to CD4 on the surface of APCs [Hioe (2001)]
- 654-D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 $\mu\text{g/ml}$: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]

723 729-D (729–30D)

Env(dis)

gp120(dis LAI)

L HIV-1 infection

human(IgG1 κ)

Ab type: CD4BS

Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu), NYU Med Center, NY, NY

References: [Laal (1994), D’Souza (1997), Li (1997), Parren (1997b), Gorny (2000)]

Table of HIV MAbs

- 729-D: Dissociation constant gp120 IIIb 0.025 – neutralizes IIIb, acts synergistically with anti-V3 MAb 447-52D [Laal (1994)]
- 729-D: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – reported here to have a lambda light chain, but originally reported in [Laal (1994)] to be IgG1kappa [D’Souza (1997)]
- 729-D: Called 720–30D – one of 14 human MAbs tested for ability to neutralize chimeric SHIV-vpu+, which expressed HIV-1 IIIb env [Li (1997)]
- 729-D: Neutralizes TCLA strains, but not primary isolates [Parren (1997b)]
- 729-D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – CD4BS MAbs 559/64-D, 654-D, 729-D, 9CL and 1331E bound with a 5–13 fold preference for the oligomer [Gorny (2000)]

724	830D (830-D)	Env(dis)	gp120(dis)	L	human(IgG1κ)
		Ab type: CD4BS References: [Wyatt (1998), Hioe (2000)]			
		<ul style="list-style-type: none"> ● 830D: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt (1998)] ● 830D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – anti-CD4 binding site MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – CD4BS MAbs 654-D, 559/64-D, 588-D, 830-D, 1027–30-D, and 1202–30D strongly diminished proliferation [Hioe (2000)] 			
725	9CL	Env(dis)	gp120(dis LAI)	HIV-1 infection	human()
		Ab type: CD4BS Donor: Susan Zolla-Pazner (Zollas01@mccr6.med.nyu), NYU Med Center, NY, NY			
		References: [Gorny (2000)]			
		<ul style="list-style-type: none"> ● 9CL: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – CD4BS MAbs 559/64-D, 654-D, 729-D, 9CL and 1331E bound with a 5–13 fold preference for the oligomer [Gorny (2000)] 			
726	anti-CD4BS summary	Env(dis)	gp120(dis)		()
		Ab type: CD4BS References: [Thali (1993), Moore & Sodroski(1996)]			
		<ul style="list-style-type: none"> ● Shared components of MAb epitopes and the discontinuous CD4 binding regions included Thr 257, Asp 368, Glu 370, Lys 421 through Trp 427 and Asp 457 [Thali (1993)] ● Anti-CD4 binding site antibodies (CD4BS) competitively inhibit CD4 binding to monomeric gp120, and they differ in precise dependence on gp120 residues, but generally require Asp-368 and Glu-370 [Moore & Sodroski(1996)] 			

727	b11	Env(dis) Ab type: CD4BS	gp120(dis) References: [Parren (1998a)]		human()
<ul style="list-style-type: none"> • b11: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)] 					
728	b13	Env(dis) Ab type: CD4BS	gp120(dis) References: [Parren (1995), Parren (1998a)]		human()
<ul style="list-style-type: none"> • b13: Fab b13 was used as a control in a hu-PBL SCID mouse study – animals were protected from HIV-1 SF2 infection by IgG1b12, somewhat by Fab b12, but not by b13 [Parren (1995), Parren & Burton(1997)] • b13: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)] 					
729	b14	Env(dis) Ab type: CD4BS	gp120(dis) References: [Parren (1998a)]		human()
<ul style="list-style-type: none"> • b14: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)] 					
730	b3	Env(dis) Ab type: CD4BS	gp120(dis) References: [Parren (1997b), Parren (1998a)]		human()
<ul style="list-style-type: none"> • b3: Neutralizes TCLA strains, but not primary isolates [Parren (1997b)] • b3: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)] 					
731	b6	Env(dis) Ab type: CD4BS	gp120(dis) References: [Parren (1997b), Parren (1998a)]	L	human()
<ul style="list-style-type: none"> • b6: Neutralizes TCLA strains, but not primary isolates [Parren (1997b)] 					

Table of HIV MAbs

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

732	BM12	Env(dis) Ab type: CD4BS	gp120(dis) References: [Kessler 2nd (1995)]	L	HIV-1 infection	human()
<ul style="list-style-type: none"> • BM12: Broad cross-clade neutralization of primary isolates – additive effect in combination with MAb 2F5 [Kessler 2nd (1995)] 						
733	D20	Env(dis) Vaccine: <i>Vector/type:</i> vaccinia Ab type: CD4BS	gp120(dis IIIB) <i>Strain:</i> IIIB Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD References: [Earl (1994), Broder (1994), Richardson (1996), Otteken (1996), Earl (1997), Sugiura (1999)]	no	Vaccine	murine(IgG)
<ul style="list-style-type: none"> • <i>HIV component:</i> oligomeric gp140 • D20: Binding completely blocked by pooled human sera [Broder (1994)] • D20: Human sera blocked binding in oligomeric ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 [Richardson (1996)] • D20: Pulse label experiments of 4 MAbs (D20, D27, T20, and T22) binding to noncleavable gp160 revealed that these anti-CD4 MAbs bound with a delay, and that the epitope formed with a $t_{1/2}$ of about 10 minutes [Otteken (1996)] • D20: Used for comparison in a study of gp41 antibodies – D20 binds to a greater extent to cell surface expressed Env than any of 38 conformation dependent anti-gp41 MAbs [Earl (1997)] • D20: A 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 [Sugiura (1999)] 						
734	D21	Env(dis) Vaccine: <i>Vector/type:</i> vaccinia Ab type: CD4BS	gp120(dis IIIB) <i>Strain:</i> IIIB Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD References: [Earl (1994), Sugiura (1999)]		Vaccine	murine(IgG)
<ul style="list-style-type: none"> • D21: A 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 [Sugiura (1999)] 						
735	D24	Env(dis) Vaccine: <i>Vector/type:</i> vaccinia Ab type: CD4BS	gp120(dis IIIB) <i>Strain:</i> IIIB Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD References: [Earl (1994), Sugiura (1999)]	no	Vaccine	murine(IgG)
<ul style="list-style-type: none"> • <i>HIV component:</i> oligomeric gp140 						

- D24: A 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 [Sugiura (1999)]

736	D25	Env(dis)	gp120(dis IIIB)		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • D25: A 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 [Sugiura (1999)] 						
737	D28	Env(dis)	gp120(dis IIIB)	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • D28: A 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 [Sugiura (1999)] 						
738	D35	Env(dis)	gp120(dis IIIB)		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • D35: A 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 [Sugiura (1999)] 						
739	D39	Env(dis)	gp120(dis IIIB)		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • D39: A 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 [Sugiura (1999)] 						
740	D42	Env(dis)	gp120(dis IIIB)		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p>						

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Ab type: CD4BS **Donor:** P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD

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

- D42: A 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 [Sugiura (1999)]

741	D52	Env(dis)	gp120(dis IIIB)		Vaccine	murine(IgG)
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Vaccine: *Vector/type:* vaccinia *Strain:* IIIB *HIV component:* oligomeric gp140

Ab type: CD4BS **Donor:** P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD

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

- D52: A 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 [Sugiura (1999)]

742	D53	Env(dis)	gp120(dis IIIB)		Vaccine	murine(IgG)
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Vaccine: *Vector/type:* vaccinia *Strain:* IIIB *HIV component:* oligomeric gp140

Ab type: CD4BS **Donor:** P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD

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

- D53: A 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 [Sugiura (1999)]

743	D60	Env(dis)	gp120(dis IIIB)	no	Vaccine	murine(IgG)
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Vaccine: *Vector/type:* vaccinia *Strain:* IIIB *HIV component:* oligomeric gp140

Ab type: CD4BS **Donor:** P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD

References: [Earl (1994), Richardson (1996), Sugiura (1999)]

- D60: A 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 [Sugiura (1999)]

744	DA48	Env(dis)	gp120(dis BRU)		HIV-1 infection	human()
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Ab type: CD4BS **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 [Parren (1998a)]

- 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 [Sullivan (1998a)]

745 DO8i	Env(dis) Ab type: CD4BS	gp120(dis BRU) References: [Parren (1998a)]		HIV-1 infection	human Fab()
		<ul style="list-style-type: none"> • DO8i: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142–10 > DA48 > L17) was markedly different than Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142–10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)] • 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 [Sullivan (1998a)] 			
746 F105 (F-105)	Env(dis) Ab type: CD4BS	gp120(dis) 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), Wisniewski (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), Cavacini (1999), Oscherwitz (1999), Baba (2000), Park (2000), Kolchinsky (2001), York (2001)]	L	HIV-1 infection	human(IgG1κ)
		<ul style="list-style-type: none"> • F105: First description of F105, binds topographically near the CD4-binding site – inhibits binding of free, infectious virions to uninfected HT-H9 cells, but does not react with virus adsorbed to uninfected HT-H9 cells – soluble rCD4 pre-bound to infected cells inhibits F105 binding – F105 inhibits infection of HT-H9 cells in standard neutralization assays with HIV-1 and MN strains [Posner (1991)] • F105: F105 neutralization escape mutants result from changes in amino acids in discontinuous regions: C2, 256–262 and C3, 386–370 • F105: Amino acid substitutions that impair F105 neutralization inhibit gp120-CD4 interaction [Thali (1992a)] • F105: MAb cDNA sequence – V H4 V71–4 rearranged with a D H D-D fusion product of dlr4 and da4, and with J H5 – V κ is from the Humvk325 germline gene joined with Jkappa 2 [Marasco (1992)] 			

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- F105: Precipitation of Δ 297–329 env glycoprotein, which has a deleted V3 loop, is much more efficient than precipitation of wild type [Wyatt (1992)]
- F105: F105 mediates ADCC against SF2 through the CD16+ population of PBMC – does not mediate complement-dependent cytotoxicity [Posner (1992b)]
- F105: Significant enhancement of F105 binding to RF infected cells preincubated with V3-specific MAbs V3–2 and V3–1 [Posner (1992a)]
- F105: Called F-105 – neutralizes IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- F105: F105 binds to and neutralizes selected lab strains and 3/9 HIV-1 primary isolates – synergistic enhancement of neutralization by seropositive sera [Posner (1993)]
- F105: No neutralization of primary isolates observed (John Moore, pers comm)
- F105: Additive MN or SF2 neutralization when combined with anti-V3 MAbs 447-52D and 257-D [Cavacini (1993a)]
- F105: Serum from all asymptomatic HIV-1 positive people tested block F105 binding, but only from 27% of symptomatic individuals [Cavacini (1993b)]
- F105: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is 2.4- and 13-fold greater, respectively, than binding to wildtype gp120 [Wyatt (1993)]
- F105: Study of synergism between F105 and sera from vaccinated volunteers with V3-loop specific neutralization activity – 2/3 sera demonstrated neutralization synergy, and 3/3 binding/fusion-inhibition synergy [Montefiori (1993)]
- F105: Study of synergism of neutralization and binding comparing F105 and sCD4 with the V3 MAbs: 50.1, 59.1, 83.1, and 58.2 – synergy was observed, and the data suggest that binding of one ligand (F105) can increase the binding of the second (*e.g.* V3 loop MAbs) due to conformational changes [Potts (1993)]
- F105: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – required >81 fold higher concentrations to neutralize the mutant than wild type [Klasse (1993a)]
- F105: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – F105 was used as a control – infected lab workers and some of the gp160 vaccinees had a MAb response that could inhibit gp120-CD4 binding, at lower titers than the infected lab workers [Pincus (1993)]
- F105: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – F105 neutralization was not affected by this mutation [Watkins (1993)]
- F105: Comparison of MAb F105 sequences with those of MAbs 21h and 15e [Bagley (1994)]
- F105: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs 48d, 21h, 15e and 17b) [Thali (1994)]
- F105: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – anti-CD4 MAbs moderately inhibit gp120 binding to GalCer, possibly through steric hindrance – binding of GalCer to gp120 inhibited but did not completely block F105 binding[Cook (1994)]
- F105: Administered intravenously to four cynomolgus monkeys, plasma pharmacokinetics and biological activity tested [Cavacini (1994b)]
- F105: Fab fragments show reduced capacity to neutralize IIIB, MN, and RF compared to intact IgG1, suggesting bivalent interaction may be important in binding and neutralization [Cavacini (1994a)]

- F105: Used as a positive control for CD4 BS antibodies in a study of the influence of oligomeric structure of Env in determining the repertoire of the Ab response [Earl (1994)]
- F105: A human CD4+ T lymphocyte line was transduced to express Fab fragments of F105 – heavy and light chains are joined by an inter-chain linker – in the transduced cells infected with HIV-1, the Fab binds intracellularly to the envelope protein and inhibits HIV-1 production – secreted Fab fragments neutralize cell-free HIV-1 – combined intra- and extracellular binding activities of the expressed Fab make transduced cells resistant to HIV-1 infection and also can protect surrounding lymphocytes by secreting neutralizing antibodies [Marasco1993, Chen (1994a)]
- F105: An immunoassay for titrating CD4BS serum antibody was developed using a gp120-coated solid phase and competition with MAb F105 – 109/110 French HIV-1+ sera and 51/56 HIV-1+ African sera had detectable CD4BS Abs using this assay, demonstrating CD4 binding site conservation among diverse subtypes – CD4BS Abs were detected soon after seroconversion and persisted – 0/21 HIV-2+ sera reacted, indicating that the HIV-1 and HIV-2 CD4BS Abs are not cross-reactive [Turbica (1995)]
- F105: Eight patient phase Ia trial for use as an immunotherapeutic – no clinical or biochemical side effects observed, plasma levels of 10 µg/ml maintained for 21 days [Posner (1995)]
- F105: Efficient neutralization of T-cell adapted lines HXBc2 and MN, no neutralization of primary isolates 89.6, ADA and YU2 – even some enhancement of infection of ADA and YU2 was observed [Sullivan (1995)]
- F105: Biotinylated F105 was used for competition studies with Ab derived from pregnant HIV-1+ women – a correlation between maternal anti-CD4 BS Abs overlapping the F105 binding site and lack of HIV-1 transmission to infants was noted [Khouri (1995)]
- F105: Changing heavy chain from IgG1 to IgG3 increased neutralization efficiency [Cavacini (1995)]
- F105: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop results in less potent inhibition of F105 binding by CRDS – binding site of F105 described as 256–257 ST, 368–370 DPE, 421 K, and 470–484 PGGGDMRDNRSELY [Jagodzinski (1996)]
- F105: Phase I study – MAb clearance in plasma has a 13 day half-life [Wolfe (1996)]
- F105: Neutralizes HIV-1 LAI less potently than V3 specific MAbs [McDougal (1996)]
- F105: F105 is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisniewski (1996)]
- F105: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)]
- F105: Binding of F105 to oligomeric gp120 occurs despite the fact it cannot neutralize primary isolates [Litwin (1996)]
- F105: Intracellular co-expression of heavy and light chains of the Fab105 fragment MAb F105 was enhanced by inclusion of an internal ribosome entry site (IRES) sequence – the Fab105 IRES expression cassette was cloned into an adeno-associated virus (AAV) shuttle vector, and transduced into human lymphocytes which were able to produce and secrete the Fab105 fragments while maintaining normal growth – several primary HIV-1 patient isolates were effectively blocked [Chen (1996)]
- F105: Neutralizes TCLA strains, but not primary isolates [Parren (1997b)]
- F105: In a multilaboratory blinded study, failed to neutralize any of nine B clade primary isolates [D'Souza (1997)]
- F105: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – F105 could only achieve 50% neutralization alone – all Ab combinations tested showed synergistic neutralization – F105 has synergistic response with MAbs 694/98-D (anti-V3), 48d, 2F5, and 2G12, and also with HIVIG [Li (1997)]

Table of HIV MAbs

- F105: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to a CD4BS MAb, F105 or sCD4 [Cao (1997)]
- F105: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31–93, are deleted [Wyatt (1997)]
- F105: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt (1998)]
- 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 [Cavacini (1998b)]
- F105: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) [Li (1998)]
- 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 [Cavacini (1998a)]
- F105: 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 [Brand (1998)]
- F105: A 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 [Sugiura (1999)]
- F105: F105 enhances viral entry of viruses carrying the YU2 envelope glycoproteins, but neutralizes HXBc2 [Sullivan (1998a)]
- 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) [Kropelin (1998)]
- F105: A triple combination of 2F5, F105 and 2G12 effectively neutralized perinatal infection of macaque infants when challenged with SHIV-vpu+ – the plasma half-life was 7.2 ± 2.2 days [Baba (2000)]
- F105: Host encoded intercellular adhesion molecule (ICAM-1) is incorporated by the HIV-1 virion and enhances viral infectivity – ICAM-1 does not modify virus sensitivity to antibodies 0.5 β or 4.8D or sCD4, but neutralizing ability of F105 was diminished in ICAM bearing virions in the presence of lymphocyte function-association antigen-1 (LFA-1) Ab [Fortin (2000)]
- F105: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form, although F105 was an exception and cannot neutralize either form of MN – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- F105: Mutations in two glycosylation sites in the V2 region of HIV-1 ADA at positions 190 and 197 (187 DNTSYRLINCNTS 199) cause the virus to become CD4-independent and able to enter cells through CCR5 alone – these same mutations tended to increase the neutralization sensitivity of the virus, including to F105 [Kolchinsky (2001)]

- F105: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAb alters some step after binding [York (2001)]
- F105: NIH AIDS Research and Reference Reagent Program: 857

747	F91 (F-91)	Env(dis)	gp120(dis)	L	HIV-1 infection	human(IgG1)
		Ab type: CD4BS		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)]				
		<ul style="list-style-type: none"> • F91: Called F-91 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)] • F91: Has strong cross-reactivity with gp120 monomers from most subtypes, A-F [Moore (1994b)] • F91: Unusual pattern of reciprocal enhancement with several anti-V2 and V3 directed MAbs – reciprocal inhibition of other CD4BS MAbs [Moore & Sodroski(1996)] • F91: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – F91 bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)] • F91: Weak inhibition of binding of Hx10 to CD4 positive or negative cells, weakly neutralizing [Mondor (1998)] • F91: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)] • F91: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MAbs 15e, F91 and IgG1b12 bound better to the deleted protein than to wild type [Binley (1998)] • F91: CD4BS MAbs 15e, 21h, and F91 bind with even lower affinity than 205-43-1 and 205-42-15 to JRFL oligomer – conclusions of this paper contrast with [Parren (1998a)] [Fouts (1998)] 				

748	GP13	Env(dis)	gp120(dis)	L	HIV-1 infection	human(IgG1)
		Ab type: CD4BS		References: [Schutten (1993), Back (1993), Bagley (1994), Schutten (1995a), Schutten (1995b), Bolmstedt (1996), Wisnewski (1996), Schutten (1996), Schutten (1997)]		
		<ul style="list-style-type: none"> • GP13: Neutralized a broad range of HIV-1 strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D), 384(Y/E) [Schutten (1993)] • GP13: Mutations in a neutralization resistant isolate obtained by passage of the IIIB isolate in chimpanzees reduced neutralization, but the escape was not as clear as seen with anti-V3 MAbs [Back (1993)] • GP13: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten (1995a)] • GP13: Neutralizes T-cell adapted viruses but not the SI strain 16.2, despite high binding affinity [Schutten (1995b)] • 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 [Bolmstedt (1996)] 				

Table of HIV MAbs

- GP13: GP13 is V H5 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)]
- GP13: IIIB neutralizing MAbs *in vitro* fail to neutralize in a mouse model *in vivo* [Schutten (1996)]
- GP13: Neutralized (50%) an SI-env chimeric virus and enhanced (>5 fold) an NSI-env chimeric virus [Schutten (1997)]
- GP13: UK Medical Research council AIDS reagent: ARP3054

749	GP44	Env(dis) Ab type: CD4BS	gp120(dis) References: [Schutten (1993), Bagley (1994), Wisnewski (1996)]	L	HIV-1 infection	human(IgG1)
<ul style="list-style-type: none"> • GP44: Exhibited a more restricted pattern of neutralizing activity than GP13 and GP68 – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D) [Schutten (1993)] • GP44: GP44 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)] 						
750	GP68	Env(dis) Ab type: CD4BS	gp120(dis) References: [Schutten (1993), Klasse (1993a), Bagley (1994), Schutten (1995a)]	L	HIV-1 infection	human(IgG1)
<ul style="list-style-type: none"> • GP68: Neutralized a broad range of HIV-1 lab strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 117(K/W), 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q), 384(Y/E), 435(Y/H) [Schutten (1993)] • GP68: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – GP68 required markedly higher concentrations to neutralize the mutant than wild type [Klasse (1993a)] • GP68: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten (1995a)] • GP68: GP68 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)] • GP68: UK Medical Research Council AIDS reagent: ARP3055 						
751	HF1.7	Env(dis) Ab type: CD4BS	gp120(dis) References: [Chanh (1987)]	L	anti-idiotypic	murine(IgM)
<ul style="list-style-type: none"> • HF1.7: An anti-Id antibody stimulated by anti-CD4 MAb Leu-3a binds to recombinant gp160, suggesting HF1.7 mimics CD4 [Chanh (1987)] 						
752	HT5 (205-43-1)	Env(dis) Ab type: CD4BS	gp120(dis) Donor: Ciba-Geigy AG (Basel, Switzerland), and Tanox Biosystems, Houston, Texas References: [Moore (1994b), Moore (1995a), Fouts (1997), Fouts (1998)]	L (weak)	HIV-1 infection	human()
<ul style="list-style-type: none"> • HT5: HT5, HT6, and HT7 are also known as 205-43-1, 205-42-15, and 205-46-9, respectively [Fouts (1998)] • HT5: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN [Moore (1995a)] 						

- HT5: 205-46-9 was cross-reactive across clades A-F, 205-43-1 very cross-reactive but not quite as extensive 205-46-9 [Moore (1994b)]
- HT5: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts (1997)]
- HT5: HT5 and HT6 bind JRSF oligomer but with low affinity, and are not neutralizing – conclusions of this paper contrast with [Parren (1998a)] [Fouts (1998)]

753	HT6 (205-42-15)	Env(dis)	gp120(dis)	L (weak)	HIV-1 infection	human()
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Ab type: CD4BS **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 [Fouts (1998)]
- HT6: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN [Moore (1995a)]
- HT6: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was not quite as extensively cross-reactive [Moore (1994b)]
- HT6: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts (1997)]
- HT6: HT5 and HT6 bind JRSF oligomer but with low affinity, and are not neutralizing – conclusions of this paper contrast with [Parren (1998a)] [Fouts (1998)]

754	HT7 (205-46-9)	Env(dis)	gp120(dis)	L (IIIB)	HIV-1 infection	human()
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Ab type: CD4BS **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 [Fouts (1998)]
- HT7: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only neutralizes IIIB well, with sporadic weak neutralization of other isolates [Moore (1995a)]
- HT7: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was cross-reactive, but not quite as extensive [Moore (1994b)]
- HT7: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts (1997)]
- HT7: Binds JRSF oligomer with high affinity, at least as high as IgG1b12, but IgG1b12 is neutralizing, H7 is not – conclusions of this paper contrast with [Parren (1998a)] – authors propose a model where H7 may inhibit CD4 binding, but cause a conformational shift which enhances CCR5 binding and thus counteracts the neutralizing effect [Fouts (1998)]

755	ICR 39.13g (ICR39.13g, 39.13g)	Env(dis)	gp120(dis)	L	Vaccine	rat(IgG2b)
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Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: CD4BS **Donor:** Jackie Cordell and C. Dean

Table of HIV MAbs

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 [Cordell (1991)]
- ICR 39.13g: Binds to a conformational epitope involved in CD4 binding – exerts a synergistic effect in combination with V3 directed MAbs [McKeating (1992a)]
- ICR 39.13g: Neutralization activity against HXB10, RF, SF-2 and MN strains of HIV-1 [McKeating (1993b)]
- ICR 39.13g: Conformational, does not bind denatured gp120 – weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- ICR 39.13g: Strongly inhibits CD4 inducible MAb 48d [Thali (1993)]
- ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)]
- ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse (1993a)]
- ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]
- ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAb 39.3b [Armstrong & Dimmock(1996)]
- ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)]
- ICR 39.13g: The 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 wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- ICR 39.13g: UK Medical Research Council AIDS reagent: ARP390

756	ICR 39.3b (39.3, 39.3b, ICR39.3b)	Env(dis)	gp120(dis)	L	Vaccine	rat(IgG2b)
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Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: CD4BS **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 [Cordell (1991)]
- ICR 39.3b: Conformational, does not bind to denatured IIIB [Moore & Ho(1993)]
- ICR 39.3b: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively [McLain & Dimmock(1994)]

- ICR 39.3b: Neutralizes only if the antibody is added prior to the attachment of the virus to the cell, in contrast to 39.13g [Armstrong & Dimmock(1996)]
- ICR 39.3b: Called 39.3b – increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs (1996)]
- ICR 39.3b: Called 39.3 – summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding [Wyatt (1998)]
- ICR 39.3b: UK Medical Research Council AIDS reagent: ARP391

757	IgG1b12 (Fab b12, Fab 3B3, MAb IgG1b12, IgG1-b12, IgG1 b12, IgGB12, b4/12, b12, 1b12)	Env(dis)	gp120(dis)	L P	HIV-1 infection	human(IgG1 κ)
		<p>Ab type: CD4BS Donor: D. Burton, Scripps Research Institute, La Jolla, CA, also J. Geltowsky and J. Pyati, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA</p> <p>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), Ditzel (1997), Ugolini (1997), Wyatt (1997), Burton & Montefiori(1997), Boots (1997), Parren (1997b), Parren (1997a), Parren & Burton(1997), Valenzuela (1998), Wyatt (1998), Mondor (1998), Parren (1998a), Connor (1998), Binley (1998), Fouts (1998), Takefman (1998), Parren (1998b), Brand (1998), Schonning (1998), Sullivan (1998a), Frankel (1998), Kropelin (1998), Stamatatos & Cheng-Mayer(1998), Poignard (1999), Jackson (1999), Hioe (1999), Montefiori & Evans(1999), Giraud (1999), Beddows (1999), Binley (1999), Grovit-Ferbas (2000), Ly & Stamatatos(2000), Nyambi (2000), Park (2000), Kolchinsky (2001), Sapphire (2001a), Sapphire (2001b), Yang (2001), York (2001), Zwick (2001a), Zwick (2001b), Zwick (2001c), Poignard (2001), Zeder-Lutz (2001), Spenlehauer (2001), Verrier (2001), Hofmann-Lehmann (2001), Xu (2001), Srivastava (2002)]</p> <ul style="list-style-type: none"> • IgG1b12: Fab b12 was derived from IgG1b12, Fab 3B3 was derived from Fab b12 by random mutagenesis and selected for increased affinity to sgp120 • IgG1b12: The original Fab fragment was derived from a combinatorial phage library from bone marrow of an HIV-1 positive individual who had been asymptomatic for six years [Burton (1991)] • IgG1b12: Anti-CD4 binding site Fab, potent neutralizing activity, greater affinity for a subpopulation of gp120 molecules suggested to be in a mature confirmation – mutations in gp120 that abrogate binding: 368 D/R or D/T, 370 E/R, and 477 D/V, of clone HXBc2 of LAI – sensitive to V1 and V2 substitutions [Roben (1994)] • IgG1b12: Very potent neutralization, of primary and lab strains, at concentrations that could be achieved by passive immunization – reduced binding with A,C, and D clade viruses relative to B clade, poor reactivity with E clade – isolates that were refractive to neutralization by sera from HIV-1+ donors could be neutralized by IgG1 b12 [Burton (1994)] • IgG1b12: Cross-reactive with some gp120s, (but not all), from clades A-D – not reactive with gp120 from clades E or F [Moore (1994b)] • IgG1b12: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau (1995)] • IgG1b12: Anti-CD4 binding site MAb – very potent neutralization of a number of primary isolates [Moore (1995a)] 				

Table of HIV MAbs

- IgG1b12: Complete protection against HIV-1 infection was achieved in hu-PBL-SCID mice by passive immunization with physiologically relevant doses – pharmacokinetics showed serum half-life of 30.2 ± 1.3 hours for Fab b12 and 7.4 ± 0.7 days for IgG1 b12 in mice, but IgG1 half-lives in human are generally between 21–23 days [Parren (1995), Parren & Burton(1997)]
- IgG1b12: Called BM12 – broad cross-clade neutralization of primary isolates – additive neutralization in combination with MAb 2F5 [Kessler 2nd (1995)]
- IgG1b12: Review: unusual properties for anti-CD4 BS MAb: sensitive to V2 substitutions, preferential recognition of the oligomer on the cell surface [Moore & Ho(1995)]
- IgG1b12: Could potentially neutralize primary isolates from within clade B, but showed a slight reduction in efficacy outside of clade B [Trkola (1995)]
- IgG1b12: Because Fab b12 shows reduction in binding when the V2 loop is deleted and when aa 183/184 PI/SG substitutions are made competition studies were done with Fab L78 and anti-V2 MAbs SC258 and 684–238 and they do not compete with IgG1b12 [Ditzel (1995)]
- IgG1b12: Fab b12 showed potent neutralization of T-cell-line-adapted strains, but much reduced neutralization of 3 primary isolates – 2 of the 3 primary isolates also had reduced binding affinity, but the third was as efficiently immunoprecipitated as HXBc2 [Sullivan (1995)]
- IgG1b12: Saturation mutagenesis of the complementarity-determining region and optimization strategies were used to create very high affinity versions of this Fab – increased affinity was dominated by a slowing of the off rate [Yang (1997)]
- IgG1b12: Potent neutralizing *ex vivo* of virus taken directly from plasma of HIV-1 infected individuals – little correlation between neutralization sensitivity of passaged virus and plasma derived virus – more effective than MAb 19b [Gauduin (1996)]
- IgG1b12: Review: Unique among anti-CD4BS MAbs in terms of being potent against both lab adapted virus and primary isolates – one of three MAbs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard (1996b)]
- IgG1b12: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50–69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Poignard (1996a)]
- IgG1b12: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola (1996a)]
- IgG1b12: Review: Only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]
- IgG1b12: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of $< 25 \mu\text{g}$ per ml for 90% viral inhibition – IgG1b12 failed to neutralize only 1/9 primary isolates, although there was some variation between test sites [D’Souza (1997)]
- IgG1b12: Inhibited some SI- and NSI-env chimeric viruses but enhanced one NSI-env chimeric virus 3 fold [Schutten (1997)]
- IgG1b12: JRCSF was cultured in the presence of IgG1b12 until a 100-fold resistance to neutralization was selected – resistance was due to three changes: V2 substitution D182N and C3 substitution P365L conferred resistance, and V2 D164N was also required for a viable virus – IgG1b12 resistant virus remained sensitive to MAbs 2G12 and 2F5 [Mo (1997)]
- IgG1b12: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – IgG1b12 bound monomer, oligomer, and neutralized JRFL [Fouts (1997)]

- IgG1b12: b12 was used in its IgG1 form – of 14 human MAbs, the most potent neutralizer of SHIV-vpu+, which expressed HIV-1 IIIB env – all Ab combinations tested showed synergistic neutralization – b12 has a synergistic response with MAbs 694/98-D (anti-V3), 2F5, and 2G12 [Li (1997)]
- IgG1b12: 35 primary isolates were tested and all were neutralized by IgG1b12 (including 4, UG270, RW92/026, ZB20, and 301727 which been had reported as not neutralized by IgG1b12 [Trkola (1995)]) – IgG1b12 could neutralize even when added after the virus to the culture – selection for 400-fold increased affinity did not enhance neutralization by antibody – IgG1b12 was more potent with greater breadth than MAb 2F5 [Kessler II (1997)]
- IgG1b12: Review: MAbs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MAbs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs' epitopes [Moore & Trkola(1997)]
- IgG1b12: Viral binding inhibition by IgG1b12 strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- IgG1b12: Major deletions in C1 and C5 and deletions of the V1V2 and V3 loops do not diminish binding [Wyatt (1997)]
- IgG1b12: This is a review that includes a description of IgG1b12, noting approximately equivalent affinities for sgp120 and unprocessed gp160, and somewhat enhanced affinity for the native oligomer on TCLA viruses – primary viruses have reduced affinity, but still in the useful range for neutralization – there can be complete protection in hu-PBL-SCID mice with Ab even when administered several hours after viral challenge – competes with sCD4, but unlike other CD4BS antibodies, it is sensitive to mutations in V2 [Burton & Montefiori(1997)]
- IgG1b12: In this review, the technique and potential application of Fab expression and selection in phage display libraries, and subsequent production of IgG molecules is discussed – b12 is 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 [Parren & Burton(1997)]
- IgG1b12: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – IgG1b12 blocks CD4 binding and is the most potent neutralizing Ab – many 15 and 21-mer phage inserts were recognized, but it was not possible to derive a consensus – common features were a W and at least one acidic residue, and one sequence was found multiple times: NWPRWWEFVDKHSS, and this peptide could compete with gp120 – two short stretches found in the phage peptides might mimic gp120 components of the epitope: positions 382–384, FFY(I), and 423–426 I(FV)I(V)NM [Boots (1997)]
- IgG1b12: Fab b12 is unusual in that it binds to gp140 and monomeric gp120 with similar affinities, and with a higher affinity to the native oligomer – authors propose this antibody may be exceptional because it binds the virus rather than viral debris – IgG1b12 can protect against infection prior to or shortly after challenge of hu-PBL-SCID mice with TCLA strains and primary strains, but the serum concentrations required were higher than for *in vitro* neutralization [Parren (1997b), Parren (1997a)]
- IgG1b12: 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 [Valenzuela (1998)]

Table of HIV MAbs

- IgG1b12: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by CD4BS Ab is direct interference with CD4 binding – IgG1b12 is an unusual CD4BS antibody because it is particularly potent as a neutralizing antibody and it is susceptible to changes in the V1-V2 stem loop structure, and so it may disrupt an interaction between CD4 and conserved amino acids on the V1-V2 stem [Wyatt (1998)]
- IgG1b12: Enhances binding of Hx10 to CD4 positive or negative HeLa cells, inhibits binding to CD4+ T-cell line A3.01 – neutralizes HeLa and A3.01 cell Hx10 infection [Mondor (1998)]
- IgG1b12: IgG1b12, Fab b12 and 3B3 derived from b12 were all included in this study – the rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142-10 > DA48 > L17) was markedly different than Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142-10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope – binding affinity of divalent IgG1b12 is 17-fold greater than monovalent Fab b12 [Parren (1998a)]
- IgG1b12: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D [Connor (1998)]
- IgG1b12: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4BS MAbs 15e, F91 and IgG1b12 bound better to the deleted protein than to wild type [Binley (1998)]
- IgG1b12: Binds JRSF oligomer with high affinity, as do 205-46-9 and 2G6, but IgG1b12 is neutralizing, the other two are not – conclusions of this paper contrast with 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 [Fouts (1998)]
- IgG1b12: Induces Complement-mediated lysis in MN but not primary isolates – primary isolates are refractive to CML [Takefman (1998)]
- IgG1b12: MAbs 2G12, 2F5 and b12 are broadly neutralizing, as are some human polyconal sera, but this paper describes a set of primary isolates that are resistant to all three MAbs and 2 broadly neutralizing sera – results indicate that resistance levels of pediatric isolates might be higher than adult isolates – resistance in general did not seem to be conferred by a loss of binding affinity for gp120 or gp41, rather by a more global perturbation of oligomeric Envelope [Parren (1998b)]
- IgG1b12: 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 [Brand (1998)]
- IgG1b12: 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 [Schonning (1998)]

- IgG1b12: 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 [Sullivan (1998a)]
- IgG1b12: anti-C1 region MAb 87–135/9 blocks gp120 interaction with CD4+ cells – blocking activity is additive when combined with antibodies which bind in the C4 region of gp120 (F105, 388/389, and b12) [Kropelin (1998)]
- IgG1b12: Deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V2, but not V1, diminished neutralization by CD4BS MAb IgG1b12, in contrast to 654.30D and IgGCD4 [Stamatatos & Cheng-Mayer(1998)]
- IgG1b12: Infection of dendritic cells cultured from CD14+ blood cells or from cadaveric human skin was blocked by neutralizing MAbs IgG1b12, or 2F5 and 2G12 delivered together, but not by control non-neutralizing anti-gp120 MAb 4.8D, indicating that NAb could interrupt early mucosal transmission events [Frankel (1998)]
- IgG1b12: 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 [Hioe (1999)]
- IgG1b12: 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 [Beddows (1999)]
- IgG1b12: 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* [Montefiori & Evans(1999)]
- IgG1b12: 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 [Jackson (1999)]
- IgG1b12: Hu-PBL-SCID mice were infected with HIV-1s JRCSF and SF162 to study the effect of NAb 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 [Poignard (1999)]

Table of HIV MAbs

- IgG1b12: 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 [Binley (1999)]
- IgG1b12: To determine the antigenicity of virus killed by thermal and chemical inactivation, retention of conformation-dependent neutralization epitopes was examined, and exposure of CD4BS epitopes was found to be enhanced (MAbs IgG1b12, 205-46-9, and 205-43-1) – binding to 2G12 and 447-52D epitopes was essentially unaltered – the 17b CD4i epitope was also exposed [Grovit-Ferbas (2000)]
- IgG1b12: SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391-95D) – V2-region glycosylation site mutations did not enhance neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows increased infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]
- IgG1b12: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 CD4BS MAbs – CD4BS MAbs bound consistently to most isolates of clade D, but poorly to isolates of other clades with the exception of broadly reactive MAb IgG1b12, binding to 22 of 26 isolates tested – 8 MAbs were tested for neutralization and MAb IgG1b12 was most potent, with 90% neutralization of 3/5 isolates tested [Nyambi (2000)]
- IgG1b12: Fab b12 was used – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- IgG1b12: Mutations in two glycosylation sites in the V2 region of HIV-1 ADA at positions 190 and 197 (187 DNTSYRLINCNTS) cause the virus to become CD4-independent and able to enter cells through CCR5 alone – these same mutations tended to increase the neutralization sensitivity of the virus, except the mutation 197 S/R which resulted in a carbohydrate addition to 195 N that disrupts the IgG1b12 binding site [Kolchinsky (2001)]
- IgG1b12: This paper describes the technical aspects of the crystallization of b12 at a resolution of 2.7 angstroms with all 12 Ig domains resolved [Saphire (2001a)]
- IgG1b12: This paper describes the biological implications of the crystal structure of b12 – a remarkable feature of this antibody is a long protruding finger-like CDR H3 that can dock in the recessed CD4-binding site – a contact residues in gp120 are modeled, with numbering based on the variable loop-deleted crystal structure of gp120 [Saphire (2001b)]
- IgG1b12: Primary isolates YU2 and ADA are more resistant to IgG1b12 neutralization than HXBc2: 90% Neutralization of HXBc2 is observed with 1.25 ug of IgG1b12, while ADA and YU2 require 2.5 and 5 ug respectively to achieve 50% neutralization, and 90% neutralization could not be achieved with 10 or 20 ug of IgG1b12, respectively [Yang (2001)]

- IgG1b12: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAb alters some step after binding [York (2001)]
- IgG1b12: b12 recognizes a conformational epitope that overlaps with the CD4 binding site – a phage displayed peptide library was used to identify a peptide which bound b12, called B2.1, which competes with b12 in competition assays – B2.1 has significant homology to the D loop of gp120: upper case letters indicate residues B2.1 shares with gp120, heRsymFSDlenrcI – one of the goals of defining peptide mimics to the b12 epitope is to develop an immunogen that can stimulate b12-like antibodies, but B2.1 cross-linked to phage and ovalbumin bound IgG1b12 did not elicit cross-reactive gp120 Abs in mice or rabbits [Zwick (2001a)]
- IgG1b12: This paper primarily concerns 4E10 and Z13, MAbs that both bind proximally to the 2F5 binding site to a conserved epitope, and that neutralize some primary isolates from clades B, C, and E – broadly neutralizing MAbs 2F5, IgG1b12, and 4E10 and Z13 fail to neutralize different subsets of viruses [Zwick (2001b)]
- IgG1b12: Neutralization synergy between anti-HIV NAb b12, 2G12, 2F5, and 4E10 was studied – a classic fixed-ratio method was used, as well as a method where one Ab was fixed at a low neutralization titer and the other was varied – using primary isolates, a two-four fold enhancement of neutralization was observed with MAb pairs, and a ten-fold enhancement with a quadruple Ab combination – no synergy was observed with any MAb pair in the neutralization of TCLA strain HXB2 – whole IgG1b12 and b12 Fab fragments behaved similarly in the neutralization assays – there was no evidence for cooperativity of binding between b12 and 2G12 to envelope spikes expressed on the cell surface of TCLA or primary isolates [Zwick (2001c)]
- IgG1b12: Intravenous passive transfer of MAb b12 provides dose-dependent protection from infection to macaques vaginally challenged with the R5 virus SHIV(162P4) – the primary isolate HIV-1SF162 is neutralized 90% (IC₉₀) by b12 at 2 µg/ml, and SHIV162P4, derived from HIV-1SF162, was neutralized by 90% at 2 µg/ml in PHA-activated PBMC from rhesus macaques – the 90% neutralization titers achieved in three groups of animals that were given 25-, 5-, and 1-mg/kg doses were approximately 1:400, 1:80, and 1:16, respectively – the half-life of IgG1 b12 in plasma was about 1 week, but while the peak b12 plasma concentration was immediately after the infusion, the peak vaginal fluid concentration was 7–14 days later [Parren2001]
- IgG1b12: Structural aspects of the interaction of neutralizing Abs with HIV-1 Env are reviewed – Env essentially has three faces, one is largely inaccessible on the native trimer, and two that exposed but have low immunogenicity on primary viruses – neutralization is suggested to occur by inhibition of the interaction between gp120 and the target cell membrane receptors as a result of steric hindrance and it is noted that the attachment of approximately 70 IgG molecules per virion is required for neutralization, which is equivalent to about one IgG molecule per spike – the 2G12, 17b and b12 epitopes are discussed in detail – the structure of CD4-bound gp120 reveals features that HIV has evolved to escape anti-CD4BS Abs like IgG1b12 despite profound functional constraints – CD4BS Abs must first access the CD4 binding site, deeply recessed within the gp120 core, and the Fab of an Ab molecule is “wider” than CD4, and in addition the binding site is flanked by variable and glycosylated regions [Poignard (2001)]
- IgG1b12: Neutralizing synergy between MAbs 1b12, 2G12 and 2F5 was studied using surface plasmon resonance to determine the binding kinetics for these three MAbs with respect to monomeric and oligomeric env protein gp160 IIIB – the 2G12 epitope is highly accessible on both monomeric and oligomeric Envs, 1b12 is highly accessible on monomers but not oligomers, and 2F5 on neither form – binding of 2G12 exposes the 2F5 epitope on gp160 oligomers [Zeder-Lutz (2001)]

Table of HIV MAbs

- IgG1b12: A luciferase-reporter gene-expressing T-cell line was developed to facilitate neutralization and drug-sensitivity assays – luciferase and p24 antigen neutralization titer end points were found comparable using NAb from sera from HIV+ donors, and MAbs 2F5, 2G12 and IgG1b12 [Spencehauer (2001)]
- IgG1b12: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 $\mu\text{g/ml}$: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 M Abs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- IgG1b12: A combination of MAbs IgG1b12, 2F5, and 2G12 was given postnatally to four neonate macaques that were then challenged with highly pathogenic SHIV89.6P – one of the four infants remained uninfected after oral challenge, two infants had no or a delayed CD4(+) T-cell decline – the most potent combination included IgG1b12, which alone does not neutralize SHIV89.6P [Hofmann-Lehmann (2001)]
- IgG1b12: Twenty HIV clade C isolates from five different countries were susceptible to neutralization by anti-clade B MAbs in a synergistic quadruple combination of mAbs IgG1b12, 2G12, 2F5, and 4E10 [Xu (2001)]
- IgG1b12: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – Abs directed against the CD4 binding site (IgGCD4 and IgG1b12) reacted slightly more strongly with the gp120 monomer than with the oligomer, as did sCD4 [Srivastava (2002)]
- IgG1b12: UK Medical Research Council AIDS reagent: ARP3065
- IgG1b12: NIH AIDS Research and Reference Reagent Program: 2640

758	IgGCD4 (IgG-CD4)	Env(dis)	gp120(dis)	human(IgG)
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Ab type: CD4BS **Donor:** Genetech

References: [Capon (1989), Stamatatos & Cheng-Mayer(1998), Ly & Stamatatos(2000), Srivastava (2002)]

- IgGCD4: An antibody-like immunoadhesins molecule was constructed incorporating the gp120-binding domain of CD4 [Capon (1989)]
- IgGCD4: Deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V2 but not V1 slightly enhanced neutralization by CD4BS MAb IgGCD4 [Stamatatos & Cheng-Mayer(1998)]
- IgGCD4: SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391–95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]

Table of HIV MAbs

- IgGCD4: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – Abs directed against the CD4 binding site (IgGCD4 and IgG1b12) reacted slightly more strongly with the gp120 monomer than with the oligomer, as did sCD4 [Srivastava (2002)]

759	L28	Env(dis) Ab type: CD4BS	gp120(dis) References: [Ditzel (1995)]	L	HIV-1 infection	human(IgG1κ)
<ul style="list-style-type: none"> • L28: Substitutions at 257 T/R, 368 D/R, 370 E/R and 370 E/Q, 475 M/S 102 E/L and 463 N/D reduce binding – binding was enhanced by removal of the V3 loop and by substitutions 45 W/S, 298 R/G, 381 E/P, 382 F/L, 420 I/R, 435 Y/H or Y/R – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel (1995)] 						
760	L33	Env(dis) Ab type: CD4BS	gp120(dis) References: [Ditzel (1995)]	L	HIV-1 infection	human(IgG1κ)
<ul style="list-style-type: none"> • L33: binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel (1995)] 						
761	L41	Env(dis) Ab type: CD4BS	gp120(dis) References: [Ditzel (1995)]	L	HIV-1 infection	human(IgG1κ)
<ul style="list-style-type: none"> • 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 – this Fab was retrieved from the library after masking with known anti-CD4BS MAbs – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel (1995)] 						
762	L42	Env(dis) Ab type: CD4BS	gp120(dis) References: [Ditzel (1995)]	L	HIV-1 infection	human(IgG1κ)
<ul style="list-style-type: none"> • L42: Substitutions at 257 T/R, 368 D/R, 370 E/R, 266 A/E and 477 D/V reduce binding – binding was significantly enhanced by 381 E/P and 382 F/L – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel (1995)] 						
763	L52	Env(dis) Ab type: CD4BS	gp120(dis) References: [Ditzel (1995)]	L	HIV-1 infection	human(IgG1κ)
<ul style="list-style-type: none"> • L52: Binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel (1995)] 						
764	L72	Env(dis) Ab type: CD4BS References: [Ditzel (1997)]	gp120(dis) Donor: Dr. Hariharam, IDEC Pharmaceuticals Corp La Jolla, CA References: [Ditzel (1997)]			murine()
<ul style="list-style-type: none"> • L72: Used to bind gp120 to solid phase to select MAbs from a phage selection library [Ditzel (1997)] 						
765	M12	Env(dis) Vaccine: <i>Vector/type:</i> vaccinia Ab type: CD4BS References: [Earl (1994), Sugiura (1999)]	gp120(dis IIIB) <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140 Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD References: [Earl (1994), Sugiura (1999)]	L	Vaccine	murine(IgG)
<ul style="list-style-type: none"> • M12: There is a p15 gag specific MAb also named M12 						

Table of HIV MAbs

- M12: A 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 $\mu\text{g/ml}$ of M12 [Sugiura (1999)]

766	M13	Env(dis)	gp120(dis IIIB)	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • M13: A 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 $\mu\text{g/ml}$ of M13 [Sugiura (1999)] 						
767	M6	Env(dis)	gp120(dis IIIB)	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • M6: A 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 [Sugiura (1999)] 						
768	MAG 116	Env(dis)	gp120(dis)	L	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120</p> <p>Ab type: CD4BS Donor: C. Y. Kang, IDEC Inc</p> <p>References: [Kang (1994)]</p> <ul style="list-style-type: none"> • MAG 116: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L – neutralizes MN, IIIB and RF [Kang (1994)] 						
769	MAG 12B	Env(dis)	gp120(dis)	L	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120</p> <p>Ab type: CD4BS Donor: C. Y. Kang, IDEC Inc</p> <p>References: [Kang (1994)]</p> <ul style="list-style-type: none"> • MAG 12B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 477 D/V – weak neutralization of IIIB [Kang (1994)] 						
770	MAG 29B	Env(dis)	gp120(dis)	L	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120</p>						

		Ab type: CD4BS Donor: C. Y. Kang, IDEC Inc References: [Kang (1994)]			
		<ul style="list-style-type: none"> • MAG 29B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 386 N/Q, 421 K/L – weak neutralization of IIIB [Kang (1994)] 			
771	MAG 3B	Env(dis) gp120(dis)	no	Vaccine	murine()
		Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120			
		Ab type: CD4BS Donor: C. Y. Kang, IDEC Inc References: [Kang (1994)]			
		<ul style="list-style-type: none"> • MAG 3B: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R or A or G, 262 N/T, 368 D/R or T, 370 E/R or Q, 381 E/P, 384 Y/E, 421 K/L, 475 M/S, 477 D/V [Kang (1994)] 			
772	MAG 55 (#55)	Env(dis) gp120(dis)	L	Vaccine	murine()
		Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120			
		Ab type: CD4BS Donor: C. Y. Kang, IDEC Inc References: [Kang (1994), Moore & Sodroski(1996)]			
		<ul style="list-style-type: none"> • MAG 55: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 470 P/L, 475 M/S, 477 D/V – neutralizes MN, IIIB and RF [Kang (1994)] • MAG 55: Called #55 – binding reciprocally inhibited by other anti-CD4 binding site MAbs, and by some C1-C5 MAbs – binding enhanced by anti-V3 MAb 110.5 and anti-V2 MAbs G3-136 and G3-4 – enhances binding of many anti-V3 and -V2 MAbs. [Moore & Sodroski(1996)] 			
773	MAG 72 (L72)	Env(dis) gp120(dis)	L	Vaccine	murine()
		Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120			
		Ab type: CD4BS Donor: C. Y. Kang or Dr. Hariharam, IDEC Pharmaceuticals Corp, La Jolla, CA References: [Kang (1994), Ditzel (1997)]			
		<ul style="list-style-type: none"> • MAG 72: Amino acid substitutions that reduce binding 10 fold: 257 T/R or A or G, 262 N/T, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 477 D/V – neutralizes MN, IIIB and RF [Kang (1994)] • MAG 72: Called L72 – used to bind gp120 to solid phase to select MAbs from a phage selection library [Ditzel (1997)] 			
774	MAG 86	Env(dis) gp120(dis)	L	Vaccine	murine()
		Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120			
		Ab type: CD4BS Donor: C. Y. Kang, IDEC Inc References: [Kang (1994)]			
		<ul style="list-style-type: none"> • MAG 86: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 470 P/L, 477 D/V – neutralizes MN, IIIB and RF [Kang (1994)] 			

Table of HIV MAbs

775	MAG 96	Env(dis)	gp120(dis)	L	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120</p> <p>Ab type: CD4BS Donor: C. Y. Kang, IDEC Inc</p> <p>References: [Kang (1994)]</p> <ul style="list-style-type: none"> • MAG 96: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R – weak neutralization of IIIB [Kang (1994)] 						
776	MTW61D	Env(dis)	gp120(dis W61D)	L	HIV-1 infection	human()
<p>Ab type: CD4BS References: [Sullivan (1998a)]</p> <ul style="list-style-type: none"> • 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 [Sullivan (1998a)] 						
777	S1-1	Env(dis)	gp120(dis)	L	HIV-1 infection	human(IgG1λ)
<p>Ab type: CD4BS References: [Lake (1992), Moran (1993), Wisnewski (1996)]</p> <ul style="list-style-type: none"> • S1-1: Neutralizes IIIB and MN without complement, and neutralizes RF and a clinical isolate with complement – binds to native but not denatured gp120 – inhibits sCD4-gp120 binding [Lake (1992)] • S1-1: Heavy (V H1) and light (V λIII) chain sequenced – no enhancing activity – similar germline sequence to MAb 86, but very different activity [Moran (1993)] • S1-1: S1-1 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)] 						
778	T13	Env(dis)	gp120(dis IIIB)	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • T13: A 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 [Sugiura (1999)] 						
779	T49	Env(dis)	gp120(dis IIIB)	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p>						

- T49: A 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 [Sugiura (1999)]

780	T56	Env(dis)	gp120(dis IIIB)	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • T56: A 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 [Sugiura (1999)] 						
781	TH9	Env(dis)	gp120(dis)	L		human(IgG1 κ)
<p>Ab type: CD4BS Donor: Michael Fung, Tanox Biosystem, USA</p> <p>References: [D'Souza (1995), Yang (1998)]</p> <ul style="list-style-type: none"> • TH9: Found to neutralize MN, but not JRCSF, two B subtype primary isolates, or a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs[D'Souza (1995)] • 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 [Yang (1998)] 						
782	D33	Env(dis)	gp120(dis IIIB)		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: CD4BS, C-term, N-term Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • D33: A 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 [Sugiura (1999)] 						
783		Env(dis)	gp120(dis)	yes		human()
<p>Ab type: CD4BS, CD4i, V3, V2 References: [Moore (2001)]</p> <ul style="list-style-type: none"> • Moore and colleagues review structural aspects of gp120 and how they relate to antigenic domains, and review the data concerning the lack of a clear relationship between genetic subtype and serotype – they suggest the primary goal in vaccine efforts should be to design an immunogen that can be shown to elicit neutralizing antibodies against a significant proportion of primary isolates – assay artifacts that can result in confused interpretations are also discussed, such as Ab binding to defective spikes, which does not affect HIV-1 infectivity, but can dominant an assay signal [Moore (2001)] 						

Table of HIV MAbs

784 17b

Env(dis conserved gp120(dis) regions in gp120)

L P (weak) HIV-1 infection

human()

Ab type: CD4i **Donor:** J. Robinson

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), Stamatatos & Cheng-Mayer(1998), Oscherwitz (1999), Hoffman (1999), Binley (1999), Grovit-Ferbas (2000), Ly & Stamatatos(2000), Park (2000), Salzwedel (2000), Stamatatos (2000), Kolchinsky (2001), York (2001), Zhang (2001), Poignard (2001), Srivastava (2002)]

- 17b: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs
- 17b: Epitope is better exposed upon CD4 binding to gp120 – competes with 15e and 21h, anti-CD4 binding site MAbs – 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 433A/L, 438 P/R and 475 M/S confer decreased sensitivity to neutralization [Thali (1993)]
- 17b: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding of 17b [Moore (1993c)]
- 17b: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 21h and 15e) [Thali (1994)]
- 17b: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 17b in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 – similar effect observed for 48d and A32 [Wyatt (1995)]
- 17b: Binds with higher affinity to monomer and oligomer, slow association rate, poor neutralization of lab strain – this is in contrast to 48d, which has very different kinetics [Sattentau & Moore(1995)]
- 17b: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) – anti-V3 MAb 5G11 enhances binding, as do C1-C4 discontinuous epitopes A32 and 2/11c – enhances binding of some anti-V2 MAbs [Moore & Sodroski(1996)]
- 17b: Binding did not result in significant gp120 dissociation from virion, in contrast to 48d, although the the gp41 epitope of MAb 50–69 was exposed [Poignard (1996a)]
- 17b: MIP-1 α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of 17b blocks this inhibition [Wu (1996)]
- 17b: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola (1996a)]
- 17b: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 17b bound monomer, oligomer, and neutralized JRFL in the presence of sCD4, but if sCD4 was not present, 17b only bound monomer [Fouts (1997)]
- 17b: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 17b has synergistic response in combination with anti-V3 MAb 694/98-D [Li (1997)]
- 17b: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the Can0A V3 peptide, suggesting Can0A V3 is a conformer that mimics the 48d – it does not bind to 17b, distinguishing the epitopes [Weinberg (1997)]
- 17b: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to a CD4BS MAb, F105, or sCD4 [Cao (1997)]

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

Table of HIV MAbs

- 17b: Deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V2 but not V1 enabled neutralization by CD4i MAbs 17b and 48d [Stamatatos & Cheng-Mayer(1998)]
- 17b: A CD4-independent viral variant of IIIB, IIIBx, was generated on CXCR4-expressing cells – IIIBx exhibited greater exposure of the 17b and 48d epitopes and enhanced neutralization by CD4i MAbs and by polyclonal human sera – the 17b epitope has significant overlap with the CCR5 coreceptor binding site [Hoffman (1999)]
- 17b: 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 [Binley (1999)]
- 17b: To determine the antigenicity of virus killed by thermal and chemical inactivation, retention of conformation-dependent neutralization epitopes was examined, and exposure of CD4BS epitopes was found to be enhanced (MAbs IgG1b12, 205-46-9, and 205-43-1) – binding to 2G12 and 447-52D epitopes was essentially unaltered – the 17b CD4i epitope was also exposed [Grovit-Ferbas (2000)]
- 17b: SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391-95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]
- 17b: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- 17b: sCD4 can activate fusion between effector cells expressing Env and target cells expressing coreceptor (CCR5 or CXCR4) alone without CD4 – CD4i MAbs 17b and 48d have little effect on a standard cell fusion assay but potently block sCD4 activated fusion – 17b was broadly cross-reactive inhibiting sCD4 activated fusion with Env from clades A, B, C, D, E, F, and F/B [Salzwedel (2000)]
- 17b: Soluble gp140 derived from SF162, a neutralization-resistant primary isolate, and SF162AV2 a neutralization-susceptible isolate with 30 amino acids deleted from the V2 loop, were generated with or without the gp120-gp41 cleavage site intact – all forms are recognized by oligomer-specific MAb T4 and show enhanced binding of CD4i MAb 17b when sCD4 is bound – the fused forms are less efficiently recognized than the cleaved forms by polyclonal neutralizing sera from HIV-infected patients – the V3 loop is more exposed on the fused form [Stamatatos (2000)]

- 17b: Mutations in two glycosylation sites in the V2 region of HIV-1 ADA at positions 190 and 197 (187 DNTSYRLINCNTS 199) cause the virus to become CD4-independent and able to enter cells through CCR5 alone – these same mutations tended to increase the neutralization sensitivity of the virus, including to 17b – only the CD4i antibodies 17b and 48d showed an increased affinity of the CD4 independent viruses relative to wild-type [Kolchinsky (2001)]
- 17b: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding – 17b bound at somewhat greater levels to 168C than to 168P, but this is not a general feature of 17b binding to primary versus TCLA strains [York (2001)]
- 17b: 17b binds to a CD4 inducible epitope which partially overlaps the CCR5 binding site – JRFL, YU2, 89.6, and HXB2 and their C1-, V1/V2-, C5 -deletion mutants were used to study how 17b binding affects gp120-CD4 interactions – 17b reduced CD4-gp120 interactions by decreasing the on-rate and increasing the off-rate of sCD4, while enhanced binding of sCD4 binding was observed for the 17b-bound, V1/V2 deleted gp120s – 17b was considered to be a surrogate for CCR5, and the authors suggest that 17b binding may shift V1/V2 into a position that interferes with CD4 binding, forcing a release [Zhang (2001)]
- 17b: Structural aspects of the interaction of neutralizing Abs with HIV-1 Env are reviewed – Env essentially has three faces, one is largely inaccessible on the native trimer, and two are exposed but have low immunogenicity on primary viruses – neutralization is suggested to occur by inhibition of the interaction between gp120 and the target cell membrane receptors as a result of steric hindrance and it is noted that the attachment of approximately 70 IgG molecules per virion is required for neutralization, which is equivalent to about one IgG molecule per spike – the 2G12, 17b and b12 epitopes are discussed in detail – the 17b epitope is masked prior to CD4 binding by the V1-V2 loop and in contrast to sCD4, the binding of cell surface CD4 to virus does not appear to make the epitope accessible to binding by 17b to allow neutralization [Poignard (2001)]
- 17b: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – 17b recognized both gp120 monomer and o-gp140 [Srivastava (2002)]

785	48d (4.8d, 4.8D)	Env(dis)	gp120(dis)	L P (weak)	HIV-1 infection	human(IgG1κ)
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Ab type: CD4i **Donor:** J. Robinson, Tulane University, New Orleans, LA, USA

References: [Thali (1993), Moore & Ho(1993), Moore (1993c), Thali (1994), Moore (1994b), D'Souza (1995), Satten-tau(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), Frankel (1998), Wyatt (1998), Mondor (1998), Parren (1998a), Sullivan (1998b), Yang (1998), Binley (1998), Stamatatos & Cheng-Mayer(1998), Oscherwitz (1999), Hoffman (1999), Fortin (2000), Ly & Stamatatos(2000), Park (2000), Salzwedel (2000), Kolchinsky (2001), Verrier (2001)]

- 48d: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs
- 48d: Epitope is better exposed upon CD4 binding to gp120 – competes with ICR 39.13, 15e and 21h, anti-CD4 binding site MAbs – inhibited by anti-CD4BS MAb ICR 39.13g and linear anti-C4 MAbs G3-42 and G3-508 – 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 421 K/L, 433A/L, 438 P/R and 475 M/S confer decreased sensitivity to neutralization [Thali (1993)]

Table of HIV MAbs

- 48d: Called 4.8d – Neutralizes IIIB – reactive with SF-2 gp120 – does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- 48d: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding of 17b [Moore (1993c)]
- 48d: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 21h, 15e and 17b) [Thali (1994)]
- 48d: Poor cross-reactivity with gp120 from most clades [Moore (1994b)]
- 48d: Called 4.8D – Found to neutralize MN, but not JRCSF, two B subtype primary isolates, or a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs[D’Souza (1995)]
- 48d: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence of sCD4 involves the V1/V2 loops, with more significant involvement of V2 – similar effect observed for 17b and A32 [Wyatt (1995)]
- 48d: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau (1995)]
- 48d: Binds with similar affinity to monomer and oligomer, moderate association rate, potent neutralization – this is in contrast to 17b, which has very different kinetics [Sattentau & Moore(1995)]
- 48d: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) – anti-C1-C4 discontinuous epitope MAbs A32 and 2/11c enhance binding – reciprocal enhanced binding with some anti-V2 MAbs [Moore & Sodroski(1996)]
- 48d: Binding resulted in gp120 dissociation from virion, mimicking sCD4, and exposure of the gp41 epitope of MAb 50–69, in contrast to CD4BS MAbs [Poignard (1996a)]
- 48d: Neutralizes JR-FL – slightly inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola (1996a)]
- 48d: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – all Ab combinations tested showed synergistic neutralization – 48d has synergistic response with MAbs 694/98-D (anti-V3) and F105 [Li (1997)]
- 48d: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the Can0A V3 peptide, suggesting Can0A V3 is a conformer that mimics the 48d, (but not 17b), epitope [Weinberg (1997)]
- 48d: Prefers CD4-gp120 complex to gp120 alone, but does not enhance fusion, in contrast to MAb CG10, in fact it inhibits syncytium formation [Lee (1997)]
- 48d: Viral binding inhibition by 48d was strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- 48d: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt (1997)]
- 48d: Neutralizes TCLA strains, but not primary isolates [Parren (1997b)]
- 48d: Summary of the implications of the crystal structure of the core of gp120 bound to CD4 and 17b with what is known about mutations that reduce NAb binding – probable mechanism of neutralization of 48d is interference with chemokine receptor binding – CD4 binding increases exposure of epitope due to V2 loop movement – 88N, 117K, 121K, 256S, 257T, N262, delta V3, E370, E381, F 382, R 419, I 420, K 421, Q 422, I 423, W 427, Y 435, P 438, M 475 mutations in HXBc2 (IIIB) decrease binding [Wyatt (1998)]
- 48d: Inhibits binding of Hx10 to both CD4 positive and CD4 negative HeLa cells [Mondor (1998)]
- 48d: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

- 48d: CD4i MAbs 17b and 48d compete with MAb CG10, and the binding sites may overlap – MAb A32 enhances binding of 17b, 48d and CG10 [Sullivan (1998b)]
- 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 [Yang (1998)]
- 48d: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – CD4i MAbs 17b and 48d bound better to the deleted protein than to wild type [Binley (1998)]
- 48d: Deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V2 but not V1 enabled neutralization by CD4i MAbs 17b and 48d [Stamatatos & Cheng-Mayer(1998)]
- 48d: Infection of dendritic cells cultured from CD14+ blood cells or from cadaveric human skin was blocked by neutralizing MAbs IgG1b12, or 2F5 and 2G12 delivered together, but not by control non-neutralizing anti-gp120 MAb 4.8D, indicating that NABs could interrupt early mucosal transmission events [Frankel (1998)]
- 48d: A CD4-independent viral variant of IIIB, IIIBx, was generated on CXCR4-expressing cells – IIIBx exhibited greater exposure of the 17b and 48d epitopes and enhanced neutralization by CD4i MAbs and by polyclonal human sera [Hoffman (1999)]
- 48d: Called 4.8D – host encoded intercellular adhesion molecule (ICAM-1) is incorporated by the HIV-1 virion and enhances viral infectivity – ICAM-1 does not modify virus sensitivity to antibodies 0.5 β or 4.8D or sCD4, but neutralizing ability of F105 was diminished in ICAM bearing virions in the presence of lymphocyte function-association antigen-1 (LFA-1) Ab [Fortin (2000)]
- 48d: SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391-95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]
- 48d: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- 48d: sCD4 can activate fusion between effector cells expressing Env and target cells expressing coreceptor (CCR5 or CXCR4) alone without CD4 – CD4i MAbs 17b and 48d have little effect on a standard cell fusion assay but potently block sCD4 activated fusion [Salzwedel (2000)]
- 48d: Mutations in two glycosylation sites in the V2 region of HIV-1 ADA at positions 190 and 197 (187 DNTSYRLINCNTS 199) cause the virus to become CD4-independent and able to enter cells through CCR5 alone – these same mutations tended to increase the neutralization sensitivity of the virus, including to 48d – only the CD4i antibodies 17b and 48d showed an increased affinity of the CD4 independent viruses relative to wild-type [Kolchinsky (2001)]

Table of HIV MAbs

- 48d: Called 4.8d – A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 $\mu\text{g/ml}$: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 48d: NIH AIDS Research and Reference Reagent Program: 1756

786	A32	Env(dis)	gp120(dis)	no	HIV-1 infection	human(IgG1)
<p>Ab type: CD4i Donor: J. Robinson, Tulane University, New Orleans, LA, USA</p> <p>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)]</p> <ul style="list-style-type: none"> • A32: Reacted with virtually every gp120 monomer of every clade tested, most conserved gp120 monomer epitope known [Moore (1994b)] • A32: Epitope is better exposed upon CD4 binding to gp120 – binding of A32 enhances binding of 48d and 17b – studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 [Wyatt (1995)] • A32: Review: epitope is distinct from CD4BS MAbs, 48d and 17b, and 2G12 [Moore & Ho(1995)] • A32: Reciprocal inhibition of binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of some anti-V2 and sCD4 inducible MAbs (48d and 17b) – very similar competition pattern to 2/11c, A32 and 211/c are unique among known human and rodent MAbs [Moore & Sodroski(1996)] • A32: Not neutralizing – binds domains that interact with gp41 – MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 and binding of A32 does not block this inhibition [Wu (1996)] • A32: Does not neutralize JR-FL, or any strain strongly – partial inhibition of gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)] • A32: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – A32 bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)] • A32: Review [Burton & Montefiori(1997)] • A32: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt (1997)] • A32: Does not neutralize TCLA strains or primary isolates [Parren (1997b)] • A32: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – A32 has a unique epitope involving mostly C2 but C1 and C4 contribute – six quite variable phage inserts were recognized, with a consensus of LPWYN – a central Trp was the most conserved element, consistent with W427 being an important residue for binding gp120 [Boots (1997)] • A32: Enhances binding of CD4i MAbs 17b and 48d, and a MAb generated in response to gp120-CD4 complex CG10 [Sullivan (1998b)] • A32: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)] 						

- A32: 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 [Binley (1999)]

787	1367	Env(dis gp41 567–647)	gp41(dis)		HIV-1 infection	human(IgG1 λ)
<p>Ab type: cluster I Donor: Susan Zolla-Pazner (Zollas01@mcr6.med.nyu) (NYU Med. Center)</p> <p>References: [Nyambi (1998), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 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 [Nyambi (1998)] • 1367: This antibody binds to a cluster I epitope in gp41, 567–647, and recognizes a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41 – this MAb doesn't react with either of the peptides N51 or C43 individually – MAbs 50–69 and 1367 had similar properties [Gorny & Zolla-Pazner(2000)] • 1367: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)] • 1367: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 1367 weakly bound to the majority of isolates – no neutralizing activity was observed when tested with 5 isolates, but 1367 did not bind well to these isolates [Nyambi (2000)] 						
788	126-6 (SZ-126.6)	Env ()	gp41(HXB2)	no	HIV-1 infection	human(IgG2 κ)
<p>Ab type: cluster II Donor: Susan Zolla-Pazner (Zollas01@mcr6.med.nyu), NYU Med Center, NY, NY</p> <p>References: [Robinson (1990b), Robinson (1991), Xu (1991), Eddleston (1993), Chen (1995), Binley (1996), Earl (1997), Gorny & Zolla-Pazner(2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 126-6: No enhancing activity for HIV-1 IIIB [Robinson (1990b)] • 126-6: No enhancing or neutralizing activity [Robinson (1991)] • 126-6: Antibody is specific for a conformational epitope [Xu (1991)] • 126-6: Called SZ-126.6 [Eddleston (1993)] • 126-6: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen (1995)] 						

Table of HIV MAbs

- 126-6: Discontinuous epitope recognizing residues between 649–668 – designated cluster II – Fabs D5, D11, G1, T3, M12, M15, S6, S8, S9, S10 block binding [Binley (1996)]
- 126-6: This cluster II MAb binds to a conformational epitope in the region 644–663 – like most cluster II MAbs (126-6, 167-D, 1281, 1342, and 1379 all reacted similarly) it binds to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, but not to C43 nor to N51 alone – MAb 126-6 was biotinylated and used as a probe to determine that anti-gp41 MAb 50–69 bound the fusogenic form of the protein in liquid phase [Gorny & Zolla-Pazner(2000)]
- 126-6: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistently to cluster II MAbs [Nyambi (2000)]
- 126-6: NIH AIDS Research and Reference Reagent Program: 1243

789	1281	Env(dis gp41 647–682)	gp41(dis)		HIV-1 infection	human(IgG1 λ)
<p>Ab type: cluster II Donor: Susan Zolla-Pazner (Zollas01@mccrcr6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny & Zolla-Pazner(2000), Gorny (2000), Verrier (2001)]</p> <ul style="list-style-type: none"> • 1281: This cluster II MAb binds to a conformational epitope in the region 644–663 – like most cluster II MAbs (126-6, 167-D, 1281, 1342, and 1379 all reacted similarly) it binds to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, but not to C43 nor to N51 alone [Gorny & Zolla-Pazner(2000)] • 1281: Binds within the region gp41 647–682 – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)] • 246-D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 μg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)] 						
790	1342	Env(dis 647–682)	gp41(dis)	no	HIV-1 infection	human(IgG1 λ)
<p>Ab type: cluster II Donor: Susan Zolla-Pazner (Zollas01@mccrcr6.med.nyu) (NYU Med. Center)</p> <p>References: [Nyambi (1998), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 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 [Nyambi (1998)] • 1342: This cluster II MAb is a conformational epitope that binds in the region 644–663 – like most cluster II MAbs (126-6, 167-D, 1281, 1342, and 1379 all reacted similarly) it binds to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, but not to C43 nor to N51 alone [Gorny & Zolla-Pazner(2000)] • 1342: Binds within the region gp41 647–682 – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)] 						

- 1342: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistently to cluster II MAbs – no neutralizing activity was observed when tested with 5 isolates, but 1342 did not bind to these isolates [Nyambi (2000)]

791 1379	Env(dis gp41 647–682)	gp41(dis)		HIV-1 infection	human(IgG1 λ)
	<p>Ab type: cluster II Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Gorny & Zolla-Pazner(2000), Gorny (2000)]</p> <ul style="list-style-type: none"> • 1379: This cluster II MAb binds to a conformational epitope in the region 644–663 – like most cluster II MAbs (126-6, 167-D, 1281, 1342, and 1379 all reacted similarly) it binds to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, but not to C43 nor to N51 alone [Gorny & Zolla-Pazner(2000)] • 1379: Binds within the region gp41 647–682 – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)] 				
792 Fab D11	Env(dis)	gp41(dis LAI)	no	HIV-1 infection	human(IgG1 κ)
	<p>Ab type: cluster II References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab D11: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 				
793 Fab D5	Env(dis)	gp41(dis LAI)	no	HIV-1 infection	human(IgG1 κ)
	<p>Ab type: cluster II References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab D5: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 				
794 Fab G1	Env(dis)	gp41(dis LAI)	no	HIV-1 infection	human(IgG1 κ)
	<p>Ab type: cluster II References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab G1: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 				
795 Fab M10	Env(dis)	gp41(dis LAI)	no	HIV-1 infection	human(IgG1 κ)
	<p>Ab type: cluster II References: [Binley (1996), Parren (1997b)]</p> <ul style="list-style-type: none"> • Fab M10: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] • Fab M10: Does not bind to MN native oligomer, but does bind to both LAI and MN rgp120 and rgp140 [Parren (1997b)] 				
796 Fab M12	Env(dis)	gp41(dis LAI)	no	HIV-1 infection	human(IgG1 κ)
	<p>Ab type: cluster II References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab M12: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 				

Table of HIV MAbs

797	Fab M15	Env(dis) Ab type: cluster II	gp41(dis LAI) References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab M15: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 						
798	Fab S10	Env(dis) Ab type: cluster II	gp41(dis LAI) References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab S10: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 						
799	Fab S6	Env(dis) Ab type: cluster II	gp41(dis LAI) References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab S6: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 						
800	Fab S8	Env(dis) Ab type: cluster II	gp41(dis LAI) References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab S8: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 						
801	Fab S9	Env(dis) Ab type: cluster II	gp41(dis LAI) References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab S9: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 						
802	Fab T3	Env(dis) Ab type: cluster II	gp41(dis LAI) References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab T3: Binds to cluster II region – competes with MAbs 126-6, Md-1 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 						
803	Md-1 (MD-1)	Env(dis) Ab type: cluster II	gp41(dis) Donor: R. A. Myers State of Maryland Dept. of Health References: [Myers (1993), Chen (1995), Binley (1996)]	no		human(IgG1 λ)
<ul style="list-style-type: none"> • Md-1: Called MD-1 – discontinuous epitope that binds in the N-terminal region – reacts exclusively with oligomer [Myers (1993)] • 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 [Chen (1995)] • 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 [Binley (1996)] • Md-1: NIH AIDS Research and Reference Reagent Program: 1223 						

Table of HIV MAbs

804	Fab A9	Env(dis) gp41(dis LAI) Ab type: cluster III References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab A9: Binds to cluster III region – competes with MAb Md-1, but not MAbs 126-6 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 					
805	Fab G15	Env(dis) gp41(dis LAI) Ab type: cluster III References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab G15: Binds to cluster III region – competes with MAb Md-1, but not MAbs 126-6 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 					
806	Fab G5	Env(dis) gp41(dis LAI) Ab type: cluster III References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab G5: Binds to cluster III region – competes with MAb Md-1, but not MAbs 126-6 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 					
807	Fab L1	Env(dis) gp41(dis LAI) Ab type: cluster III References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab L1: Binds to cluster III region – competes with MAb Md-1, but not MAbs 126-6 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 					
808	Fab L11	Env(dis) gp41(dis LAI) Ab type: cluster III References: [Binley (1996)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab L11: Binds to cluster III region – competes with MAb Md-1, but not MAbs 126-6 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 					
809	Fab L2	Env(dis) gp41(dis LAI) Ab type: cluster III Donor: P. Perrin and D. Burton (Scripps Research Institute, La Jolla, California) References: [Binley (1996), Earl (1997)]	no	HIV-1 infection	human(IgG1 κ)
<ul style="list-style-type: none"> • Fab L2: Binds to cluster III region – competes with MAb Md-1, but not MAbs 126-6 and D50 – conformation sensitive – variable regions sequenced [Binley (1996)] 					
810	Chessie 8	Env() gp41() Ab type: cytoplasmic domain Donor: G. Lewis References: [Lewis (1991), Poubourios (1995), Rovinski (1995)]			murine(IgG)
<ul style="list-style-type: none"> • Chessie 8: Used to precipitate gp160 in immunoblots in a study examining the feasibility of using unprocessed gp160 glycoprotein as an immunogen [Rovinski (1995)] 					
811	T22	Env(dis) gp120(dis IIIB) Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140 Ab type: Env oligomer Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD		Vaccine	murine(IgG)

Table of HIV MAbs

References: [Earl (1994), Otteken (1996), Sugiura (1999)]

- T22: Pulse label experiments of 4 MAbs (D20, D27, T20, and T22) binding to noncleavable gp140 revealed that these anti-CD4BS MAbs bound with a delay, and that the epitope formed with a $t_{1/2}$ of about 10 minutes [Otteken (1996)]
- T22: A 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 block CD4 binding [Sugiura (1999)]

812	8F101	Env(dis)	gp120(dis)		Vaccine	murine(IgG)
		Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120				
		Ab type: gp120-CD4 complex References: [DeVico (1995)]				
		<ul style="list-style-type: none"> • 8F101: MAbs specifically reactive to crosslinked gp120 and CD4 were derived (8F101, 8F102) – conformation dependent – competition studies indicate the epitope is immunogenic in infected humans [DeVico (1995)] 				
813	8F102	Env(dis)	gp120(dis)		Vaccine	murine(IgG)
		Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> HXB2 <i>HIV component:</i> gp120				
		Ab type: gp120-CD4 complex References: [DeVico (1995)]				
		<ul style="list-style-type: none"> • 8F102: MAbs specifically reactive to crosslinked gp120 and CD4 were derived (8F101, 8F102) – conformation dependent – competition studies indicate the epitope is immunogenic in infected humans [DeVico (1995)] 				
814	CG-10 (CG10)	Env(dis)	gp120(dis IIIB)	L	Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>Strain:</i> IIIB <i>HIV component:</i> gp120				
		Ab type: gp120-CD4 complex Donor: Jonathan Gershoni, Tel Aviv University, Isreal				
		References: [Gershoni (1993), Wu (1996), Lee (1997), Rizzuto (1998), Sullivan (1998b), Oscherwitz (1999)]				
		<ul style="list-style-type: none"> • CG-10: Reacts exclusively with sCD4-gp120 complex, not with sCD4 or gp120 alone [Gershoni (1993)] • CG-10: Called CG10 – MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4, and MAb CG10 does not block this inhibition [Wu (1996)] • 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 [Lee (1997)] • 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 70% reduction in CG10 binding [Rizzuto (1998)] 				

- CG-10: Called CG10 – CD4BS MAb 15e competes with CG-10 binding, probably due to the disruption of CD4-gp120 by 15e – CD4i MAbs 17b and 48d compete and the binding sites may overlap – MAb A32 enhances binding of 17b, 48d and CG10 – MAbs C11, 2G12 and 212A do not affect CG10 binding – CG-10 can bind gp120 with V1/V2 and V3 deleted – HXBc2 mutations Δ 119–205, 314 G/W, 432 K/A, 183,184 PI/SG decrease CG-10 recognition, HXBc2 mutations Δ 298–327 (V3), 384 Y/E, 298 R/G, 435 Y/S enhance recognition – the CD4 contribution to the CG10 epitope maps to the CD4 CDR2-like loop – CG10 can neutralize HIV-1 in the presence of sCD4 even though it does not do so in the context of cell surface CD4 binding to gp120 [Sullivan (1998b)]

815	CG-25	Env(dis)	gp120(dis)	L	Vaccine	murine(IgG1)
	Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>HIV component:</i> gp120					
	Ab type: gp120-CD4 complex References: [Gershoni (1993)]					
	● CG-25: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 [Gershoni (1993)]					
816	CG-4 (CG4)	Env(dis)	gp120(dis)	no	Vaccine	murine(IgG1)
	Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>HIV component:</i> gp120					
	Ab type: gp120-CD4 complex Donor: Jonathan Gershoni, Tel Aviv University, Isreal					
	References: [Gershoni (1993)]					
	● CG-4: Reacts with gp120 and sCD4-gp120 complex, not with sCD4 [Gershoni (1993)]					
817	CG-76	Env(dis)	gp120(dis)	L	Vaccine	murine(IgG1)
	Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>HIV component:</i> gp120					
	Ab type: gp120-CD4 complex References: [Gershoni (1993)]					
	● CG-76: Reacts equally well with sCD4-gp120 and sCD4, but not with purified gp120 [Gershoni (1993)]					
818	CG-9	Env(dis)	gp120(dis)	L	Vaccine	murine(IgG1)
	Vaccine: <i>Vector/type:</i> sCD4-gp120 complex <i>HIV component:</i> gp120					
	Ab type: gp120-CD4 complex References: [Gershoni (1993)]					
	● CG-9: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 [Gershoni (1993)]					
819	NC-1	Env(dis)	gp41(dis IIIB)		Vaccine	murine(IgG2a)
	Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> IIIB <i>HIV component:</i> a peptide that folds into a six helix bundle like gp41					
	Ab type: helical core 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)]					

Table of HIV MAbs

820	105–518	Env()	gp41(608–637 HAM112, O group)		Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HAM112 (group O) <i>HIV component:</i> gp160 Ab type: immunodominant region References: [Scheffel (1999)] <ul style="list-style-type: none"> • 101–518: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity [Scheffel (1999)] </p>						
821	2A2	Env()	gp41()	no	HIV-1 infection	human(IgG1 κ)
<p>Ab type: N-term References: [Weissenhorn (1996)] <ul style="list-style-type: none"> • Soluble gp41(21–166) forms a rod like structure that can be visualized with electron microscopy, and 2A2 binds to one end of the rod [Weissenhorn (1996)] </p>						
822	AC4	Env(dis 1–204)	gp120(dis IIIB)	yes	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> gp160 Ab type: N-term References: [Dickey (2000)] <ul style="list-style-type: none"> • AC4: Three MABs, ID6, AC4, and AD3 that bind to a discontinuous N-term first 204 aa of gp120 and generate ADCC were elicited through vaccination of BALBc mice with rec gp160 – these MABs do not depend on glycosylation and are cross-reactive with viruses from clades B and CRF01(AE) [Dickey (2000)] </p>						
823	AD3	Env(dis 1–204)	gp120(dis IIIB)	yes	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> gp160 Ab type: N-term References: [Dickey (2000)] <ul style="list-style-type: none"> • AD3: There may be two Abs with this name that bind to the N-term region of gp120 [Cook (1994), Dickey (2000)] • AD3: Three MABs, ID6, AC4, and AD3 that bind to a discontinuous N-term first 204 aa of gp120 and generate ADCC were elicited through vaccination of BALBc mice with rec gp160 – these MABs do not depend on glycosylation and are cross-reactive with viruses from clades B and CRF01(AE) [Dickey (2000)] </p>						
824	AD3	Env(dis 1–193)	gp120(dis BH10)			murine(IgG1)
<p>Ab type: N-term References: [Ugen (1993), Cook (1994)] <ul style="list-style-type: none"> • AD3: There may be two Abs with this name that bind to the N-term region of gp120 [Cook (1994), Dickey (2000)] • AD3: MABs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MABs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAB binding [Cook (1994)] • AD3: NIH AIDS Research and Reference Reagent Program: 2342 </p>						
825	ID6	Env()	gp120(1–193 BH10) ined amino terminus			murine(IgG1)
<p>Ab type: N-term References: [Ugen (1993), Cook (1994)] <ul style="list-style-type: none"> • ID6: There may be two Abs with this name that bind to the N-term region of gp120 [Cook (1994), Dickey (2000)] • ID6: MABs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MABs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAB binding [Cook (1994)] </p>						

- ID6: NIH AIDS Research and Reference Reagent Program: 2343

826	ID6	Env(dis 1–204)	gp120(dis IIIB)	yes	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> gp160</p> <p>Ab type: N-term References: [Dickey (2000)]</p> <ul style="list-style-type: none"> • ID6: There may be two Abs with this name that bind to the N-term region of gp120 [Cook (1994), Dickey (2000)] • ID6: Three MABs, ID6, AC4, and AD3 that bind to a discontinuous N-term first 204 aa of gp120 and generate ADCC were elicited through vaccination of BALBc mice with rec gp160 – these MABs do not depend on glycosylation and are cross-reactive with viruses from clades B and CRF01(AE) [Dickey (2000)] 						
827	31A1	Env()	gp41()	no	<i>in vitro</i> stimulation	human(IgM κ / λ)
<p>Ab type: p24+gp41 References: [Pollock (1989)]</p> <ul style="list-style-type: none"> • 31A1: Denatured virus was used for <i>in vitro</i> stimulation to generate Abs – Reacts with both p24 and gp41 [Pollock (1989)] 						
828	39A64	Env()	gp41()	no	<i>in vitro</i> stimulation	human(IgM κ / λ)
<p>Ab type: p24+gp41 References: [Pollock (1989)]</p> <ul style="list-style-type: none"> • 39A64: Denatured virus was used for <i>in vitro</i> stimulation to generate Abs – Reacts with both p24 and gp41 [Pollock (1989)] 						
829	39B86	Env()	gp41()	no	<i>in vitro</i> stimulation	human(IgM κ / λ)
<p>Ab type: p24+gp41 References: [Pollock (1989)]</p> <ul style="list-style-type: none"> • 39B86: Denatured virus was used for <i>in vitro</i> stimulation to generate Abs – Reacts with both p24 and gp41 [Pollock (1989)] 						
830	9303	Env()	gp41()	no		murine()
<p>Ab type: p24+gp41 Donor: Du Pont</p> <p>References: [McDougal (1996)]</p>						
831	polyclonal	Env(dis)	Env(dis)	yes	HIV-1 infection	human()
<p>Ab type: V1-V2 and V3-V5 References: [Gordon & Delwart(2000)]</p> <ul style="list-style-type: none"> • Primary isolates have great differences in susceptibility to neutralization – the variation in V1V2 and V3-V5 was measured by HTA in a set of viruses with a range of neutralization susceptibilities, and greater variability was uncorrelated with resistance to neutralization [Gordon & Delwart(2000)] 						
832	11/68b	Env(dis)	gp120(dis)	L (HXB2)	Vaccine	rat(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>Ab type: V1V2 Donor: Shotton and Dean</p> <p>References: [McKeating (1993b), Shotton (1995), Peet (1998)]</p> <ul style="list-style-type: none"> • 11/68b: Changes at residues 183/184 (PI/SG) within V2, 435 (Y/H) in C4, abrogate binding [McKeating (1993b)] • 11/68b: 435 (Y/H) in C4 does not abrogate binding (John Moore, per comm, 1996) • 11/68b: Cross-competes with MABs 62c, 66c, 66a, and CRA-4 – similar to MAb 62c – HXB2 neutralization escape mutant had a D/N substitution at residue 185 – non-reciprocal inhibition of binding of CRA-3 and CRA-6 [Shotton (1995)] 						

Table of HIV MAbs

- 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 wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 11/68b: UK Medical Research Council AIDS reagent: ARP3041

833	62c	Env(dis)	gp120(dis)	no	Vaccine	rat(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>Ab type: V1V2 References: [Shotton (1995)]</p> <ul style="list-style-type: none"> • 62c: Cross-competes with MAbs 11/68b, 66c, 66a, and CRA-4 – same cross-competition group as MAb 11/68b – non-reciprocal inhibition of binding of CRA-3 and CRA-6 – substitutions 176–177 FY/AT, 179–180 LD/DL, 183–184 PI/SG, and 191–193 YSL/GSS abrogate binding – binds but does not neutralize Hx10 [Shotton (1995)] • 62c: UK Medical Research Council AIDS reagent: ARP3075 						
834	CRA-6 (CRA6)	Env(dis)	gp120(dis)	no		murine()
<p>Ab type: V1V2 References: [Shotton (1995)]</p> <ul style="list-style-type: none"> • CRA-6: Called CRA6 – same competition group as CRA-3 [Shotton (1995)] 						
835	L15	Env(dis)	gp120(dis)	P (weak)	HIV-1 infection	human(IgG1)
<p>Ab type: V1V2 References: [Ditzel (1997), Parren (1997b)]</p> <ul style="list-style-type: none"> • L15: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – 2 anti-V2 Fabs were obtained with very similar epitopes, L15 and L17 – deletions in V1 and V2 abolished binding, and rodent anti-V2 MAbs SC258, CRA3, G3-G4,G3-136, BAT-085, and 52–684 all compete with L15 [Ditzel (1997)] • L15: Does not neutralize TCLA strains but neutralizes some primary isolates weakly [Parren (1997b)] 						
836	T52	Env(dis)	gp120(dis IIIB)		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: V1V2 Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • T52: A 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 [Sugiura (1999)] 						
837	T54	Env(dis)	gp120(dis IIIB)	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: V1V2 Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p>						

- T54: A 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 [Sugiura (1999)]

838	1088	Env()	gp120()			()
<p>Ab type: V2 References: [Berman (1997)]</p> <ul style="list-style-type: none"> • 1088: Binds weakly to 2/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)] 						
839	110-B	Env(dis)	gp120(dis)	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> infected-cell lysate <i>Strain:</i> BRU <i>HIV component:</i> virus</p> <p>Ab type: V2 Donor: Hybridolabs, Institute Pasteur, Paris, France</p> <p>References: [Moore (1993a)]</p> <ul style="list-style-type: none"> • 110-B: specific for BH10, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 168 K/L, 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192–194 YSL/GSS [Moore (1993a)] 						
840	1357	Env()	gp120()			human(IgG1κ)
<p>Ab type: V2 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Nyambi (1998), Gorny (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 1357: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 anti-V2 MAbs, which showed weak and sporadic binding, with the most frequent binding to C and D clades [Nyambi (2000)] • 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 [Nyambi (1998)] • 1357: Blocks binding of MAb 697-D to rgp120, and doesn't react with a protein from which V1V2 has been deleted – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V2 MAbs 697-D, 1357 and 1361 favored the monomer by approximately 2 fold [Gorny (2000)] • 1357: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 anti-V2 MAbs, which showed weak and sporadic binding, with the most frequent binding to C and D clades [Nyambi (2000)] 						
841	1361	Env()	gp120()		Vaccine	human(IgG1κ)
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> gp120</p> <p>Ab type: V2 Donor: Susan Zolla-Pazner (Zollas01@mrcrc6.med.nyu) (NYU Med. Center)</p> <p>References: [Nyambi (1998), Gorny (2000), Nyambi (2000)]</p> <ul style="list-style-type: none"> • 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 [Nyambi (1998)] 						

Table of HIV MAbs

- 1361: Blocks binding of MAb 697-D to gp120, and doesn't react with a protein from which V1V2 has been deleted – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V2 MAbs 697-D, 1357 and 1361 favored the monomer by approximately 2 fold [Gorny (2000)]
- 1361: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 anti-V2 MAbs, which showed weak and sporadic binding, with the most frequent binding to C and D clades [Nyambi (2000)]

842	1393A	Env() gp120()			HIV-1 infection	()
		Ab type: V2 References: [Nyambi (2000)]				
		<ul style="list-style-type: none"> • 1393A: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 anti-V2 MAbs, which showed weak and sporadic binding, with the most frequent binding to C and D clades [Nyambi (2000)] 				
843	66a	Env(dis) gp120(dis)		L (HXB2)	Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120				
		Ab type: V2 References: [Shotton (1995)]				
		<ul style="list-style-type: none"> • 66a: Substitutions 176–177 FY/AT, 179–180 LD/DL, 183–184 PI/SG, and 191–193 YSL/GSS abrogate binding – same competition group as CRA4 [Shotton (1995)] • 66a: UK Medical Research Council AIDS reagent: ARP3074 				
844	66c	Env(dis) gp120(dis)		L (HXB2)	Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120				
		Ab type: V2 References: [Shotton (1995)]				
		<ul style="list-style-type: none"> • 66c: Substitutions 176–177 FY/AT, 179–180 LD/DL, 183–184 PI/SG, and 191–193 YSL/GSS abrogate binding – same competition group as CRA4 [Shotton (1995)] 				
845	684–238 (52–684- 238, 52– 684)	Env(dis) gp120(dis)		L	Vaccine	murine()
		Vaccine: <i>Vector/type:</i> purified protein <i>Strain:</i> IIIB <i>HIV component:</i> gp120				
		Ab type: V2 Donor: Gerry Robey, Abbott Laboratories				
		References: [Moore (1993a), Thali (1993), Gorny (1994), Ditzel (1995), Moore & Sodroski(1996), Ditzel (1997)]				
		<ul style="list-style-type: none"> • 684–238: Specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – neutralizes BH10 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177FY/AT, 179/180LD/DL, 183/184PI/SG, and 192–194YSL/GSS [Moore (1993a)] • 684–238: Weakly neutralizing, IC 50 = 84 µg/ml [Gorny (1994)] • 684–238: Does not compete with IgG1b12, reciprocal inhibition with MAbs L39, L40, and L78 [Ditzel (1995)] • 684–238: Limited reciprocal enhancement of binding with anti-V3 and C4 region antibodies – reciprocal inhibition with V2 region antibodies [Moore & Sodroski(1996)] 				

846	830A	Env() Ab type: V2	gp120() References: [Nyambi (2000)]		HIV-1 infection	()
<ul style="list-style-type: none"> ● 830A: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 anti-V2 MAbs, which showed weak and sporadic binding, with the most frequent binding to C and D clades [Nyambi (2000)] 						
847	CRA-3 (CRA3)	Env(dis)	gp120(dis)	no	Vaccine	murine(IgG2a)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>Ab type: V2 Donor: Mark Page, NIBSC AIDS reagent project, Potters Bar, Herts, UK</p> <p>References: [Moore & Ho(1993), Moore (1993a), Thali (1993), Shotton (1995), Moore & Sodroski(1996), Ditzel (1997)]</p> <ul style="list-style-type: none"> ● CRA-3: Conformational, does not bind well to denatured gp120 [Moore & Ho(1993)] ● CRA-3: specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192–194 YSL/GSS – epitope probably involves stem of V1/V2 loop structure [Moore (1993a)] ● CRA-3: Many MAbs enhance binding, including some anti-C5, C1, V4, and C4 MAbs – enhances binding of only a small number of anti-V3 loop MAbs [Moore & Sodroski(1996)] ● CRA-3: Called CRA3 – Same competition group as CRA6 [Shotton (1995)] ● CRA-3: UK Medical Research Council AIDS reagent: ARP324 						
848	CRA-4 (CRA4)	Env(dis)	gp120(dis)	L (HXB2)	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120</p> <p>Ab type: V2 Donor: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325</p> <p>References: [McKeating (1993b), Moore & Ho(1993), Moore (1993a), Thali (1993), Shotton (1995), Moore & Sodroski(1996)]</p> <ul style="list-style-type: none"> ● CRA-4: Changes at residues 191/192/193 (YSL/GSS) within V2, 435 (Y/H) in C4, abrogate binding – type-specific neutralization [McKeating (1993b)] ● CRA-4: Conformational, does not bind well to denatured gp120 [Moore & Ho(1993)] ● CRA-4: Specific for BH10 and HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192–194 YSL/GSS [Moore (1993a)] ● CRA-4: Cross-competes with MAbs 11/68b, 62c, 66c, 66a – similar to 66c and 66a – non-reciprocal inhibition by MAbs 12b, 60b and CRA-6 [Shotton (1995)] ● CRA-4: The only MAbs that enhanced binding were anti-V3 MAb 5G11 and anti-C1 MAb 135/9 binding – reciprocal inhibition of anti-V2 MAbs [Moore & Sodroski(1996)] ● CRA-4: UK Medical Research Council AIDS reagent: ARP325 						

Table of HIV MAbs

849	L17	Env(dis) Ab type: V2	gp120(dis) References: [Ditzel (1997), Parren (1998a)]			human Fab()
<ul style="list-style-type: none"> • L17: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142–10 > DA48 > L17) was markedly different than Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142–10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)] 						
850	SC258 (52–581-SC258)	Env(dis) Vaccine: <i>Vector/type:</i> purified protein	gp120(dis) <i>Strain:</i> IIIIB <i>HIV component:</i> gp120	L	Vaccine	murine()
<p>Ab type: V2 Donor: Gerry Robey, Abbott Laboratories</p> <p>References: [Moore (1993a), Thali (1993), Gorny (1994), Yoshiyama (1994), Moore (1994b), Ditzel (1995), Moore & Sodroski(1996), Trkola (1996a), Ditzel (1997)]</p> <ul style="list-style-type: none"> • 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 [Moore (1993a)] • SC258: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity – 177 Y/H inhibits SC258 neutralization [Yoshiyama (1994)] • SC258: Very poor reactivity with gp120 molecules outside of clade B [Moore (1994b)] • SC258: Does not compete with IgG1b12 – reciprocal inhibition with MAbs L39, L40, and L78 [Ditzel (1995)] • SC258: Several MAbs binding to various gp120 epitopes enhance binding, but the only MAb that SC258 enhanced binding of was anti-CD4 binding site MAb F91 – reciprocal inhibition with V2 region antibodies [Moore & Sodroski(1996)] • SC258: Does not inhibit gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study – listed as not neutralizing [Trkola (1996a)] 						
851	L25	Env(dis) Ab type: V2-CD4BS	gp120(dis) References: [Ditzel (1995), Ditzel (1997), Parren (1997b)]	L (weak)	HIV-1 infection	human(IgG1)
<ul style="list-style-type: none"> • L25: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – a single anti-V2-CD4 BS Fab was obtained with with sensitivity to substitutions in the V2 and CD4 BS regions – rodent anti-V2 MAb SC258 competes with L25 [Ditzel (1997)] • L25: Neutralizes TCLA strains weakly, but not primary isolates [Parren (1997b)] 						
852	L39	Env(dis) Ab type: V2-CD4BS	gp120(dis) References: [Ditzel (1995)]	no	HIV-1 infection	human(IgG1 κ)

						<ul style="list-style-type: none"> • L39: This Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L39 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684–238 – heavy and light chain variable region sequence is available [Ditzel (1995)] 	
853	L40	Env(dis)	gp120(dis)	no	HIV-1 infection	human(IgG1κ)	<p>Ab type: V2-CD4BS References: [Ditzel (1995)]</p> <ul style="list-style-type: none"> • L40: This Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L40 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding only partially affected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684–238 – heavy and light chain variable region sequence is available [Ditzel (1995)]
854	L78	Env(dis)	gp120(dis)	L	HIV-1 infection	human(IgG1κ)	<p>Ab type: V2-CD4BS References: [Ditzel (1995)]</p> <ul style="list-style-type: none"> • L78: Substitutions at V2: (152/153 GE/SM, 183/184 PI/SG, 191/193 YL/GS), 262 N/T, V3 (314 G/W), CD4BS (257 T/R, 368 D/R, 370 E/R) inhibit binding, and some C4 and C5 substitutions enhance binding – this Fab does not inhibit sCD4 binding, but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – Fab neutralizes MN and LAI – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684–238 – heavy and light chain variable region sequence is available [Ditzel (1995)]
855	110.J	Env()	gp120()			()	<p>Ab type: V3 Donor: F. Traincard, Pasteur Institute, France References: [Thali (1993), Moore & Sodroski(1996)]</p> <ul style="list-style-type: none"> • 110.J: Inhibits sCD4-inducible anti-CD4 binding site MAb 48d [Thali (1993)] • 110.J: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs [Moore & Sodroski(1996)]
856	1334-D (1334, 1334D)	Env()	gp120(HIV451)		HIV-1 infection	human(IgG1κ)	<p>Ab type: V3 Donor: Susan Zolla-Pazner (Zollas01@mrcrcr6.med.nyu) (NYU Med. Center) References: [Zolla-Pazner (1999a), Zolla-Pazner (1999b), Gorny (2000)]</p> <ul style="list-style-type: none"> • 1334-D: This MAb was selected on oligomeric gp160 from HIV451 [Zolla-Pazner (1999a)] • 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-Pazner (1999b)]

Table of HIV MAbs

- 1334-D: Called 1334 – binds to V3 peptides from MN, SF2, NY5, RF, and CDC4 strains as well as x-reactivity with peptides from A, C, D, F, G, and H subtypes – was suggested to be IgG1lambda here – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V3 MAbs 447-52D, 838-D, and 1334 bound with a 7–10 fold preference for the oligomer [Gorny (2000)]
- 1334-D: Called 1334D – A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MAbs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 1334D showed intermediate cross-reactivity [Nyambi (2000)]

857 55/68b	Env() Ab type: V3 References: [Peet (1998)]	gp120(300–315)		()
	<ul style="list-style-type: none"> • 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 wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)] 			
858 5G11	Env() Ab type: V3 References: [Moore & Sodroski(1996)]	gp120()		()
	<ul style="list-style-type: none"> • 5G11: Binds to conformation sensitive epitope in the V3 loop – reciprocal inhibition of other V3 loop MAbs – reciprocal enhancement of some C1-C5 MAbs (unusual for an anti-V3 MAb) and CD4 binding site MAbs – and enhances binding of V2 MAbs [Moore & Sodroski(1996)] 			
859 9305	Env() Ab type: V3 References: [McDougal (1996)]	gp120()	L	murine()
860 AG1121 (1121)	Env() Ab type: V3 References: [Sullivan (1995), Cao (1997)]	gp120()	L	()
	<ul style="list-style-type: none"> • AG1121: Recognizes monomeric gp120 from T-cell adapted line HXBc2 and primary isolate 89.6 equally well, but 89.6 was three-fold less sensitive to neutralization by AG1121 than HXBc2 [Sullivan (1995)] • AG1121: Called 1121 – Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao (1997)] 			

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861	anti-gp120/V3	Env()	gp120()	Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein, virus-like particle <i>Strain:</i> A clade 94UG018 <i>HIV component:</i> Gag, Pol, Nef, gp120 Ab type: V3 Donor: Intracel Co References: [Buonaguro (2001)]			
		<ul style="list-style-type: none"> • Anti-V3: HIV-1 pr55 gag-based virus-like particles (VLP) carrying Nef and Pol open reading frames as well as gp120 of the clade A isolate 94UG018 were created using a Baculovirus expression system to package additional ORFS into the VLP – anti-V3 and anti-p24 antibodies were used to assess the expression levels and Gag and gp120-TM were found to be expressed at comparable levels on the VLP 			
862	D47	Env()	gp120(IIIB)	Vaccine	murine()
	Vaccine:	<i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> Env Ab type: V3 Donor: Patricia Earl, NIAID, NIH References: [Earl (1994), Richardson (1996), Otteken (1996), Wyatt (1997), Earl (1997), Salzwedel (2000)]			
		<ul style="list-style-type: none"> • D47: Used for capture of oligomeric Env for antigen capture ELISA – binding of this antibody to oligomeric Env IIIB was not blocked by human sera from the US, consistent with a low prevalence of IIIB-like V3 strains [Richardson (1996)] • D47: Pulse label experiments of MAb binding to noncleavable gp160 revealed that this anti-V3 MAb bound immediately and binding stayed constant through chase period [Otteken (1996)] • D47: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)] • D47: Used for comparison in a study of gp41 antibodies – D47 binds to a greater extent to cell surface expressed Env than any of 38 conformation dependent anti-gp41 MAbs [Earl (1997)] • D47: sCD4 can activate fusion between effector cells expressing Env and target cells expressing coreceptor (CCR5 or CXCR4) alone without CD4 – V3 MAb D47 is strain specific and can inhibit sCD4 mediated infection, but only of the closely related LAV Env, while anti-CD4i MAbs were broadly cross-neutralizing [Salzwedel (2000)] 			
863	F5.5	Env()	gp120(IIIB)		murine()
		Ab type: V3 Donor: Hybridolabs, Institute Pasteur References: [Altmeyer (1999)]			
		<ul style="list-style-type: none"> • 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 [Altmeyer (1999)] 			

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864	G3-1472	Env() Ab type: V3 References: [Moore & Sodroski(1996)]	gp120() Donor: M. Fung References: [Moore & Sodroski(1996)]			()
<ul style="list-style-type: none"> • G3-1472: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs – binding inhibited by anti-C4 MAbs [Moore & Sodroski(1996)] 						
865	K24	Env() Ab type: V3 References: [Altmeyer (1999)]	gp120(IIIB) Donor: Hybridolabs, Institute Pasteur References: [Altmeyer (1999)]			murine()
<ul style="list-style-type: none"> • 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 [Altmeyer (1999)] 						
866	M096/V3	Env(dis 309–318 + 329–338) Ab type: V3 References: [Ohlin (1992)]	gp120(dis 309–318) References: [Ohlin (1992)]	IQRGPGRAV + AHCNISRAKW	<i>in vitro</i> stimulation	human(IgM)
<ul style="list-style-type: none"> • M096: Generated in response to IIIB Env 286–467 upon <i>in vitro</i> stimulation of uninfected-donor lymphocytes, and binds to two peptides: 309–318 + 329–338 [Ohlin (1992)] 						
867	polyclonal	Env() Vaccine: <i>Vector/type:</i> canarypox prime with recombinant protein boost MN, gp41 LAI, Gag LAI, partial Pol LAI, rgp120 SF2 Ab type: V3 References: [Verrier (2000)]	gp120() References: [Verrier (2000)]	yes <i>Strain:</i> MN, SF2, LAI <i>Stimulatory Agents:</i> MF59	Vaccine <i>HIV component:</i> gp120	human()
<ul style="list-style-type: none"> • Serum Abs elicited by this vaccine reacted with V3 peptides from clades B, C, and F, reacted weakly with V3 peptides from clades A, D, G, and H, and did not react with V3 peptides from clades E and O – neutralizing activity against 5 of 14 primary isolates tested was observed, including one B clade X4 virus, two dual-tropic B clade viruses, and one clade B and one clade C R5 virus [Verrier (2000)] 						
868	polyclonal	Env() Ab type: V3 References: [Sidorova(1999)]	gp120(303–325) References: [Sidorova(1999)]	no	<i>in vitro</i> stimulation	human(IgM)
<ul style="list-style-type: none"> • Polyspecific anti-MN-24 antibodies were raised through V3 peptide, MN-24 stimulation of human cells, followed by EBV transformation: they react with homologous and heterologous peptides and may be autoantibodies [Sidorova(1999)] 						
869	TH1	Env() Ab type: V3 References: [D'Souza (1995), Yang (1998)]	gp120() Donor: Michael Fung, Tanox Biosystem, USA References: [D'Souza (1995), Yang (1998)]	L (MN,JRCSF)		human(IgG1λ)

- TH1: Found to neutralize MN and JRCSF, but not two B subtype primary isolates, nor a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs [D'Souza (1995)]
- 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 [Yang (1998)]

870	11/75a/21/41	Env(dis)	gp120(dis)			()
		Ab type: V3 discontinuous		References: [McKeating (1992a), Peet (1998)]		
		<ul style="list-style-type: none"> • 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 wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)] 				
871	41.1 (ICR41.1i, ICR41)	Env(dis)	gp120(dis HXB10)	L (HXB2)	Vaccine	rat(IgG2a)
		Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> gp120				
		Ab type: V3 discontinuous 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)]				
		<ul style="list-style-type: none"> • 41.1: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 41.1 is not affected [Reitz (1988), Klasse (1993a)] • 41.1: Called ICR41.1i – Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – neutralization mediated by 3 molecules of IgG per virion – most efficient at neutralization of the three MAbs studied – acts with multi-hit kinetics [McLain & Dimmock(1994)] • 41.1: Called ICR41.1i – IgG2c? – Neutralization was affected if the Ab was added after the virus bound to the host cells at 24 degrees C or below [Armstrong & Dimmock(1996)] • 41.1: Called ICR41.1i – Neutralization occurs by blocking a post-fusion internalization event, in contrast to MAb F58 [Armstrong (1996)] • 41.1: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs (1996)] • 41.1: Viral binding inhibition by 41.1 was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)] 				
872	55/45a/11	Env(dis)	gp120(dis)			()
		Ab type: V3 discontinuous		References: [Peet (1998)]		
		<ul style="list-style-type: none"> • 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 wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)] 				

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873	1108	Env()	Env()		HIV-1 infection	human(IgG1λ)
<p>Ab type: V3 mimotype References: [Zolla-Pazner (1999a), Zolla-Pazner (1999b)]</p> <ul style="list-style-type: none"> • 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-Pazner (1999b)] • 1108: The sequence of peptide 987, used to select MAb 1108, is ADGAWRSVHLGPGRGSGSGMGK [Zolla-Pazner (1999a)] 						
874	MO101/V3,C4	Env(dis 314–323 + 494–503)	gp120(dis 314–323)	GRAFVTIGKI + LGVAPTKAKR	<i>in vitro</i> stimulation	human(IgM)
<p>Ab type: V3-C4 References: [Ohlin (1992)]</p> <ul style="list-style-type: none"> • MO101: Generated in response to IIIB Env 286–467 upon <i>in vitro</i> stimulation of uninfected-donor lymphocytes – reacts with peptides 314–323 + 494–503 from the V3 and C4 regions [Ohlin (1992)] 						
875	D27	Env(dis)	gp120(dis IIIB)		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: V3-CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Otteken (1996), Sugiura (1999)]</p> <ul style="list-style-type: none"> • 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 [Otteken (1996)] • D27: A 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 [Sugiura (1999)] 						
876	D56	Env(dis)	gp120(dis IIIB)	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Ab type: V3-CD4BS Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • D56: A 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 μg/ml of D56 was required to achieve 50% neutralization of HIV-1 NL4–3 [Sugiura (1999)] 						
877	polyclonal	Env()	gp120(IIIB)		Vaccine	rabbit()
<p>Vaccine: <i>Vector/type:</i> peptide <i>Strain:</i> MN <i>HIV component:</i> gp120 V3/C4 <i>Stimulatory Agents:</i> mucosal adjuvant CT</p> <p>Ab type: V3C4 References: [Zinckgraf (1999)]</p> <ul style="list-style-type: none"> • Nasal mucosal immunization and boosting of HIV peptide and was superior for inducing serum IgG and vaginal secretory IgA compared to nasal immunization and vaginal boosting – vaginal immunization and boosting resulted low serum IgG and vaginal IgA and a high vaginal IgG response [Zinckgraf (1999)] 						

878 2G12
(c2G12)

Env(dis)

gp120(dis)

L P HIV-1 infection

human(IgG1 κ)

Ab type: V3V4, carbohydrates **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), Parren (1999), Mascola (1999), Mascola (2000), Binley (1999), Baba (2000), Grovit-Ferbas (2000), Park (2000), Mascola & Nabel(2001), Zwick (2001c), Barnett (2001), Moore (2001), Poignard (2001), Zeder-Lutz (2001), Verrier (2001), Stiegler (2001), Spenlehauer (2001), Hofmann-Lehmann (2001), Xu (2001), Savarino (2001), Armbruster (2002)]

- 2G12: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells [Buchacher (1994)]
- 2G12: Highly potent Cross-clade neutralizing activity [Trkola (1995)]
- 2G12: Conformationally sensitive epitope destroyed by mutations altering the N-linked glycosylation sites near the base of the V3 loop and the amino-terminal flank of the V4 loop [Trkola (1996b)]
- 2G12: Binding weakly enhanced by some anti-C1, -C4, -V3, and CD4 binding site MABs – unusual in that 2G12 binding neither enhanced or inhibited the binding of other MABs included in the study [Moore & Sodroski(1996)]
- 2G12: Review: binding site is distinct from CD4BS MABs epitope and is unique among known gp120 MABs, human or rodent [Moore & Ho(1995)]
- 2G12: Review: exceptional capacity to neutralize primary isolates in terms of both breadth and potency – one of three MABs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard (1996b)]
- 2G12: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola (1996a)]
- 2G12: Neutralizes primary isolates, HXB2, and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]
- 2G12: Review: Only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MABs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]
- 2G12: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 μ g per ml for 90% viral inhibition – neutralized 6 of 9 primary isolates [D'Souza (1997)]
- 2G12: A JRCSF variant that was selected for IgG1b12 resistance remained sensitive to MABs 2G12 and 2F5, for combination therapy [Mo (1997)]
- 2G12: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 2G12 bound monomer, and weakly bound oligomer and neutralized JRFL [Fouts (1997)]
- 2G12: One of 14 human MABs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB Env – 2G12 was a strong neutralizer of SHIV-vpu+ – all Ab combinations tested showed synergistic neutralization – 2G12 has synergistic response with MABs 694/98-D (anti-V3), 2F5, F105, and b12 [Li (1997)]

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- 2G12: Review: MAbs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MAbs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs' epitopes [Moore & Trkola(1997)]
- 2G12: Using concentrations of Abs achievable *in vivo*, the triple combination of 2F5, 2G12 and HIVIG was found to be synergistic to have the greatest breadth and magnitude of response against 15 clade B primary isolates [Mascola (1997)]
- 2G12: Viral binding inhibition by 2G12 was strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- 2G12: Review that discusses this MAb – reacts with residues at the base of the V3 loop and V4, and most of the changes that reduce binding are glycosylation sites – it is not clear whether the binding site is peptidic or direct carbohydrate [Burton & Montefiori(1997)]
- 2G12: Neutralizes TCLA strains and primary isolates [Parren (1997b)]
- 2G12: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIV-1 LAI in hu-PBL-SCID mice, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus (1998)]
- 2G12: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- 2G12: Summary of the implications of the crystal structure of gp120 combined with what is known about mutations that reduce NAb binding – probable mechanism of neutralization by 2G12 is unknown, but dependent on proper glycosylation and 2G12 is predicted to be oriented towards the target cell when bound, so neutralization may be due to steric hindrance – mutations in positions N 295, T 297, S 334, N 386, N 392 and N 397 HXBc2 (IIIB) decrease 2G12 binding, and the binding region is 25 angstroms from the CD4 binding site – probably the Ab binds in part to carbohydrates, which may account for both its broad reactivity and the scarcity of Abs in the same competition group [Wyatt (1998)]
- 2G12: Enhances Hx10 binding to CD4 positive or negative HeLa cells, but inhibited binding to CD4+ T-cell line A3.01 – neutralizes Hx10 infection of the HeLa cells [Mondor (1998)]
- 2G12: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D [Connor (1998)]
- 2G12: Does not compete with binding of MAb generated in response to gp120-CD4 complex, CG10 [Sullivan (1998b)]
- 2G12: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer – MAb 2G12 was the only exception to this, showing reduced binding efficiency [Binley (1998)]
- 2G12: A wide range of neutralizing titers was observed that was independent of co-receptor usage [Trkola (1998)]
- 2G12: Notes that 2G12 and 2F5, potent neutralizing antibodies, were identified by screening for cell surface (oligomeric Envelope) reactivity [Fouts (1998)]
- 2G12: Induces Complement-mediated lysis in MN but not primary isolates – primary isolates are refractive to CML [Takefman (1998)]

- 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 [Parren (1998b)]
- 2G12: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) [Li (1998)]
- 2G12: Discussed in a review of the antigenic and receptor binding-domains of gp120 in relation to the structure of the molecule – antibodies are discussed by category (anti-V2, anti-V3, CD4i, CD4BS...), however as 2G12 binds to a rarely immunogenic region, and it is dependent on glycosylation, it was discussed individually [Wyatt & Sodroski(1998)]
- 2G12: 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 [Kunert (1998)]
- 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 [Schonning (1998)]
- 2G12: Infection of dendritic cells cultured from CD14+ blood cells or from cadaveric human skin was blocked by neutralizing MAbs IgG1b12, or 2F5 and 2G12 delivered together, but not by control non-neutralizing anti-gp120 MAb 4.8D, indicating that NAb could interrupt early mucosal transmission events [Frankel (1998)]
- 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 cell lines over PBMCs is that markers (X-Gal) can be added for staining to simplify the assay – the consensus of the meeting was that these engineered cell lines did not improve the sensitivity of detection of primary isolate neutralization – D. Burton and J. Mascola presented results concerning passive immunization and protection of hu-PBL-SCID mice and macaques, respectively, and both found combinations of MAbs that were able to achieve 99% neutralization *in vitro* corresponded to efficacy *in vivo* [Montefiori & Evans(1999)]
- 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 [Beddows (1999)]
- 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 [Altmeyer (1999)]

Table of HIV MAbs

- 2G12: Hu-PBL-SCID mice were infected with HIV-1s JRCSF and SF162 to study the effect of NAb on an established infection – no significant differences in the initial rate of decrease in viral load or the plateau levels of viral RNA between the b12 treated and control mice were seen – in most of the Ab treated mice b12 escape mutants were observed with varying patterns of mutations – a combination of b12, 2G12 and 2F5 protected 1/3 mice, and an isolate from one of the other two was resistant to neutralization by all three MAbs [Poignard (1999)]
- 2G12: Review of the neutralizing Ab response to HIV-1 [Parren (1999)]
- 2G12: Combinations of HIVIG, 2F5, 2G12 were administered in passive-transfer experiments 24 hours prior to challenge with pathogenic SHIV 89.6PD – 3/6 animals given HIVIG/2F5/2G12 were completely protected, the others had reduced viremia and normal CD4 counts – 1/3 monkeys given 2F5/2G12 showed transient infection, the other two had reduced viral load – all monkeys that received HIVIG, 2F5, or 2G12 alone became infected and developed high-level plasma viremia, although animals that got HIVIG or 2G12 had a less profound CD4 T cell decline [Mascola (1999)]
- 2G12: Because HIV-1 is most often transmitted across mucosal surfaces, the ability of passive transfer of infused HIVIG/2F5/2G12 to protect against mucosal exposure of macaques to pathogenic SHIV 89.6PD was studied – HIVIG/2F5/2G12 protected 4/5 animals against vaginal challenge, 2F5/2G12 combined protected 2/5 animals, and 2G12 alone protected 2/4 animals – in contrast, Mascola and co-workers had previously shown single MAbs could not protect against intravenous challenge – Ab treated animals that got infected through vaginal inoculation had low viral loads and only modest declines in CD4 counts – the infused Abs were detected in the nasal, vaginal, and oral mucosa [Mascola (2000)]
- 2G12: Combinations of HIVIG, 2F5, 2G12 were administered in passive-transfer experiments 24 hours prior to challenge with pathogenic SHIV 89.6PD – 3/6 animals given HIVIG/2F5/2G12 were completely protected, the others had reduced viremia and normal CD4 counts – 1/3 monkeys given 2F5/2G12 showed transient infection, the other two had reduced viral load – all monkeys that received HIVIG, 2F5, or 2G12 alone became infected and developed high-level plasma viremia, although animals that got HIVIG or 2G12 had a less profound CD4 T cell decline [Mascola (1999)]
- 2G12: 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 NAb 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 [Binley (1999)]
- 2G12: A triple combination of 2F5, F105 and 2G12 effectively neutralized perinatal infection of macaque infants when challenged with SHIV-vpu+ – the mean plasma half-life of 2G12 was 14.0 ± 7.9 days, the longest of the three Abs [Baba (2000)]
- 2G12: To determine the antigenicity of virus killed by thermal and chemical inactivation, retention of conformation-dependent neutralization epitopes was examined, and exposure of CD4BS epitopes was found to be enhanced (MAbs IgG1b12, 205-46-9, and 205-43-1) – binding to 2G12 and 447-52D epitopes was essentially unaltered – the 17b CD4i epitope was also exposed [Grovit-Ferbas (2000)]

- 2G12: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – 2G12 was an exception and could not neutralize MN in either form [Park (2000)]
- 2G12: Neutralization synergy between anti-HIV NABs b12, 2G12, 2F5, and 4E10 was studied – a classic fixed-ratio method was used, as well as a method where one Ab was fixed at a low neutralization titer and the other was varied – using primary isolates, a two-four fold enhancement of neutralization was observed with MAb pairs, and a ten-fold enhancement with a quadruple Ab combination – no synergy was observed with any MAb pair in the neutralization of TCLA strain HXB2 – there was no evidence for cooperativity of binding between b12 and 2G12 to envelope spikes expressed on the cell surface of TCLA or primary isolates [Zwick (2001c)]
- 2G12: Review of studies in macaques that have shown immune control of pathogenic SHIV viremia, improved clinical outcome, and protection, and the implications of the observations for HIV vaccines [Mascola & Nabel(2001)]
- 2G12: SF162 Δ V2 is a virus that has a 30 amino acid deletion in the V2 loop that does not abrogate its infectivity but renders it highly susceptible to neutralization – when incorporated into a codon-optimized DNA vaccine with a CMV promoter and delivered by gene gun, SF162 Δ V2 gave higher neutralizing Ab titers against SF162 than did SF162 itself, and Abs that cross-neutralized non-homologous primary isolates were obtained only when SF162 Δ V2, but not intact SF162, was used as the immunogen – Control MAbs 2F5 and 2G12 could neutralize all of the following primary isolates: 91US056(R5), 92US714(R5), 92US660(R5), 92HT593(R5X4), and BZ167(R5X4), while after the first protein boost, the sera from two SF162 Δ V2 immunized macaques could neutralize 91US056(R5), 92US714(R5), 92US660(R5) and ADA(R5), but not 92HT593(R5X4) or 92US657(R5) – the pattern of cross-recognition shifted after the second boost [Barnett (2001)]
- 2G12: Moore and colleagues review structural aspects of gp120 and how they relate to antigenic domains, and review the data concerning the lack of a clear relationship between genetic subtype and serotype – an exception exists for human MAb 2G12, which does not recognize CRF01 envelopes because of an unusual additional disulfide bond in the V4 loop region that appears to be unique to the subtype E, CRF01 gp120 protein [Moore (2001)]
- 2G12: Structural aspects of the interaction of neutralizing Abs with HIV-1 Env are reviewed – Env essentially has three faces, one is largely inaccessible on the native trimer, and two that exposed but have low immunogenicity on primary viruses – neutralization is suggested to occur by inhibition of the interaction between gp120 and the target cell membrane receptors as a result of steric hindrance and it is noted that the attachment of approximately 70 IgG molecules per virion is required for neutralization, which is equivalent to about one IgG molecule per spike – the 2G12, 17b and b12 epitopes are discussed in detail – although it is potently neutralizing, 2G12 does not interfere with CD4 and coreceptor binding, and this Ab specificity is uncommon in sera from HIV-1-infected individuals [Poignard (2001)]
- 2G12: Neutralizing synergy between MAbs 1b12, 2G12 and 2F5 was studied using surface plasmon resonance to determine the binding kinetics for these three MAbs with respect to monomeric and oligomeric Env protein gp160 IIIB – the 2G12 epitope is highly accessible on both monomeric and oligomeric Envs, 1b12 is highly accessible on monomers but not oligomers, and 2F5 on neither form – binding of 2G12 exposes the 2F5 epitope on gp160 oligomers – 2G12-gp160 oligomer interactions were best fitted to a two state model, with the first complex having a high association constant and fast dissociation, that is stabilized by conformational changes induced by the binding of a second MAb [Zeder-Lutz (2001)]
- 2G12: A luciferase-reporter gene-expressing T-cell line was developed to facilitate neutralization and drug-sensitivity assays – luciferase and p24 antigen neutralization titer end points were found comparable using NAb from sera from HIV+ donors, and MAbs 2F5, 2G12 and IgG1b12 [Spencehauer (2001)]

Table of HIV MAbs

- 2G12: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 $\mu\text{g/ml}$: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 2G12: A combination of MAbs IgG1b12, 2F5, and 2G12 was given postnatally to four neonate macaques that were then challenged with highly pathogenic SHIV89.6P – one of the four infants remained uninfected after oral challenge, two infants had no or a delayed CD4(+) T-cell decline [Hofmann-Lehmann (2001)]
- 2G12: Twenty HIV clade C isolates from five different countries were susceptible to neutralization by anti-clade B MAbs in a synergistic quadruple combination of mAbs IgG1b12, 2G12, 2F5, and 4E10 [Xu (2001)]
- 2G12: Chloroquine reduces the HIV-1-infectivity of H9 IIIB cells, apparently through altering the conformation of envelope – there is a reduction of reactivity of 2G12 to its epitope in chloroquine treated cultures [Savarino (2001)]
- 2G12: A phase I trial in seven HIV+ individuals was conducted with MAbs 2F5 and 2G12 – no clinical or laboratory abnormalities were observed throughout the study – eight infusions were administered over a 4-week period – the elimination half-life ($t_{1/2}$) was calculated to be 7.94 (range, 3.46–8.31) days for 2F5 and 16.48 (range, 12.84–24.85) days for 2G12 [Armbruster (2002)]
- 2G12: UK Medical Research council AIDS reagent: ARP3030
- 2G12: NIH AIDS Research and Reference Reagent Program: 1476

879	MO101/V3,C4	Env(dis 314–323 + 494–503)	gp120(dis 314–323)	GRAFVTIGKI + LGVAPTKAKR	<i>in vitro</i> stimulation	human(IgM)
		Ab type: V3-C5		References: [Ohlin (1992)]		
		• MO101: Generated through <i>in vitro</i> stimulation of uninfected-donor lymphocytes with pB1 containing IIIB Env 286–467 – reacts with peptides from the V3 and C4 regions, positions 314–323 + 494–503, peptides GRAFVTIGKI + LGVAPTKAKR [Ohlin (1992)]				
880	MO101/V3,C4	Env(dis 314–323 + 494–503)	gp120(dis 494–503)	GRAFVTIGKI + LGVAPTKAKR	<i>in vitro</i> stimulation	human(IgM)
		Ab type: V3-C5		References: [Ohlin (1992)]		
		• MO101: Generated through <i>in vitro</i> stimulation of uninfected-donor lymphocytes with pB1 containing IIIB Env 286–467 – reacts with peptides from the V3 and C4 regions, positions 314–323 + 494–503, peptides GRAFVTIGKI + LGVAPTKAKR [Ohlin (1992)]				
881		Env()	gp120(IIIB)		Vaccine	murine(IgG1)
	Vaccine:	<i>Vector/type:</i> vaccinia	<i>Strain:</i> IIIB	<i>HIV component:</i> gp120	<i>Stimulatory Agents:</i> GM-CSF	
		References: [Rodriguez (1999)]				
		• The murine antibody response to a chimeric of granulocyte-macrophage colony stimulating factor GM-CSF/gp120 in vaccinia was not higher titer than the response to a gp120-vaccinia construct, but the breadth of the antibody response was greater, in particular to the C-term region of gp120				
		• A cellular response of greater intensity was triggered to the GM-CSF/gp120 vaccinia construct, as measured by gamma IFN production in an Elispot assay				

Table of HIV MAbs

882	102–135	Env(dis 549–673)	gp41(dis HAM112, O group)	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HAM112 (group O) <i>HIV component:</i> gp160</p> <p>References: [Scheffel (1999)]</p> <ul style="list-style-type: none"> • 102–135: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity – 102–135 bound to two non-contiguous peptides in combination, assumed to combine to form some type of helical structure, and not to either peptide individually [Scheffel (1999)] 					
883	1025	Env(dis)	gp120(dis)		()
<p>References: [Berman (1997)]</p> <ul style="list-style-type: none"> • 1025: Binds to 1/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)] 					
884	105–134	Env()	gp41(652–681 HAM112, O group)	Vaccine	murine(IgG1 κ)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> HAM112 (group O) <i>HIV component:</i> gp160</p> <p>References: [Scheffel (1999)]</p> <ul style="list-style-type: none"> • 105–134: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity [Scheffel (1999)] 					
885	10E9	Env()	gp41()	HIV-1 infection	murine(IgG1)
<p>References: [Papsidero (1988)]</p> <ul style="list-style-type: none"> • 10E9: 100/100 HIV+ human sera could inhibit 10E9 binding [Papsidero (1988)] 					
886	126–50	Env(dis)	gp41(dis HXB2)	no HIV-1 infection	human(IgG2 κ)
<p>References: [Robinson (1990b), Tyler (1990), Robinson (1991), Xu (1991)]</p> <ul style="list-style-type: none"> • 126–50: No enhancing activity for HIV-1 IIIB [Robinson (1990b)] • 126–50: Serves as target for antibody-dependent cellular cytotoxicity ADCC [Tyler (1990)] • 126–50: No enhancing or neutralizing activity [Robinson (1991)] • 126–50: Specific for a conformational epitope [Xu (1991)] 					
887	12H2	Env(dis 530–677)	gp41(dis 530–677 HXB2)	no Vaccine	murine(IgM κ)
<p>Vaccine: <i>Vector/type:</i> Semliki-Forest Virus <i>HIV component:</i> Env</p> <p>References: [Giraud (1999)]</p> <ul style="list-style-type: none"> • 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 is properly expressed [Giraud (1999)] 					

Table of HIV MAbs

888	13.10 (No. 13)	Env()	gp120()	no	HIV-1 infection	human(IgG1λ)
<p>Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Lake (1989), Moran (1993), Wisnewski (1996)]</p> <ul style="list-style-type: none"> • 13.10: First HIV-1 specific human-mouse hybridoma that produces a MAb that binds to gp120 and gp160 [Lake (1989)] • 13.10: Heavy (V H1) and light (V λII) chain sequenced – no enhancing or neutralizing activity – called No. 13 [Moran (1993)] • 13.10: 13.10 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)] • 13.10: NIH AIDS Research and Reference Reagent Program: 377 						
889	1B1	Env()	Env()	L	HIV-1 infection	human()
<p>Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, Vienna, Austria References: [Buchacher (1994), Purtscher (1994), Kunert (1998)]</p> <ul style="list-style-type: none"> • 1B1: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)] • 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 [Kunert (1998)] 						
890	1F7	Env()	Env()	L	HIV-1 infection	human()
<p>Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, Vienna, Austria References: [Buchacher (1994), Purtscher (1994), Kunert (1998), Grant (2000)]</p> <ul style="list-style-type: none"> • 1F7: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)] • 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 [Kunert (1998)] • 1F7: There is an anti-idiotypic MAb named 1F7 that was raised against pooled IgG from HIV-1+ subjects that recognizes a set of antibodies against HIV Gag, Pol, and Env, and this MAb is reported to inhibit anti-HIV CTL activity – this is not the same as the 1F7 described by Buchacher <i>et al.</i> [Grant (2000)] 						
891	31710B	Env()	gp41()			human(IgG1)
<p>References: [Alsmadi & Tilley(1998)]</p> <ul style="list-style-type: none"> • 31710B: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against all four strains [Alsmadi & Tilley(1998)] 						
892	3D5	Env()	Env()	L	HIV-1 infection	human()
<p>Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, Vienna, Austria References: [Buchacher (1994), Purtscher (1994), Kunert (1998)]</p> <ul style="list-style-type: none"> • 3D5: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)] 						

Table of HIV MAb

- 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 [Kunert (1998)]

893	3H6	Env()	gp41()				murine()
<p>References: [Pinter (1995)]</p> <ul style="list-style-type: none"> • 3H6: There is another MAb with this ID that recognizes Rev [Orsini (1995)] • 3H6: Generated in response to virus grown in protein-free medium [Pinter (1995)] 							
894	6E10	Env(dis)	gp120(dis)	L	Vaccine		()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> gp160</p> <p>Donor: Phil Berman</p> <p>References: [Berman (1991)]</p>							
895	7–1054	Env()	gp36(HIV-2)	no			murine()
<p>References: [Scheffel (1999)]</p> <ul style="list-style-type: none"> • Binds HIV-2 gp36, used as a control in a study of group O MAbs [Scheffel (1999)] 							
896	A9	Env()	gp120(IIIB)		Vaccine		murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> chimeric GM-CSF <i>Strain:</i> IIIB <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> GM-CSF</p> <p>References: [del Real (1999)]</p> <ul style="list-style-type: none"> • A9: Murine antibody response to the chimeric construction granulocyte-macrophage colony stimulating factor GM-CSF/gp120 was tested, MAbs from normal mice were gp120 specific, MAbs from nude mice bound gp120 but were polyreactive, and from reconstituted mice were half way between – the anti-gp120 response used a high frequency of VH81X, VHQ52, and VH7183 genes, a family used during fetal life and associated with autoimmunity – A9 was a gp120 from a BALBc reconstructed nude mouse and had VH gene 7183–2 [del Real (1999)] 							
897	B4	Env()	gp120(IIIB)		Vaccine		murine(IgM)
<p>Vaccine: <i>Vector/type:</i> chimeric GM-CSF <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>References: [del Real (1999)]</p> <ul style="list-style-type: none"> • B4: Murine antibody response to the chimeric construction granulocyte-macrophage colony stimulating factor GM-CSF/gp120 was tested, MAbs from normal mice were gp120 specific, MAbs from nude mice bound gp120 but were polyreactive, and from reconstituted mice were half way between – the anti-gp120 response used a high frequency of VH81X, VHQ52, and VH7183 genes, a family used during fetal life and associated with autoimmunity – B4 was an anti-gp120 from a BALBc reconstructed nude mouse and had VH gene J606 [del Real (1999)] 							
898	B5	Env()	gp120(IIIB)		Vaccine		murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> chimeric GM-CSF <i>Strain:</i> IIIB <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> GM-CSF</p> <p>References: [del Real (1999)]</p>							

Table of HIV MAbs

- B5: Murine antibody response to the chimeric construction granulocyte-macrophage colony stimulating factor GM-CSF/gp120 was tested, MAbs from normal mice were gp120 specific, MAbs from nude mice bound gp120 but were polyreactive, and from reconstituted mice were half way between – the anti-gp120 response used a high frequency of VH81X, VHQ52, and VH7183 genes, a family used during fetal life and associated with autoimmunity – B5 was a gp120 specific MAb from a BALBc mouse and had VH gene J558 [del Real (1999)]

899	B6	Env()	gp120(IIIB)		Vaccine	murine(IgM)
		Vaccine: <i>Vector/type:</i> chimeric GM-CSF <i>Strain:</i> IIIB <i>HIV component:</i> gp120				
		References: [del Real (1999)]				
		<ul style="list-style-type: none"> • B6: Murine antibody response to the chimeric construction granulocyte-macrophage colony stimulating factor GM-CSF/gp120 was tested, MAbs from normal mice were gp120 specific, MAbs from nude mice bound gp120 but were polyreactive, and from reconstituted mice were half way between – the anti-gp120 response used a high frequency of VH81X, VHQ52, and VH7183 genes, a family used during fetal life and associated with autoimmunity – B6 was a gp120 from a BALBc reconstructed nude mouse and had VH gene J558 [del Real (1999)] 				
900	BAT267	Env()	gp120()	L	Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> IIIB <i>HIV component:</i> virus				
		References: [Fung (1987)]				
901	BAT401	Env()	gp120()	L	Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> IIIB <i>HIV component:</i> virus				
		References: [Fung (1987)]				
902	BAT509	Env()	gp120()	L	Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> inactivated virus <i>Strain:</i> IIIB <i>HIV component:</i> virus				
		References: [Fung (1987)]				
903	C31	Env()	gp120()	no	HIV-1 infection	human(IgG1 κ)
		References: [Boyer (1991)]				
		<ul style="list-style-type: none"> • C31: Broadly-reactive group specific MAb – high yield cultivation of human MAb [Boyer (1991)] 				
904	D1	Env(dis)	gp41(dis IIIB)		Vaccine	murine(IgG)
		Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140				
		References: [Otteken (1996)]				
		<ul style="list-style-type: none"> • D1: MAbs D1, D16, had T37 bind to oligomeric gp160 equally well – pulse label experiments of MAb binding to noncleavable gp160 revealed that these MAbs bound with a delay, epitopes forming with a half-life of 30 min [Otteken (1996)] 				

905	D12	Env(dis)	gp41(dis IIIB)	L	Vaccine	murine(IgG)
<i>Vaccine:</i> Vector/type: vaccinia Strain: IIIB HIV component: oligomeric gp140						
References: [Broder (1994), Richardson (1996), Earl (1997), Otteken (1996), LaBranche (1999)]						
<ul style="list-style-type: none"> • D12: One of 18 MAbs (e.g. D4 and D40) that bind to a conformation-dependent epitope in gp41 that bind preferentially, but not exclusively, to oligomers – neutralizes IIIB and SF2 [Broder (1994)] • 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 [Richardson (1996)] • D12: MAbs D10 and D12 are very easily blocked by human sera from HIV+ individuals [Earl (1997)] • D12: MAbs D4, D10, D11, D12, and D41 all bind only to complete oligomer – pulse label experiments of MAb binding to noncleavable gp160 revealed that these MAbs bound with a delay, epitopes forming with a half-life of 30 min [Otteken (1996)] • D12: D12 was used in WB of HIV-1 transmembrane proteins in a study which showed that determinants of HIV-1 CD4 independence map outside regions required for coreceptor specificity – IIIBx, a CD4-independent variant of IIIB, has a truncated gp41 [LaBranche (1999)] 						
906	D16	Env(dis)	gp41(dis IIIB)	L	Vaccine	murine(IgG)
<i>Vaccine:</i> Vector/type: protein HIV component: dimeric Env						
References: [Earl (1994), Weissenhorn (1996), Earl (1997)]						
<ul style="list-style-type: none"> • D16: Precipitates both oligomeric gp140 and soluble monomeric gp41(21–166)that lacks the fusion peptide and membrane anchor, along with MAbs D16, D38, D40, D41, and D54 [Weissenhorn (1996)] • D16: One of eleven MAbs (D16, D17, D31, D36, D37, D40, D44, D55, D59, T37, and T45) that are conformation dependent and that can block the binding of the MAb D50 that binds to the linear peptide gp41(642–665) – reactive with 9/10 HIV-1 strains all except HIV-1 ADA, which has the change E659D and E662A that may result in the loss of binding (ELLE to DLLA) [Earl (1997)] 						
907	D4	Env()	gp120(IIIB)		Vaccine	murine(IgG1)
<i>Vaccine:</i> Vector/type: chimeric GM-CSF Strain: IIIB HIV component: gp120						
References: [del Real (1999)]						
<ul style="list-style-type: none"> • D4: Murine antibody response to the chimeric construction granulocyte-macrophage colony stimulating factor GM-CSF/gp120 was tested, MAbs from normal mice were gp120 specific, MAbs from nude mice bound gp120 but were polyreactive, and from reconstituted mice were half way between – the anti-gp120 response used a high frequency of VH81X, VHQ52, and VH7183 genes, a family used during fetal life and associated with autoimmunity – D4 was a gp120 from a BALBc reconstructed nude mouse and had VH gene J558 [del Real (1999)] 						
908	D43	Env(dis)	gp41(dis HXB2)		Vaccine	murine(IgG)
<i>Vaccine:</i> Vector/type: protein HIV component: dimeric Env						
References: [Earl (1994), Richardson (1996), Earl (1997)]						
<ul style="list-style-type: none"> • 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 [Richardson (1996)] 						

Table of HIV MAbs

- 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 [Earl (1997)]

909	F223	Env() gp120()		no	HIV-1 infection	human(IgG3λ)
<p>References: [Cavacini (1999)]</p> <ul style="list-style-type: none"> • F223: binds to HIV-1 gp120 – also binds to uninfected lymphocytes, binding to a 159-kd auto-antigen expressed on most B cells and a small fraction of T and NK cells – the antibody enhances HIV-1 infection in a complement-dependent manner – F223 light chains have a strong homology with VLgamma2, the heavy chain to the germline gene VH3-H.11 – N-linked carbohydrates are key for recognition of both gp120 and the autoantigen – MAb 3D6 also uses VH3 and has autoreactivity [Cavacini (1999)] 						
910	F285	Env() Env()			HIV-1 infection	human(IgG1)
<p>References: [Wisnewski (1995), Wisnewski (1996)]</p> <ul style="list-style-type: none"> • F285: F285 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)] 						
911	F7	Env() gp120(III B)			Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> chimeric GM-CSF <i>Strain:</i> III B <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> GM-CSF</p> <p>References: [del Real (1999)]</p> <ul style="list-style-type: none"> • F7: Murine antibody response to the chimeric construction granulocyte-macrophage colony stimulating factor GM-CSF/gp120 was tested, MAbs from normal mice were gp120 specific, MAbs from nude mice bound gp120 but were polyreactive, and from reconstituted mice were half way between – the anti-gp120 response used a high frequency of VH81X, VHQ52, and VH7183 genes, a family used during fetal life and associated with autoimmunity – F7 was a gp120 specific MAb from a BALBc mouse and had VH gene 7183(81X), previously found expressed only in fetal liver [del Real (1999)] 						
912	Fab A12	Env(dis) gp41(dis LAI)		no	HIV-1 infection	human(IgG1κ)
<p>References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab A12: Uncharacterized epitope – variable regions sequenced [Binley (1996)] 						
913	Fab A2	Env(dis) gp41(dis LAI)		no	HIV-1 infection	human(IgG1λ)
<p>References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab A2: Uncharacterized epitope – variable regions sequenced [Binley (1996)] 						
914	Fab L9	Env(dis) gp41(dis LAI)		no	HIV-1 infection	human(IgG1κ)
<p>References: [Binley (1996)]</p> <ul style="list-style-type: none"> • Fab L9: Uncharacterized epitope – variable regions sequenced [Binley (1996)] 						
915	G12	Env() gp120(III B)			Vaccine	murine(IgM)
<p>Vaccine: <i>Vector/type:</i> chimeric GM-CSF <i>Strain:</i> III B <i>HIV component:</i> gp120</p> <p>References: [del Real (1999)]</p>						

- G12: Murine antibody response to the chimeric construction granulocyte-macrophage colony stimulating factor GM-CSF/gp120 was tested, MAbs from normal mice were gp120 specific, MAbs from nude mice bound gp120 but were polyreactive, and from reconstituted mice were half way between – the anti-gp120 response used a high frequency of VH81X, VHQ52, and VH7183 genes, a family used during fetal life and associated with autoimmunity – G12 was a gp120 from a BALBc reconstructed nude mouse and had VH gene 7183–6 [del Real (1999)]

916	G2	Env()	gp120(IIIB)	Vaccine	murine(IgM)
<p>Vaccine: <i>Vector/type:</i> chimeric GM-CSF <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>References: [del Real (1999)]</p> <ul style="list-style-type: none"> • G2: Murine antibody response to the chimeric construction granulocyte-macrophage colony stimulating factor GM-CSF/gp120 was tested, MAbs from normal mice were gp120 specific, MAbs from nude mice bound gp120 but were polyreactive, and from reconstituted mice were half way between – the anti-gp120 response used a high frequency of VH81X, VHQ52, and VH7183 genes, a family used during fetal life and associated with autoimmunity – G2 was a gp120 from a BALBc reconstructed nude mouse and had VH gene Q52 [del Real (1999)] 					
917	H2	Env(dis)	gp41(dis)		human(IgM κ)
<p>Donor: BioInvent, Lund, Sweden, commercial</p> <p>References: [Muller (1991)]</p> <ul style="list-style-type: none"> • H2: Anti-idiotypic MAbs (10B3 and 2A11) against MAb H2 were generated by immunization of BALBc mice with H2 – they also react with seropositive sera [Muller (1991)] 					
918	H8	Env()	gp120(IIIB)	Vaccine	murine(IgM)
<p>Vaccine: <i>Vector/type:</i> chimeric GM-CSF <i>Strain:</i> IIIB <i>HIV component:</i> gp120</p> <p>References: [del Real (1999)]</p> <ul style="list-style-type: none"> • H8: Murine antibody response to the chimeric construction granulocyte-macrophage colony stimulating factor GM-CSF/gp120 was tested, MAbs from normal mice were gp120 specific, MAbs from nude mice bound gp120 but were polyreactive, and from reconstituted mice were half way between – the anti-gp120 response used a high frequency of VH81X, VHQ52, and VH7183 genes, a family used during fetal life and associated with autoimmunity – H8 was a gp120 from a BALBc reconstructed nude mouse and had VH gene Q52 [del Real (1999)] 					
919	HBW4	Env()	gp120(IIIB)	HIV-1 infection	human(IgG1 λ)
<p>References: [Moran (1993), Wisniewski (1995), Wisniewski (1996)]</p> <ul style="list-style-type: none"> • HBW4: Heavy (V HIII) and light (V λII) chain sequenced [Moran (1993)] • HBW4: HBW4 is V H2 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisniewski (1996)] 					
920	K14	Env(dis)	gp41(dis)	no	human(IgG1)
<p>References: [Teeuwssen (1990), Schutten (1995a), Schutten (1995b), Schutten (1996), Schutten (1997)]</p>					

Table of HIV MAbs

- 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 [Teeuwssen (1990)]
- K14: Reduced affinity for both SI and NSI viruses relative to MAb MN215, failed to neutralize SI strain [Schutten (1995b)]
- K14: In a study of NSI and SI virus neutralization, K14 did not influence viral entry [Schutten (1997)]

921	M25	Env()	gp41()			Vaccine	murine(IgG κ)
		Vaccine: <i>Vector/type:</i> purified HIV-1					
		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 [Watkins (1996)]					
922	MAG 6B	Env(dis)	gp120(dis)	no		Vaccine	murine()
		Vaccine: <i>Vector/type:</i> sCD4-gp120 complex		<i>Strain:</i> HXB2	<i>HIV component:</i> gp120		
		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 [Kang (1994)]					
923	MO28	Env(dis 632–691)	gp41(dis)	no		<i>in vitro</i> stimulation	human(IgM)
		References: [Ohlin (1989)]					
		• MO28: This antibody was raised by <i>in vitro</i> stimulation with a recombinant Env penv9 – the discontinuous epitope involves hydrophobic regions 632–646, 677–681 and 687–691, proximal to and spanning the transmembrane region – this specificity is unusual in HIV-1 positive sera [Ohlin (1989)]					
924	MO30	Env(dis 632–691)	gp41(dis)	no		<i>in vitro</i> stimulation	human(IgM)
		References: [Ohlin (1989)]					
		• MO30: This antibody was raised by <i>in vitro</i> stimulation with a recombinant Env penv9 – the discontinuous epitope involves hydrophobic regions 632–646, 677–681 and 687–691, proximal to and spanning the transmembrane region – this specificity is unusual in HIV-1 positive sera [Ohlin (1989)]					
925	MO43	Env(dis 632–691)	gp41(dis)	no		<i>in vitro</i> stimulation	human(IgM)
		References: [Ohlin (1989)]					
		• MO43: This antibody was raised by <i>in vitro</i> stimulation with a recombinant Env penv9 – the discontinuous epitope of MO43 involves hydrophobic regions 632–646, 677–681 and 687–691, proximal to and spanning the transmembrane region – this specificity is unusual in HIV-1 positive sera [Ohlin (1989)]					
926	multiple Fabs	Env()	gp120()			HIV-1 infection	human()

<p>References: [Burton (1991)]</p> <ul style="list-style-type: none"> • A panel of anti-gp120 Fabs was generated by antigen selection from a random combinatorial library prepared from bone marrow from an asymptomatic individual [Burton (1991)] 						
927	multiple MAbs	Env(dis)	gp120(dis)		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> protein <i>HIV component:</i> gp120</p> <p>References: [Denisova (1996)]</p> <ul style="list-style-type: none"> • When gp120 was used as an immunogen, in contrast to gp120 bound to an anti-V3 MAb, few MAbs were generated and all bound better to the native than to the denatured protein – MAbs generated were: G1B12, G2F7, G9G8, G12F12, G1B8, G11F11, G9E8, G1B11, G1B6, G6F2, G2E7 [Denisova (1996)] 						
928	multiple MAbs	Env(dis)	gp120(dis)		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> gp120-CD4 complex <i>HIV component:</i> gp120</p> <p>References: [Denisova (1996)]</p> <ul style="list-style-type: none"> • When gp120-CD4 was used as an immunogen, in contrast to gp120 bound to an anti-V3 MAb, few MAbs were generated and all bound better to the native than to the denatured protein – MAbs generated were: CG43, CG41, CG49, CG53, CG42, CG4, CG46, CG40, CG52, CG51, CG48, CG50, CG125, CG124, CG121 [Denisova (1996)] 						
929	multiple MAbs	Env()	gp120()		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> protein-Ab complex <i>HIV component:</i> gp120 complexed with MAb M77</p> <p>References: [Denisova (1996)]</p> <ul style="list-style-type: none"> • When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes, as well as an array of MAbs to discontinuous epitope – 10 of 36 MAbs were mapped to linear epitopes and are mentioned elsewhere in this database, the others are: GV5H1, GV4D5, GV4G10, GV1A8, GV10H5, GV8E11, GV2H4, GV6E6, GV1F7, GV1G9, GV4G5, GV6B12, GV1E8, GV2B7, GV1B11, GV6H5, GV6G2, GV6B5, GV1E10, GV5E3, GV5B9, GV5F4, GV6G4, GV1A12, GV5C11, GV6B6, GV3C10 [Denisova (1996)] 						
930	N2–4	Env()	gp41()	no	HIV-1 infection	human(IgG1 κ)
<p>Donor: Evan Hersh and Yoh-Ichi Matsumoto</p> <p>References: [Robinson (1990b)]</p> <ul style="list-style-type: none"> • N2–4: No enhancing activity for HIV-1 IIIB [Robinson (1990b)] • N2–4: NIH AIDS Research and Reference Reagent Program: 528 						
931	N70–2.3a	Env(dis 272–509)	gp120(dis)	no	HIV-1 infection	human(IgG1)
<p>Donor: J. Robinson, Tulane University, LA</p> <p>References: [Robinson (1990a), Takeda (1992)]</p>						

Table of HIV MAbs

- N70–2.3a: Broad reactivity [Robinson (1990a)]
- N70–2.3a: Fc receptor mediated enhancement of HIV-1 infection – binds a conformational site in the carboxyl half of gp120, distinct from 1.5e [Takeda (1992)]

932	P43110	Env(dis)	gp120(dis)			()
<p>Donor: Advanced Biosciences (Kensington, MD)</p> <p>References: [di Marzo Veronese (1992), VanCott (1995)]</p> <ul style="list-style-type: none"> • P43110: Does not recognized denatured form of the gp120 protein [VanCott (1995)] 						
933	P5–3	Env()	gp120()		HIV-1 infection	human(IgG1λ)
<p>Donor: Evan Hersh and Yoh-Ichi Matsumoto</p> <p>References: [Robinson (1990b), Pincus (1991)]</p> <ul style="list-style-type: none"> • P5–3: No enhancing activity for HIV-1 IIIB [Robinson (1990b)] • P5–3: Poor immunotoxin activity when coupled to RAC – isotype specified as: IgG3lambda [Pincus (1991)] • P5–3: NIH AIDS Research and Reference Reagent Program: 378 						
934	polyclonal	Env()	Env()		P and L HIV-1 infection	human(IgG3)
<p>References: [Scharf (2001)]</p> <ul style="list-style-type: none"> • IgG3: HIVIG was separated into immunoglobulin classes and IgG3 neutralization of HIV strains X4, R5 and X4R5 strains was superior to IgG1 and IgG2, and IgG3 was also a more potent inhibitor of viral fusion – the IgG3 advantage was lost when only Fabs were considered, indicating the IgG3 neutralization efficacy is enhanced due to a longer hinge region of the heavy chain in comparison to IgG1 and IgG2 [Scharf (2001)] 						
935	polyclonal	Env()	gp160(IIIB)	none	HIV-1 infection, Vaccine	human()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> NL4–3 <i>HIV component:</i> gp160 <i>Stimulatory Agents:</i> alum</p> <p>References: [Cox (1999)]</p> <ul style="list-style-type: none"> • 60 asymptomatic HIV-1 infected patients were vaccinated with rec gp160 in alum, produced in a baculovirus expression vector in insect cells (VaxSyn), 64 received placebo, and all were followed in a 5 year longitudinal study – a mean of 78% of vaccinated and 82% of those receiving placebo had demonstrable ADCC at the different time intervals in the study, and the vaccine did not enhance ADCC production – patients with rapid and slow disease progression showed similar ADCC levels [Cox (1999)] 						
936	polyclonal	Env()	gp160(89.6)		yes Vaccine	Rhesus macaque()
<p>Vaccine: <i>Vector/type:</i> modified vaccinia Ankara <i>Strain:</i> 89.6 <i>HIV component:</i> SIVmac239 Gag/Pol and HIV-1 89.6P Env</p> <p><i>Stimulatory Agents:</i> IL2/Ig</p> <p>References: [Barouch (2001)]</p> <ul style="list-style-type: none"> • Four rhesus macaques were vaccinated with a modified vaccinia Ankara (MVA) vaccine that elicited strong CTL responses as well as antibody responses 						

Table of HIV MAbs

- The animals were infected when challenged with pathogenic SHIV-89.6P, but had potent CTL responses, secondary NAb responses upon challenge, partial preservation of CD4+ T-cell counts, lower viral loads, and no evidence of disease or mortality by day 168 after challenge – monkeys that got a sham vaccine had high viral load, progressed to disease, and 2/4 were dead by day 168 [Barouch (2001)]

937	polyclonal	Env()	gp120(SF2)	L	Vaccine	murine, baboon()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> SF2 <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> PLG+MF59 microparticles</p> <p>References: [O’Hagan (2000)]</p> <ul style="list-style-type: none"> • Microparticles were used as an adjuvant for entrapped HIV-1 gp120 and induced strong serum IgG responses in mice – polylactide co-glycolide polymer (PLG) microparticles in combination with MF59 had the highest response [O’Hagan (2000)] 						
938	polyclonal	Env()	gp120(SF2)		Vaccine	mouse, guinea pig, macaque()
<p>Vaccine: <i>Vector/type:</i> DNA, recombinant protein <i>Strain:</i> SF2 <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> PLG microparticles, aluminum phosphate, MF-59</p> <p>References: [O’Hagan (2001)]</p> <ul style="list-style-type: none"> • DNA vaccines of codon-optimized Env and Gag genes driven by CMV promoters and absorbed on to PLG microparticles were more effective than naked DNA at eliciting strong Ab responses (more rapid, higher titer, more stable), comparable to gp120 in MF-59 [O’Hagan (2001)] 						
939	polyclonal	Env()	gp140(US4)		Vaccine	mouse, guinea pig, macaque()
<p>Vaccine: <i>Vector/type:</i> DNA <i>Strain:</i> US4 <i>HIV component:</i> gp140 <i>Stimulatory Agents:</i> PLG microparticles, aluminum phosphate, MF-59</p> <p>References: [O’Hagan (2001)]</p> <ul style="list-style-type: none"> • DNA vaccines of codon-optimized Env and Gag genes driven by CMV promoters were absorbed on to PLG microparticles were more effective than naked DNA at eliciting strong Ab responses (more rapid, higher titer, more stable), comparable to gp120 in MF-59 [O’Hagan (2001)] 						
940	polyclonal	Env()	gp120()	L	HIV-1 infection	chimpanzee(IgG)
<p>References: [Shibata (1999)]</p> <ul style="list-style-type: none"> • 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 – <i>in vitro</i> neutralization correlated with protection <i>in vivo</i> [Shibata (1999)] 						
941	polyclonal	Env()	gp160(MN)	L P	HIV-1 infection	human(IgA)
<p>References: [Moja (2000)]</p>						

Table of HIV MAbs

- 15 samples isolated from parotid saliva were selected for study of anti-Env IgA – IgA neutralizing activity was detected that was not directed at either EDELKWA or the V3 loop [Moja (2000)]

942	polyclonal	Env()	Env()		yes	HIV-1 infection	human()
<p>References: [Kim (2001)]</p> <ul style="list-style-type: none"> • After HAART reduction of viral load to <400 for three visits over a 12 month interval, 2/11 patients were found to have increased anti-Env Ab binding titers, and neutralizing Abs titers increased against primary isolates US1, and CM237 – no NAB titer increase was seen to more readily neutralized isolate BZ167 – this suggests that in certain individuals the control of HIV-1 by HAART may augment immune control of HIV [Kim (2001)] 							
943	polyclonal	Env()	Env()		yes	HIV-1 exposed seronegative	human(IgA)
<p>References: [Kaul (2001)]</p> <ul style="list-style-type: none"> • Kaul <i>et al.</i> provide a concise summary of the findings concerning the presence of Mucosal IgA in highly exposed, uninfected subjects, arguing for a role in protection [Kaul (2001)] 							
944	polyclonal	Env()	gp120(SF2)		yes	Vaccine	macaque()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> SF2 <i>HIV component:</i> gp120, p24 <i>Stimulatory Agents:</i> ISCOM</p> <p>References: [Heeney (1998)]</p> <ul style="list-style-type: none"> • The immune responses induced in Rhesus monkeys using two different immunization strategies were studied – one vaccine group was completely protected from challenge infection, the other vaccinees and controls became infected – protected animals had high titers of heterologous NABs, and HIV-1-specific T helper responses – increases in RANTES, MIP-1α and MIP 1 beta produced by circulating CD8+ T cells were also associated with protection [Heeney (1998)] 							
945	polyclonal	Env()	gp120()			Vaccine	macaque()
<p>Vaccine: <i>Vector/type:</i> peptide, recombinant protein <i>Strain:</i> SF2, SF33 <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> ISCOM, MF59</p> <p>References: [Verschoor (1999)]</p> <ul style="list-style-type: none"> • Attempts were made to broaden immune responses induced in Rhesus monkeys by immunization of animals previously immunized that had resisted homologous challenge, with a second immunization with ISCOM-peptides or a boost with gp120 from SF33 – animals didn't survive a second challenge heterologous challenge virus SHIV(SF33) raising concerns about early antigenic sin [Verschoor (1999)] 							
946	polyclonal	Env()	gp120()		L	Vaccine	()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> SF2, MN <i>HIV component:</i> gp120</p> <p>References: [McElrath (2000)]</p>							

- After 3 immunizations, 210/241 (87%) HIV-1 uninfected vaccinees in a phase II trial developed NAb – of 140 patients receiving 4 vaccinations, 53% had persistent neutralizing antibodies to homologous virus, and 34% to heterologous virus, measured at day 728 after initial immunization – immunogens were well tolerated– but IVDUs had a decreased Ab response relative to lower risk groups [McElrath (2000)]

947	polyclonal	Env()	gp120()		Vaccine	murine()
	Vaccine:	<i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> GM-CSF/gp120 chimera				
		References: [Rodriguez (1999)]				
		<ul style="list-style-type: none"> • The murine antibody response to a chimeric of granulocyte-macrophage colony stimulating factor GM-CSF/gp120 in vaccinia was not higher titer than the response to a gp120-vaccinia construct, but the breadth of the antibody response was greater [Rodriguez (1999)] • A cellular response of greater intensity was triggered to the GM-CSF/gp120 vaccinia construct, as measured by proliferation and gamma IFN production in an Elispot assay 				
948	polyclonal	Env()	gp120(YU2)		Vaccine	murine(IgG)
	Vaccine:	<i>Vector/type:</i> stabilized Env trimer <i>Strain:</i> YU2, HXBc2 <i>HIV component:</i> Env				
		Donor: Joseph Sodroski, Harvard Medical School				
		References: [Yang (2001)]				
		<ul style="list-style-type: none"> • Soluble Env trimers were created that were designed to mimic functional Env oligomers – stabilized trimers could induce neutralizing antibodies more effectively than gp120, and Abs to the YU2 trimer were cross-reactive within clade B and could neutralize several primary and TCLA reactive strains – the stabilized trimers did not neutralize primary isolates outside the B clade, from clades C, D, and E – HXBc2 stabilized trimer antigen elicited strong neutralizing Abs against the homologous isolate HXBc2 TCLA strain, but not against primary isolates [Yang (2001)] 				
949	polyclonal	Env()	gp120(MN)		Vaccine	human()
	Vaccine:	<i>Vector/type:</i> recombinant protein <i>Strain:</i> MN <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> QS-21, alum				
		References: [Evans (2001)]				
		<ul style="list-style-type: none"> • Vaccination with QS-21 adjuvant and rsgp120 elicited stronger and more sustained neutralizing antibody responses and lymphocyte proliferation with lower doses of rsgp120 than alum formulations, suggesting QS-21 may be a means to reduce the dose of soluble protein [Evans (2001)] 				
950	polyclonal	Env()	gp120()	yes	HIV-1 infection	human()
		References: [Binley (2000)]				
		<ul style="list-style-type: none"> • HAART inhibited the development of anti-gp120 Ab when initiated during primary infection and sometimes in patients treated within 2 years of HIV-1 infection – HAART during primary infection usually did not inhibit the development of weak NAb responses against autologous virus – 3/4 patients intermittently adherent developed high titers of autologous NAb, largely coincident with brief viremic periods [Binley (2000)] 				
951	polyclonal	Env()	gp120(SIV)	yes	HIV-1 infection	macaque()
		References: [Reitter (1998)]				

Table of HIV MAbs

- This study was not done with HIV-1, but concerned an SIV mutated strain that lacked 4th, 5th and 6th sites for N-linked glycosylation – monkeys infected with the mutant viruses had increased neutralizing activity in their sera relative to monkeys infected with the parental strain

952	polyclonal	Env()	gp120()	yes	Vaccine	human()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> SF2 <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> MF-59</p> <p>References: [Nitayaphan (2000)]</p> <ul style="list-style-type: none"> • A phase I/II trial was conducted in 52 seronegative Thais immunizing with rgp120 SF2 – the vaccine was safe and 39/40 developed NAb responses to the autologous SF2, while 22/40 were able to cross-neutralize the heterologous strain MN [Nitayaphan (2000)] 						
953	polyclonal	Env()	gp120()	yes	Vaccine	baboon()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> SF2 (subtype B), CM235 (CRF01) <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> MF-59</p> <p>References: [VanCott (1999)]</p> <ul style="list-style-type: none"> • Immunization with rgp120 CM235 (CRF01) induced Abs capable of neutralizing TCLA subtype E (CRF01) and subtype B isolates, while rgp120SF2 induced Abs could only neutralize subtype B TCLA isolates – neither immunogen induced Abs capable of neutralizing primary HIV-1 isolates – both rgp120CM235 and rgp120SF2 induced Abs to regions within C1, V1/V2, V3, and C5, but unique responses were induced by rgp120CM235 to epitopes within C2, and by rgp120SF2 to multiple epitopes within C3, V4, and C4 – CM235 baboon sera bound 3- to 12-fold more strongly than the SF2 baboon sera to all subtype E gp120s while binding to subtype B gp120s (except SF2) were within two to threefold for the SF2 and CM235 baboon sera [VanCott (1999)] 						
954	polyclonal	Env()	gp120()		HIV-1 infection	human(IgG)
<p>References: [Binley (1997b)]</p> <ul style="list-style-type: none"> • Retention of anti-Env antibodies and loss of anti-Gag antibodies during disease progression was studied, and suggested to be the result of the loss of T-cell help and the unique ability of Env to stimulate B cells even in a backdrop of declining CD4 cells, because of the ability of Env to bind to the CD4 molecule [Binley (1997b)] 						
955	polyclonal	Env()	gp120(W61D)	L	Vaccine	human()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> W61D <i>HIV component:</i> gp120</p> <p>References: [Beddows (1999)]</p> <ul style="list-style-type: none"> • 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 [Beddows (1999)] 						
956	polyclonal	Env()	gp120()	L	Vaccine	Rhesus macaque()
<p>Vaccine: <i>Vector/type:</i> virus-like particle <i>HIV component:</i> Pr55gag, anchored gp120, V3+CD4 linear domains</p>						

Table of HIV MAbs

962	polyclonal	Env() References: [Neshat (2000)]	gp120()			human(Ig V_H3)
		<ul style="list-style-type: none"> HIV-1 gp120 appears to be a B cell superantigen that binds to members of the V_H3 Ig gene family – the gp120 binding site was localized to the Fab portion of the Ab, and discontinuous residues in the V_H region were critical [Neshat (2000)] 				
963	polyclonal	Env() Vaccine: <i>Vector/type:</i> DNA <i>Strain:</i> ADA, IIIB, 89.6 <i>HIV component:</i> gp120 <i>Stimulatory Agents:</i> C3d fusion	gp120(BH10)		Vaccine	murine(IgG)
		<ul style="list-style-type: none"> gp120 was fused with murine complement protein C3d in a DNA vaccine to enhance the titers of Ab to Env – fusion to C3d resulted in a more rapid onset of Ab response and avidity maturation, after three immunizations in BALB/c mice with DNA on a gold bead delivered with a gene gun, but not in a strong neutralizing Ab response [Ross (2001)] 				
964	polyclonal	Env() References: [Sarmati (2001)]	gp120()	none	P HIV-1 infection	human()
		<ul style="list-style-type: none"> Some HIV-1 infected patients have increasing CD4 counts despite failing ARV – no correlation was found between NAb and viral load in these patients [Sarmati (2001)] 				
965	polyclonal	Env() References: [Llorente (1999)]	gp120(IIIB)		no	human(IgM)
		<ul style="list-style-type: none"> Combinatorial antibody analysis by phage display and flow cytometry demonstrated that gp120 in HIV-1 negative people is recognized by IgM, but not IgG Abs – IgM Fab reactivity is observed throughout the entire sequence of HIV-1 IIIB gp120 and is characterized by low affinity binding and near germline configuration reflecting a lack of maturation of the IgM repertoire – no neutralizing activity was observed in a non-infected individual before isotope switching [Llorente (1999)] 				
966	polyclonal	Env() Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> SF2 <i>HIV component:</i> gp120	gp120(SF2)		L Vaccine	human(IgM)
		<ul style="list-style-type: none"> High risk volunteers were vaccinated with SF2 gp120 – 3 breakthrough cases were studied – SF2 neutralizing Abs were observed, but Ab titers to autologous virus were never high and took 6 months after HIV-1 infection to develop – viral loads were similar to HIV-1 infected individuals who had not been vaccinated [Locher (1999)] 				
967	polyclonal	Env() Vaccine: <i>Vector/type:</i> formaldehyde-fixed whole-cell <i>HIV component:</i> gp120	gp120(subtypes A-E)		yes Vaccine	murine(IgG)
		<ul style="list-style-type: none"> In this study, immunogens were generated that were thought to capture transient envelope-CD4-coreceptor structures that arise during HIV binding and fusion by formaldehyde-fixation of cocultures of cells expressing HIV-1 Env and those expressing CD4 and CCR5 receptors – these cells elicited NAb in CD4- and CCR5-transgenic mice that neutralized 23/24 primary isolates from clades A-E [LaCasse (1999)] 				

- A retraction was printed (Science 296:1025, 2002) noting that an unknown cytotoxic effect of these complex sera accounted for a major fraction of the neutralization reported in [LaCasse (1999)] [Nunberg(2002)]

968	polyclonal	Env()	gp140(IIIB)	L	Vaccine	rabbit(IgG)
	Vaccine:	<i>Vector/type:</i> recombinant protein adjuvant, QS-21 adjuvant		<i>Strain:</i> IIIB	<i>HIV component:</i> gp140, gp120	<i>Stimulatory Agents:</i> MPL-SE
		References: [Earl (2001)]				
		<ul style="list-style-type: none"> • Immunization of rabbits with oligomeric gp140 induced production of higher levels of cross-reactive neutralizing Abs than immunization with gp120 – immunization of Rhesus macaques with gp140 yielded strong NAb against IIIB, modest against other lab-adapted strains, and no NAb activity against primary isolates – most neutralizing activity could not be blocked by a V3 peptide – 3/4 vaccinated macaques showed no viral replication upon intravenous challenge with SHIV-HXB2 [Earl (2001)] 				
969	polyclonal	Env()	gp140(SF162ΔV2)	yes	Vaccine	rabbit, Rhesus macaque(IgG)
	Vaccine:	<i>Vector/type:</i> DNA, CMV promotor elements		<i>Strain:</i> SF162, SF162ΔV2	<i>HIV component:</i> gp140	<i>Stimulatory Agents:</i> MF-59C
		References: [Barnett (2001)]				
		<ul style="list-style-type: none"> • SF162ΔV2 is a virus that has a 30 amino acid deletion in the V2 loop that does not abrogate its infectivity but renders it highly susceptible to neutralization – when incorporated into a codon-optimized DNA vaccine with a CMV promoter and delivered by gene gun, SF162ΔV2 gave higher neutralizing Ab titers against SF162 than did SF162 itself, and Abs that cross-neutralized non-homologous primary isolates were obtained only when SF162ΔV2, but not intact SF162, was used as the immunogen – Control MAbs 2F5 and 2G12 could neutralize all of the following primary isolates: 91US056(R5), 92US714(R5), 92US660(R5), 92HT593(R5X4), and BZ167(R5X4), while after the first protein boost, the sera from two SF162ΔV2 immunized macaques could neutralize 91US056(R5), 92US714(R5), 92US660(R5) and ADA(R5), but not 92HT593(R5X4) or 92US657(R5) – the pattern of cross-recognition shifted after the second boost [Barnett (2001)] 				
970	polyclonal	Env()	gp120(SF162ΔV2)		Vaccine	Rhesus macaque()
	Vaccine:	<i>Vector/type:</i> DNA prime with recombinant protein boost		<i>Strain:</i> SF162ΔV2	<i>HIV component:</i> gp140	<i>Stimulatory Agents:</i> MF-59C
		References: [Cherpelis (2001b), Cherpelis (2001a)]				
		<ul style="list-style-type: none"> • Two animals were immunized both intradermally and intramuscularly at weeks 0, 4, and 8 with a codon optimized DNA vector expressing the SF162V2 gp140 envelope with an intact gp120-gp41 cleavage site, and both developed lymphoproliferative responses and potent neutralizing Abs – CD8+ T lymphocytes were depleted in the animals and they were challenged with SHIV162P4 – at peak viremia, plasma viral levels in the vaccinated animals were 1 to 4 logs lower than those in the unvaccinated animals [Cherpelis (2001b)] 				

Table of HIV MAbs

- HIV-1 SF162ΔV2 gp140 envelope was used in a DNA-prime plus protein-boost vaccination methodology in Rhesus macaques, the animals were depleted of their CD8+ T lymphocytes, and challenged with pathogenic SHIV(SF162P4) – the vaccinated macaques had lower peak viremia, rapidly cleared virus from the periphery, and developed delayed seroconversion to SIV core antigens relative to non-vaccinated controls [Cherpelis (2001a)]

971	polyclonal	Env() References: [Ahmad (2001)]	gp160()	no HIV-1 infection	human()
		<ul style="list-style-type: none"> • High CD4+ T-cell count and low viral load was correlated with high ADCC anti-HIV-1 Env Ab titers in a study of 46 HIV-1 infected individuals from all disease stages [Ahmad (2001)] 			
972	polyclonal	Env(dis) References: [Beirnaert (2001)]	gp160(dis)	P HIV-1 infection	human(IgG)
		<ul style="list-style-type: none"> • Neutralizing antibodies are thought to inhibit HIV entry by blocking either binding or fusion – six broadly cross-neutralizing sera that can neutralize group M and O viruses inhibit the binding to PBMCs – the nine primary isolates tested in this study represented very diverse subtypes and recombinant forms, and different co-receptor usage [Beirnaert (2001)] 			
973	polyclonal	Env(dis) References: [Beirnaert (2000)]	gp160(dis)	P HIV-1 infection	human(IgG)
		<ul style="list-style-type: none"> • Sera from 66 HIV individuals from diverse geographic locations could neutralize primary isolates to different extents: broad cross-neutralizing isolates could neutralize 14 primary isolates from HIV-1 group M clades A-H and three O isolates, limited cross-neutralizing sera neutralized some isolates, and non-neutralizing sera – 6/7 broadly neutralizing sera, were from African women despite only 14/66 study subjects being women – ability to neutralize three key isolates, MNlab (envB/gagB, X4 coreceptor), VI525 (envG/gagH, envA/gagA, R5X4) and CA9 (Group O, R5) was predictive of being able to neutralize an additional set of 14 primary isolates [Beirnaert (2000)] 			
974	polyclonal	Env() Vaccine: <i>Vector/type:</i> recombinant protein References: [Bai (2000)]	gp41(539–684 BH10) <i>HIV component:</i> gp41	Vaccine	murine(IgG)
		<ul style="list-style-type: none"> • Murine rsgp41 antisera recognized a common epitope on human IFN-α (aa 29–35 and aa 123–140) and on human IFN-β (aa 31–37 and aa 125–142), suggesting that elevated levels of Ab to IFNs found in HIV+ individuals may be due to a cross-reactive gp41 response [Bai (2000)] 			
975	polyclonal	Env() Vaccine: <i>Vector/type:</i> recombinant protein References: [Bai (2000)]	gp41(539–684 BH10) <i>HIV component:</i> gp41	Vaccine	murine(IgG)
		<ul style="list-style-type: none"> • There is a common epitope in HIV-1 gp41, and IFNα and IFNβ 			

Table of HIV MAbs

976	T20	Env(dis)	gp120(dis IIIB)	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Otteken (1996), Sugiura (1999)]</p> <ul style="list-style-type: none"> • T20: Pulse label experiments of 4 MAbs (D20, D27, T20, and T22) binding to noncleavable gp140 revealed that these anti-CD4BS MAbs bound with a delay, and that the epitope formed with a $t_{1/2}$ of about 10 minutes [Otteken (1996)] • T20: A 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 block CD4 binding [Sugiura (1999)] 						
977	T27	Env(dis)	gp120(dis IIIB)	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>Donor: P. Earl, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD</p> <p>References: [Earl (1994), Sugiura (1999)]</p> <ul style="list-style-type: none"> • T27: A 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 block CD4 binding [Sugiura (1999)] 						
978	T3	Env(dis)	gp41(dis HXB2)		Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> tetrameric Env <i>HIV component:</i> Env</p> <p>References: [Earl (1994), Earl (1997), Zwick (2001b)]</p> <ul style="list-style-type: none"> • 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 [Earl (1997)] • T3: T3 partially competes with MAb Z13, but not MAb 4E10, both of which bind to gp41 proximally to the 2F5 epitope and have a broad neutralizing potential [Zwick (2001b)] 						
979	T30	Env(dis)	gp41(dis)	no	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> tetrameric Env <i>HIV component:</i> Env</p> <p>References: [Earl (1994), Earl (1997)]</p> <ul style="list-style-type: none"> • T30: Binds in 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 [Earl (1997)] 						
980	T4	Env(dis)	gp41(dis IIIB)	L	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> vaccinia <i>Strain:</i> IIIB <i>HIV component:</i> oligomeric gp140</p> <p>References: [Earl (1994), Broder (1994), Richardson (1996), Weissenhorn (1996), Earl (1997), Otteken (1996), Binley (1999), Stamatatos (2000), Srivastava (2002)]</p>						

Table of HIV MAbs

- T4: one of five MAbs (T4, T6, T9, T10 and T35) in a competition group that bind to a conformation-dependent epitope in gp41 and is oligomer specific – neutralizes IIIB and SF2 [Broder (1994)]
 - T4: Does not bind to soluble monomeric gp41(21–166) that lacks the fusion peptide and membrane anchor, only to the oligomer gp140, as does T6 [Weissenhorn (1996)]
 - T4: This antibody, along with 7 others (M10, D41, D54, T6, T9, T10 and T35), can block the linear murine MAb D61, and the human MAb 246-D, which both bind to the immunodominant region near the two Cys in gp41 – most of these antibodies are oligomer dependent – all of the MAbs are reactive with ten different HIV-1 strains – members of this competition group are blocked by sera from HIV-1+ individuals [Earl (1997)]
 - T4: MAbs T4 and T6 bind only to oligomer, and pulse chase experiments indicate that the epitope is very slow to form, requiring one to two hours [Otteken (1996)]
 - T4: 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 [Binley (1999)]
 - T4: Soluble gp140 derived from SF162, a neutralization-resistant primary isolate, and SF162AV2 a neutralization-susceptible isolate with 30 amino acids deleted from the V2 loop, were generated with or without the gp120-gp41 cleavage site intact – all forms are recognized by oligomer-specific MAb T4 and show enhanced binding of CD4i MAb 17b when sCD4 is bound – the fused forms are less efficiently recognized than the cleaved forms by polyclonal neutralizing sera from HIV-infected patients – the V3 loop is more exposed on the fused form [Stamatatos (2000)]
 - T4: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – T4 recognized o-gp140 [Srivastava (2002)]
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Table 14: Nef

MAB ID	HXB2 Location	Author's Location	Sequence	Neutralizing	Immunogen	Species (Isotype)
981 4H4	Nef(1–33)	Nef(1–33 IIIB)	MGGKWSKSSVVGWPTVRER-MRRAPTVRERMRRRAEPAADG-VGAA		Vaccine	human(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> IIIB <i>HIV component:</i> Nef</p> <p>References: [Otake (1994)]</p> <ul style="list-style-type: none"> • 4H4: This MAb, elicited by vaccination with a Nef fusion protein, could not detect Nef protein on the cell surface – C-term anti-Nef Abs could [Otake (1994)] 						
982 polyclonal	Nef(9–24)	Nef(9–24)	SVIGWLTVRERMRRAE	no	Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> DNA <i>Strain:</i> BRU <i>HIV component:</i> Nef</p> <p>References: [Tahtinen (2001)]</p> <ul style="list-style-type: none"> • BALB/c mice were immunized with a pBN-vector expressing HIV-1 nef, rev, or tat genes – DNA loaded onto gold microparticles was delivered using a gene gun, and DNA dissolved in saline was given intradermally or intramuscularly – Nef gene gun immunized mice showed the strongest and most long-lasting (6 months) Ab, CTL and proliferative responses – the highest IgG1/IgG2a ratio was observed in the gene gun immunized mice – three Ab binding sites were found in Nef using peptide mapping, although some sera reacted only to complete Nef – Rev- or-Tat immunized mice did not generate an Ab response [Tahtinen (2001)] 						
983 13/042	Nef(11–20)	Nef(11–24 BH10)	VGWPTVRERM		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef</p> <p>References: [Schneider (1991)]</p> <ul style="list-style-type: none"> • 13/042: Epitope mapped by overlapping decapeptides – core: TVRERM [Schneider (1991)] 						
984 13/035	Nef(15–24)	Nef(11–24 BH10)	TVRERMRRAE		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef</p> <p>References: [Schneider (1991)]</p> <ul style="list-style-type: none"> • 13/035: Epitope mapped by overlapping decapeptides – core: TVRERM [Schneider (1991)] 						
985 AM5C6	Nef(dis 28–43 + 78–92)	Nef(dis 28–43 BH10)	DGVGAASRDLEKHGAI + KA-AVDLSHFLK		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef</p> <p>References: [Schneider (1991)]</p> <ul style="list-style-type: none"> • AM5C6: Epitope mapped by overlapping decapeptides – core: SRDL – also reacts with Nef(78–92) [Schneider (1991)] 						

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986	AM5C6	Nef(dis 28–43 + 78–92)	Nef(dis 28–43 BH10)	DGVGAASRDLEKHGAI + KA-AVDLSHFLK		Vaccine	murine()
		Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef					
		References: [Schneider (1991)]					
		• AM5C6: Epitope mapped by overlapping decapeptides – core: KAAVDL – also reacts with Nef(28–43) [Schneider (1991)]					
987	25/03	Nef(30–43)	Nef(30–43 BH10)	VGAASRDLEKHGAI		Vaccine	murine()
		Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef					
		References: [Schneider (1991)]					
		• 25/03: Epitope mapped by overlapping decapeptides – core: ASRDLEK [Schneider (1991)]					
988	26/76	Nef(30–43)	Nef(30–43 BH10)	VGAASRDLEKHGAI		Vaccine	murine()
		Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef					
		References: [Schneider (1991)]					
		• 26/76: Epitope mapped by overlapping decapeptides – core: SRDLEK [Schneider (1991)]					
989	3F2	Nef(31–40)	Nef(31–40 BRU)	GAASRDLEKH		Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BRU <i>HIV component:</i> Nef					
		References: [Ovod (1992), Saito (1994), Ranki (1995)]					
		• 3F2: Reacted with Nef from different HIV-1 strains (BRU, IIIB, RF, MN) [Ovod (1992)]					
		• 3F2: Faintly cross-reactive with astrocytes of uninfected control samples [Ranki (1995)]					
		• 3F2: UK Medical Research Council AIDS reagent: EVA3067.1					
990	3D12	Nef(31–50)	Nef(31–50 BRU)	GAASRDLEKHGAISSNTAA		Vaccine	murine(IgG1)
		Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BRU <i>HIV component:</i> Nef					
		References: [Ovod (1992), Saito (1994), Ranki (1995)]					
		• 3D12: There is an anti-RT MAb that also has this name (see [Chiba (1997)])					
		• 3D12: Reacted with Nef from different HIV-1 strains (BRU, IIIB, RF, MN) [Ovod (1992)]					
		• 3D12: Over-expression of Nef in astrocytes from postmortem pediatric CNS tissues [Saito (1994)]					
		• 3D12: One of four antibodies used in combination to show HIV Nef protein expressed in astrocytes from 7/14 brain samples from HIV+ individuals – Nef expression associated with dementia [Ranki (1995)]					
		• 3D12: UK Medical Research Council AIDS reagent: EVA3067.2					
991	polyclonal	Nef(49–64)	Nef(49–64)	AATNAACAWLEAQEEE	no	Vaccine	murine(IgG)
		Vaccine: <i>Vector/type:</i> DNA <i>Strain:</i> BRU <i>HIV component:</i> Nef					
		References: [Tahtinen (2001)]					

- BALB/c mice were immunized with a pBN-vector expressing HIV-1 nef, rev, or tat genes – DNA loaded onto gold microparticles was delivered using a gene gun, and DNA dissolved in saline was given intradermally or intramuscularly – Nef gene gun immunized mice showed the strongest and most long-lasting (6 months) Ab, CTL and proliferative responses – the highest IgG1/IgG2a ratio was observed in the gene gun immunized mice – three Ab binding sites were found in Nef using peptide mapping, although some sera reacted only to complete Nef – Rev- or-Tat immunized mice did not generate an Ab response [Tahtinen (2001)]

992	3G12	Nef(51–71)	Nef(51–71 BRU)	TNAACAWLEAQEEEEVGFPVT	Vaccine	murine(IgG2a)	
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BRU <i>HIV component:</i> Nef							
References: [Ovod (1992)]							
● 3G12: Reacted with Nef from different HIV-1 strains (BRU, IIIB, RF, MN) [Ovod (1992)]							
993	13/058	Nef(60–73)	Nef(60–73 BH10)	AQEEEEVGFPVTPQ	Vaccine	murine()	
Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef							
References: [Schneider (1991)]							
● 13/058: Epitope mapped by overlapping decapeptides – core: EEVGFP [Schneider (1991)]							
994	26/028	Nef(60–73)	Nef(60–73 BH10)	AQEEEEVGFPVTPQ	Vaccine	murine()	
Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef							
References: [Schneider (1991)]							
● 26/028: Epitope mapped by overlapping decapeptides – core: EEVGFPV [Schneider (1991)]							
995	2E3	Nef(61–80)	Nef(61–80 BRU)	QEEEEVGFPVTPQVPLRPMT	Vaccine	murine(IgG1)	
Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BRU <i>HIV component:</i> Nef							
References: [Ovod (1992), Nilsen (1996)]							
● 2E3: There are two MAbs with the name 2E3 – the other one binds to integrase [Nilsen (1996)]							
● 2E3: Two isomorphous forms of Nef were identified, 2E3 reacted with the p24 but not p27 form, and was strain specific (MN and BRU reactive, not IIIB or RF) [Ovod (1992)]							
996	polyclonal	Nef(66–97)	Nef(66–97 LAI)	VGFPVTPQVPLRPMTYKAAV- DLSHFLKEKGG	no	Vaccine	human(IgG)
Vaccine: <i>Vector/type:</i> lipopeptide <i>Strain:</i> LAI <i>HIV component:</i> Nef <i>Stimulatory Agents:</i> QS21							
References: [Pialoux (2001)]							
● 28 subjects were vaccinated with six HIV-1 peptides that were selected to be particularly rich in CTL epitopes, presented in lipopeptides with or without adjuvant QS21 – HIV-specific Ab responses were detected in 10/28, proliferative in 11/14, and CTL in 13/24 (54%) of testable volunteers – 10/28 had Ab responses to this peptide (N1), 11/24 had proliferative responses, and CTL responses were detected [Pialoux (2001)]							

Table of HIV MAbs

997	F14.11	Nef(83–88)	Nef(83–88)	AAVDLS	Vaccine	murine(IgG2a κ)
<p>Vaccine: <i>Vector/type:</i> peptide <i>HIV component:</i> Nef</p> <p>References: [De Santis (1991), Chang (1998)]</p> <ul style="list-style-type: none"> • F14.11: The MAb was made to a six aa region of Nef that is similar to a region found in thymosin alpha 1 protein – the MAb binds to the natural Nef protein [De Santis (1991)] • F14.11: Used as a control in a study of Nef-specific single chain Abs constructed from AG11 and EH1 [Chang (1998)] 						
998	31/03	Nef(83–103)	Nef(82–103 BH10)	AAVDLSHFLKEKGGLEGLIHS	Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef</p> <p>References: [Schneider (1991)]</p> <ul style="list-style-type: none"> • 31/03: Epitope mapped by overlapping decapeptides – mapping suggests complex epitope in this region [Schneider (1991)] 						
999	polyclonal	Nef(dis Nef)	Nef(dis 117–147 LAI)	TQGYFPDWQNYTPGPGVRYP-LTFGWYKLV	no Vaccine	human(IgG)
<p>Vaccine: <i>Vector/type:</i> lipopeptide <i>Strain:</i> LAI <i>HIV component:</i> Nef <i>Stimulatory Agents:</i> QS21</p> <p>Ab type: Nef References: [Pialoux (2001)]</p> <ul style="list-style-type: none"> • 28 subjects were vaccinated with six HIV-1 peptides that were selected to be particularly rich in CTL epitopes, presented in lipopeptides with or without adjuvant QS21 – HIV-specific Ab responses were detected in 25/28, proliferative in 3/24, and CTL in 13/24 (54%) of testable volunteers – 20/28 had antibody responses to this particular peptide (N2), 3/24 had proliferative responses, and CTL responses were detected [Pialoux (2001)] 						
1000	polyclonal	Nef(118–133)	Nef(118–133)	QGYFPDWQNYTPGPGV	no Vaccine	murine(IgG)
<p>Vaccine: <i>Vector/type:</i> DNA <i>Strain:</i> BRU <i>HIV component:</i> Nef</p> <p>References: [Tahtinen (2001)]</p> <ul style="list-style-type: none"> • BALB/c mice were immunized with a pBN-vector expressing HIV-1 nef, rev, or tat genes – DNA loaded onto gold microparticles was delivered using a gene gun, and DNA dissolved in saline was given intradermally or intramuscularly – Nef gene g unimmunized mice showed the strongest and most long-lasting (6 months) Ab, CTL and proliferative responses – the highest IgG1/IgG2a ratio was observed in the gene gun immunized mice – three Ab binding sites were found in Nef using peptide mapping, although some sera reacted only to complete Nef – Rev- or-Tat immunized mice did not generate an Ab response [Tahtinen (2001)] 						
1001	F1	Nef(148–157)	Nef(148–157 IIIB)	VEPDKVEEAN		murine(IgM)
<p>References: [Fujii (1993), Otake (1994), Fujii (1996c), Fujii (1996b)]</p> <ul style="list-style-type: none"> • F1: The C-term end of Nef is accessible to Abs at the cell surface – stained IIIB/M10, but not MN/M10, cells [Otake (1994), Fujii (1993)] • F1: Insect cells expressing myristylated Nef proteins on their cell surface can induce cytolysis of unstimulated CD4+ cells – this response is not due to MHC restricted CTL activity – the cell surface of Nef expressing insect cells carry Nef that can be recognized by MAbs E7 and E9 but not F1 [Fujii (1996c)] • F1: A carboxy-terminal domain of Nef on the cell surface induces cytolysis of CD4+ T cells [Fujii (1996b)] 						

Table of HIV MAbs

1002	2F2	Nef(151–170)	Nef(151–170 BRU)	DKVEEANKGENTSLLHPVSL	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef</p> <p>References: [Ovod (1992), Saito (1994), Ranki (1995)]</p> <ul style="list-style-type: none"> • 2F2: Strain specific (MN and BRU reactive, not IIIB or RF) [Ovod (1992)] • 2F2: Over-expression of Nef in astrocytes from postmortem pediatric CNS tissue [Saito (1994)] • 2F2: One of four antibodies used in combination to show HIV Nef protein expressed in astrocytes from 7/14 brain samples from HIV+ individuals – Nef expression associated with dementia [Ranki (1995)] • 2F2: UK Medical Research Council AIDS reagent: EVA3067.3 						
1003	E9	Nef(158–181)	Nef(158–206 IIIB)	KGENTSLLHPVSLHGMDDPER-EVL		murine(IgM)
<p>References: [Fujii (1993), Otake (1994), Fujii (1996c), Fujii (1996b)]</p> <ul style="list-style-type: none"> • E9: The C-term end of Nef is accessible to Abs at the cell surface – stained IIIB/M10, but not MN/M10, cells [Otake (1994), Fujii (1993)] • E9: A carboxy-terminal domain of Nef on the cell surface induces cytolysis of CD4+ T cells [Fujii (1996b)] • E9: Insect cells expressing myristylated Nef proteins on their cell surface can induce cytolysis of unstimulated CD4+ cells – this response is not due to MHC restricted CTL activity – the cell surface of Nef expressing insect cells carry Nef that can be recognized by MAbs E7 and E9 but not F1 [Fujii (1996c)] 						
1004	3E6	Nef(161–180)	Nef(161–180 BRU)	NTSLLHPVSLHGMDDPEREV	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BRU <i>HIV component:</i> Nef</p> <p>References: [Ovod (1992), Saito (1994), Ranki (1995)]</p> <ul style="list-style-type: none"> • 3E6: Reacted with Nef from different HIV-1 strains (BRU, IIIB, RF, MN) [Ovod (1992)] • 3E6: Faintly cross-reactive with astrocytes of uninfected control samples [Ranki (1995)] • 3E6: UK Medical Research Council AIDS reagent: EVA3067.4 						
1005	2A3	Nef(171–190)	Nef(171–190 BRU)	HGMDDPEREVLEWRFD SRLA	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BRU <i>HIV component:</i> Nef</p> <p>References: [Ovod (1992)]</p> <ul style="list-style-type: none"> • 2A3: Reacted with Nef from different HIV-1 strains (BRU, IIIB, MN, but not RF) [Ovod (1992)] 						
1006	2E4	Nef(171–190)	Nef(171–190 BRU)	HGMDDPEREVLEWRFD SRLA	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BRU <i>HIV component:</i> Nef</p> <p>References: [Ovod (1992)]</p> <ul style="list-style-type: none"> • 2E4: Reacted with Nef from different HIV-1 strains (BRU, IIIB, MN but not RF) [Ovod (1992)] 						
1007	3A2	Nef(171–190)	Nef(171–190 BRU)	HGMDDPEREVLEWRFD SRLA	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BRU <i>HIV component:</i> Nef</p>						

Table of HIV MAbs

<p>References: [Ovod (1992), Saito (1994), Ranki (1995)]</p> <ul style="list-style-type: none"> • 3A2: Reacted with Nef from different HIV-1 strains (BRU, IIIB, RF, MN) [Ovod (1992)] • 3A2: Over-expression of Nef in astrocytes from postmortem pediatric CNS tissue [Saito (1994)] • 3A2: One of four antibodies used in combination to show HIV Nef protein expressed in astrocytes from 7/14 brain samples from HIV+ individuals – Nef expression associated with dementia [Ranki (1995)] • 3A2: UK Medical Research Council AIDS reagent: EVA3067.5 						
1008	2H12	Nef(171–190)	Nef(171–190 BRU)	HGMDDPEREVLEWRFD SRLA	Vaccine	murine(IgG1)
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BRU <i>HIV component:</i> Nef</p> <p>Ab type: Nef References: [Ovod (1992), Saito (1994), Ranki (1995)]</p> <ul style="list-style-type: none"> • 2H12: Reacted with Nef from different HIV-1 strains (BRU, IIIB, RF, MN) [Ovod (1992)] • 2H12: Over-expression of Nef in astrocytes from postmortem pediatric CNS tissue [Saito (1994)] • 2H12: One of four antibodies used in combination to show HIV Nef protein expressed in astrocytes from 7/14 brain samples from HIV+ individuals – Nef expression associated with dementia [Ranki (1995)] 						
1009	NF1A1	Nef(173–206)	Nef(173–206)	MDDPEREVLEWRFD SRLAFH-HVARELHPEYFKNC		murine()
<p>References: [Kaminchik (1990)]</p> <ul style="list-style-type: none"> • NF1A1: Recognizes the Nef protein of the two isolates BH10 and LAV1 – low affinity [Kaminchik (1990)] 						
1010	polyclonal	Nef(182–205)	Nef(182–205 LAI)	EWRFD SRLAFHHVARELHPEY-FKN	no Vaccine	human(IgG)
<p>Vaccine: <i>Vector/type:</i> lipopeptide <i>Strain:</i> LAI <i>HIV component:</i> Nef <i>Stimulatory Agents:</i> QS21</p> <p>References: [Pialoux (2001)]</p> <ul style="list-style-type: none"> • 28 subjects were vaccinated with six HIV-1 peptides that were selected to be particularly rich in CTL epitopes, presented in lipopeptides with or without adjuvant QS21 – HIV-specific Ab responses were detected in 0/28, proliferative in 9/14, and CTL were detected in some of the testable volunteers [Pialoux (2001)] 						
1011	E7	Nef(192–206)	Nef(192–206 IIIB)	HHVARELHPEYFKNC		murine(IgM)
<p>References: [Fujii (1993), Otake (1994), Fujii (1996c), Fujii (1996a), Fujii (1996b), Fujii (1996d)]</p> <ul style="list-style-type: none"> • E7: The C-term end of Nef is accessible to Abs at the cell surface – stained IIIB/M10, but not MN/M10, cells [Otake (1994), Fujii (1993)] • E7: Insect cells expressing myristylated Nef proteins on their cell surface can induce cytolysis of unstimulated CD4+ cells – this response is not due to MHC restricted CTL activity – the cell surface of Nef expressing insect cells carry Nef that can be recognized by MAbs E7 and E9 but not F1 [Fujii (1996c)] • E7: Nef forms a homomeric oligomerizing structure, and using E7 and membrane immunofluorescence or immunoelectron microscopy, was shown to clusters on the surface of HIV-1 infected CD4+ cells [Fujii (1996a)] • E7: A carboxy-terminal domain of Nef on the cell surface induces cytolysis of CD4+ T cells [Fujii (1996b)] 						

- E7: Soluble Nef inhibits proliferation of CD4+ cells, and Nef cross-linking by MAbs may induce anti-CD4 cytotoxic activity – sera from HIV+ individuals contain soluble Nef, thus this may be important for immune dysfunction and disease progression [Fujii (1996d)]

1012	AE6	Nef(194–206)	Nef(LAI)	VARELHPEYFKNC	Vaccine	murine(IgG1 κ)
Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef						
Ab type: C-term Donor: Frank Jirik, Centre for Molecular Med and Therapeutics, U. B. C., Vancouver, B. C. Canada						
References: [Chang (1998)]						
<ul style="list-style-type: none"> • AE6: The light and heavy chains of three MABs (AG11, AE6, EH1) specific to C-terminus of NEF were cloned and variable regions sequenced – the complementarity determining regions (CDR) of AG11 and AE6 were highly related (95.1% at the DNA level) and bound LAI Nef, but not SF2 Nef – EH1 bound to SF2 and LAI and cross-competed AG11 and AE6 but had a distinctive CDR (57.9% similar to AG11) – single chain Abs were constructed from AG11 and EH1 [Chang (1998)] 						
1013	AG11	Nef(194–206)	Nef(LAI)	VARELHPEYFKNC	Vaccine	murine(IgG1 κ)
Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef						
Ab type: C-term Donor: Frank Jirik, Centre for Molecular Med and Therapeutics, U. B. C., Vancouver, B. C. Canada						
References: [Chang (1998)]						
<ul style="list-style-type: none"> • AG11: The light and heavy chains of three MABs (AG11, AE6, EH1) specific to C-terminus of NEF were cloned and variable regions sequenced – the complementarity determining regions (CDR) of AG11 and AE6 were highly related (95.1% at the DNA level) and bound LAI Nef, but not SF2 Nef – EH1 bound to SF2 and LAI and cross-competed AG11 and AE6 but had a distinctive CDR (57.9% similar to AG11) – single chain Abs were constructed from AG11 and EH1 and subcloned into a eukaryotic expression vector with a green fluorescent protein marker to allow intracellular expression – the single chain Abs bind Nef intracellularly and may be useful to better understand the role of Nef and as a gene therapy model [Chang (1998)] 						
1014	EH1	Nef(194–206)	Nef(SF2)	MARELHPEYYKDC	Vaccine	murine(IgG1 κ)
Vaccine: <i>Vector/type:</i> recombinant protein <i>HIV component:</i> Nef						
Ab type: C-term Donor: Frank Jirik, Centre for Molecular Med and Therapeutics, U. B. C., Vancouver, B. C. Canada						
References: [Chang (1998)]						
<ul style="list-style-type: none"> • EH1: The light and heavy chains of three MABs (AG11, AE6, EH1) specific to C-terminus of NEF were cloned and variable regions sequenced – the complementarity determining regions (CDR) of AG11 and AE6 were highly related (95.1% at the DNA level) and bound LAI Nef, but not SF2 Nef – EH1 bound to SF2 and LAI and cross-competed AG11 and AE6 but had a distinctive CDR (57.9% similar to AG11) – single chain Abs were constructed from AG11 and EH1 and subcloned into a eukaryotic expression vector with a green fluorescent protein marker to allow intracellular expression – the single chain Abs bind Nef intracellularly and may be useful to better understand the role of Nef and as a gene therapy model [Chang (1998)] 						

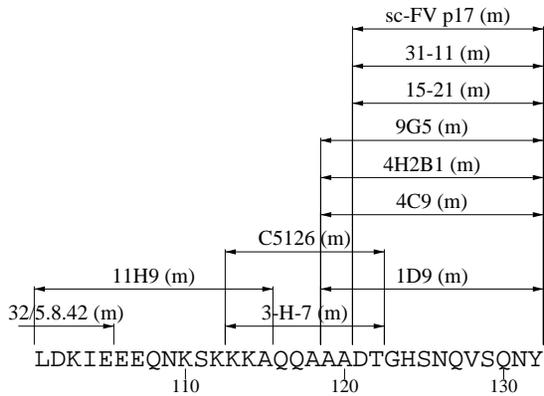
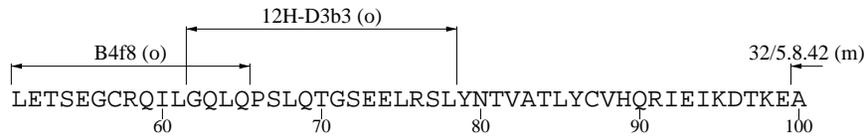
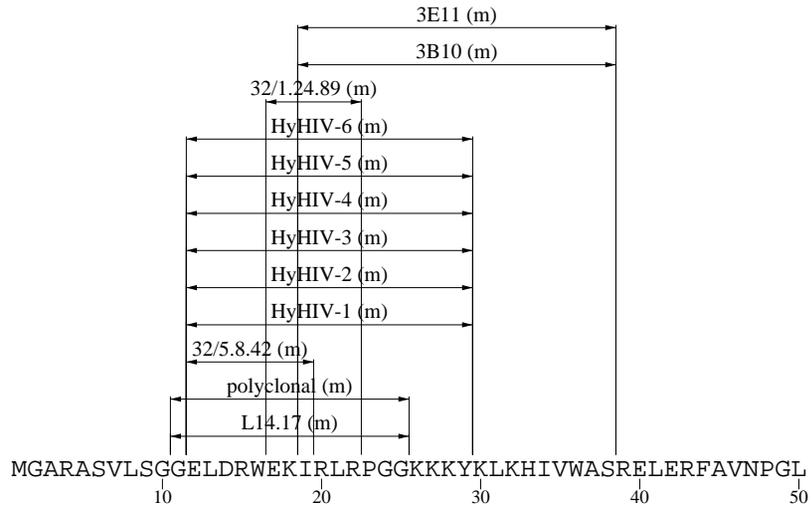
Table of HIV MAbs

1015	6.1	Nef(dis 167–182, 191–205, 193–206)	Nef(dis JR-CSF)			murine()
<p>References: [Ranki (1995)]</p> <ul style="list-style-type: none"> • 6.1: Raised against CNS primary isolates, stains astrocytes more densely than other Nef MAbs – Nef expression associated with dementia [Ranki (1995)] • 6.1: NIAID Repository number 1123 [Ranki (1995)] 						
1016	AE6	Nef()	Nef()			murine()
<p>Ab type: C-term Donor: James Hoxie, Div of AIDS, NIAID, NIH</p> <p>References: [Greenway (1994), Tornatore (1994)]</p> <ul style="list-style-type: none"> • AE6: NIH AIDS Research and Reference Reagent Program: 709 						
1017	NF2B2	Nef()	Nef(20–78 BH10)		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> Nef</p> <p>References: [Kaminchik (1990)]</p> <ul style="list-style-type: none"> • NF2B2: Recognizes the Nef protein of the two isolates BH10 and LAV1 [Kaminchik (1990)] • NF2B2: NIH AIDS Research and Reference Reagent Program: 456 						
1018	NF3A3	Nef()	Nef(20–78 BH10)		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> Nef</p> <p>References: [Kaminchik (1990)]</p> <ul style="list-style-type: none"> • NF3A3: Recognizes the Nef protein of the two isolates BH10 and LAV1 – low affinity [Kaminchik (1990)] 						
1019	NF8B4	Nef(dis)	Nef(dis BH10)		Vaccine	murine()
<p>Vaccine: <i>Vector/type:</i> recombinant protein <i>Strain:</i> BH10 <i>HIV component:</i> Nef</p> <p>References: [Kaminchik (1990)]</p> <ul style="list-style-type: none"> • NF8B4: Does not recognize Nef CNBr cleavage products – recognizes intact BH10 Nef but not LAV1 Nef [Kaminchik (1990)] 						

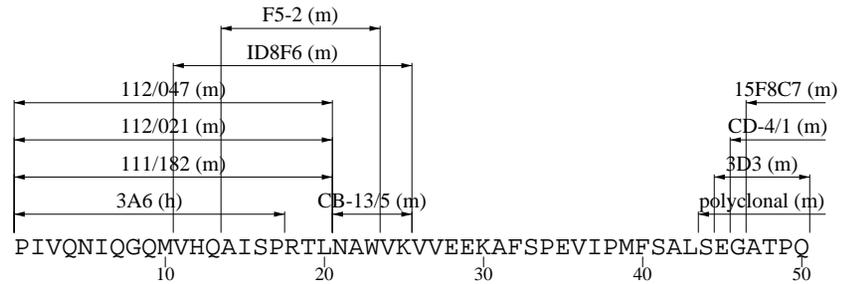
Part IV-C: Maps of MAb Locations Plotted by Protein

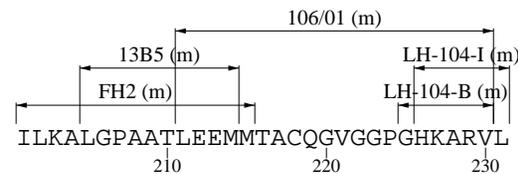
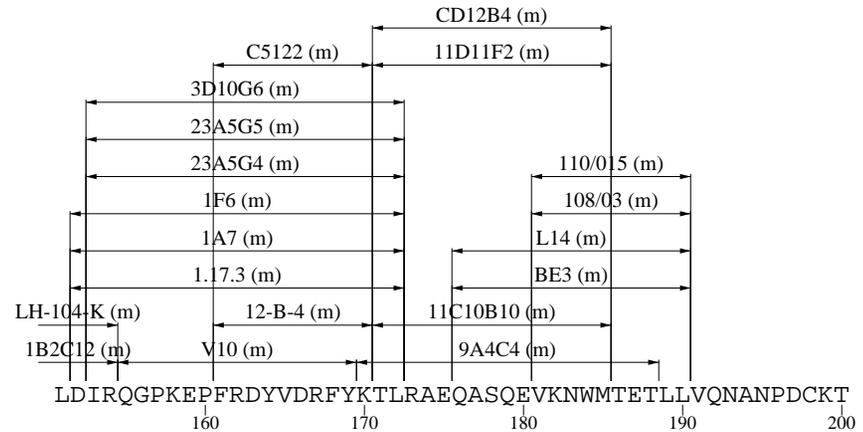
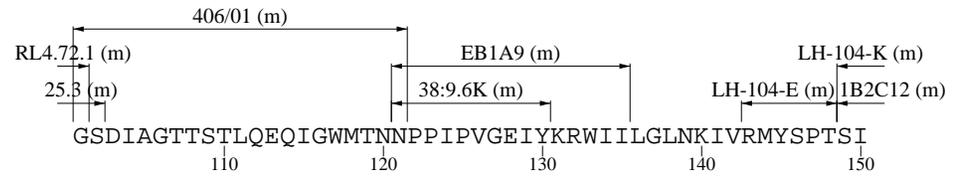
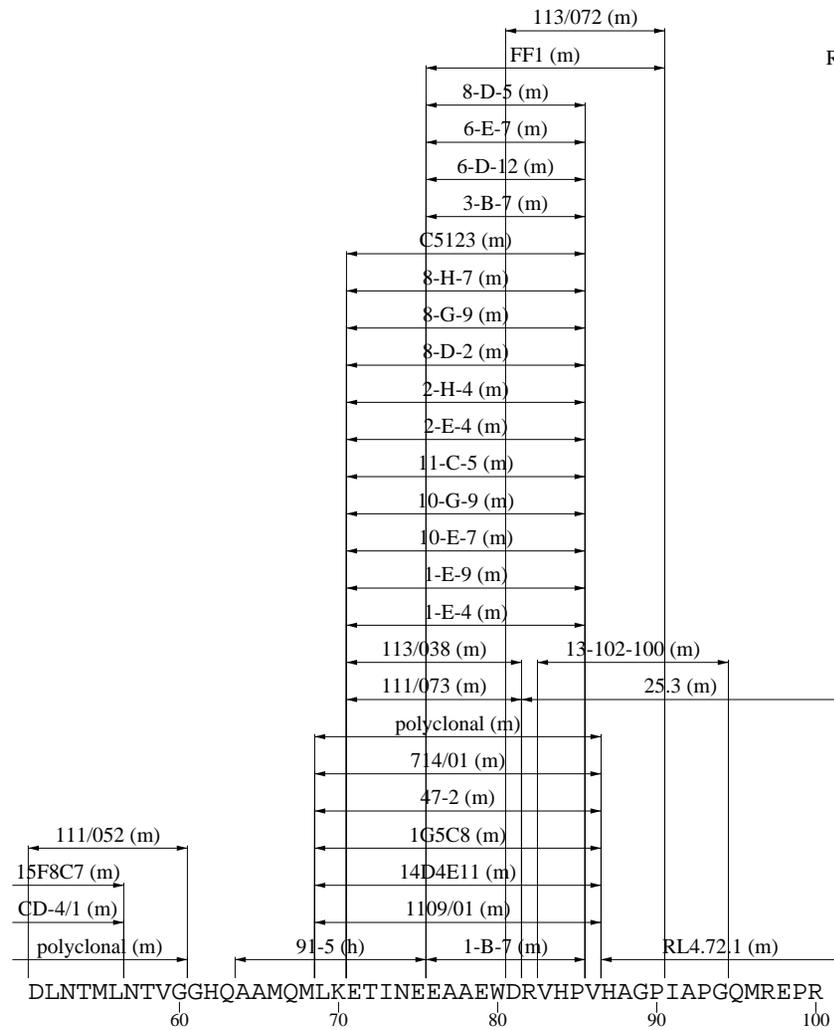
Only linear epitopes <22 amino acids long are shown with their antibody ID. Abbreviations: (h) human, (p) non-human primate, (m) murine, (o) other.

p17 Antibody Map

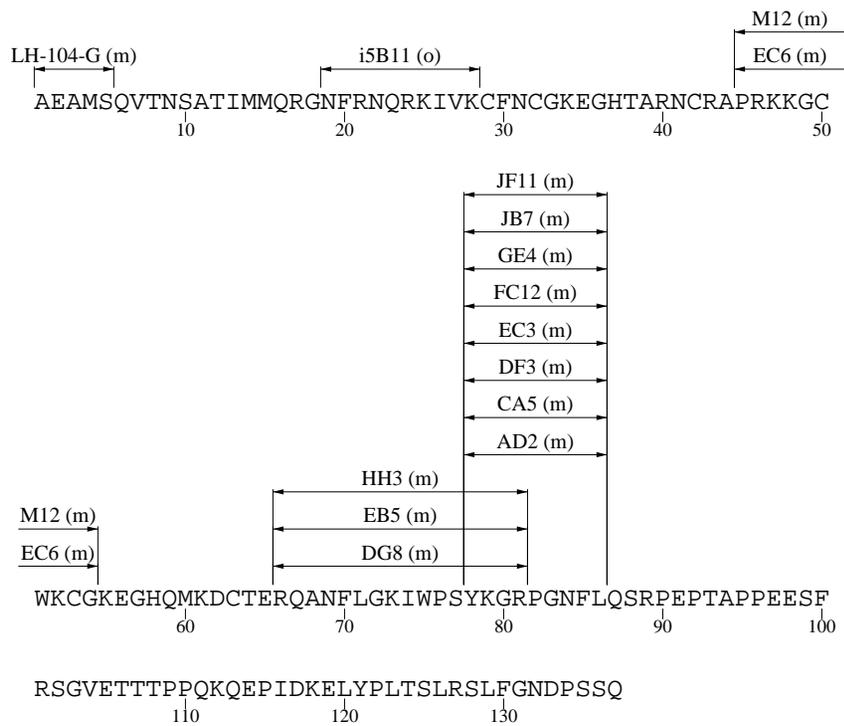


p24 Antibody Map

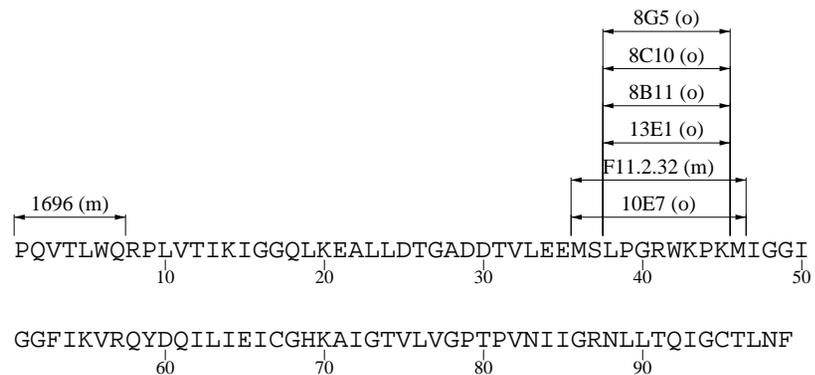




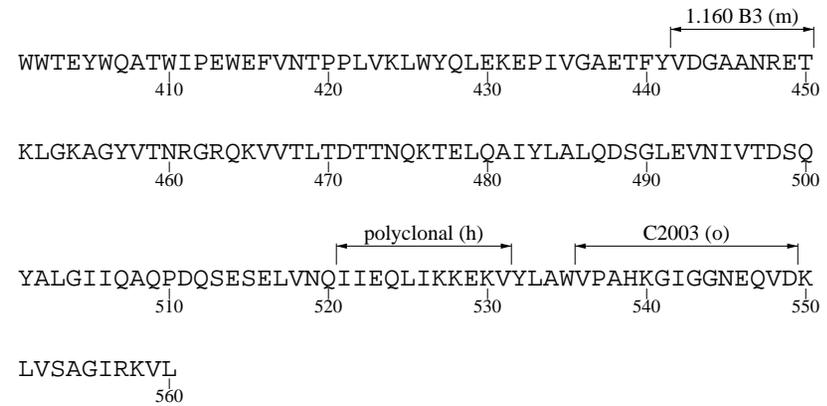
p2p7p1p6 Antibody Map



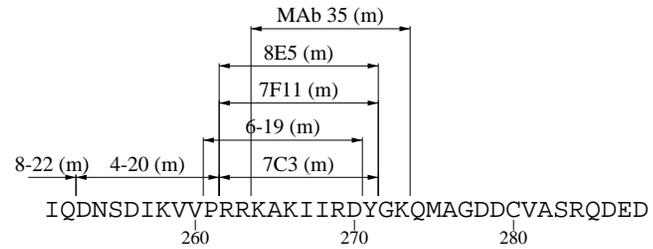
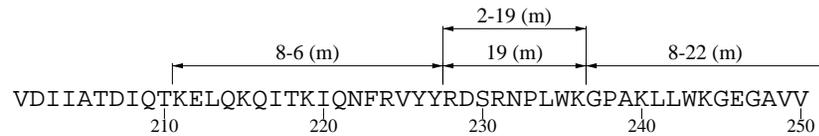
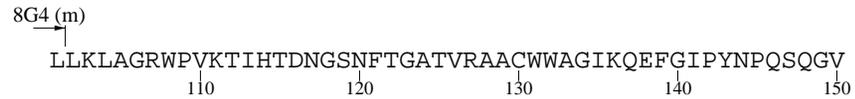
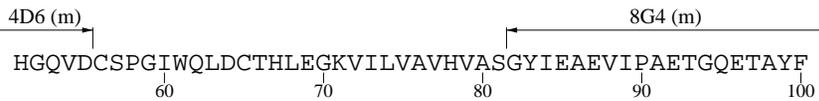
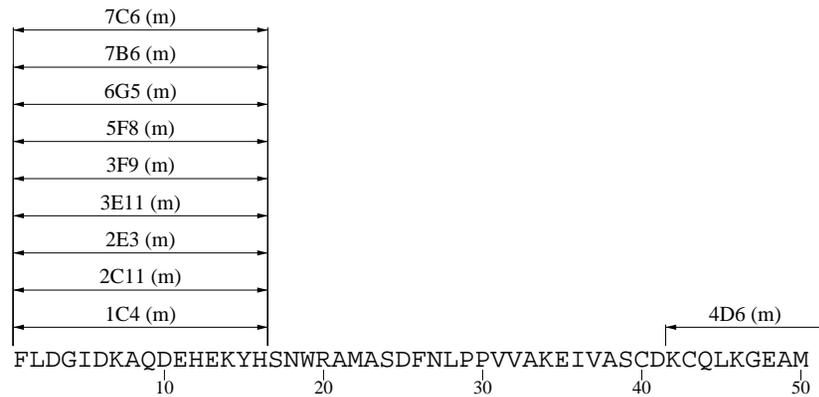
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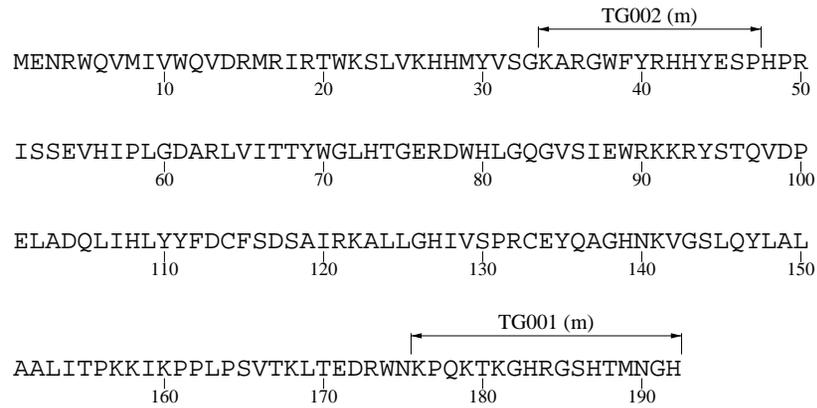
RT Antibody Map



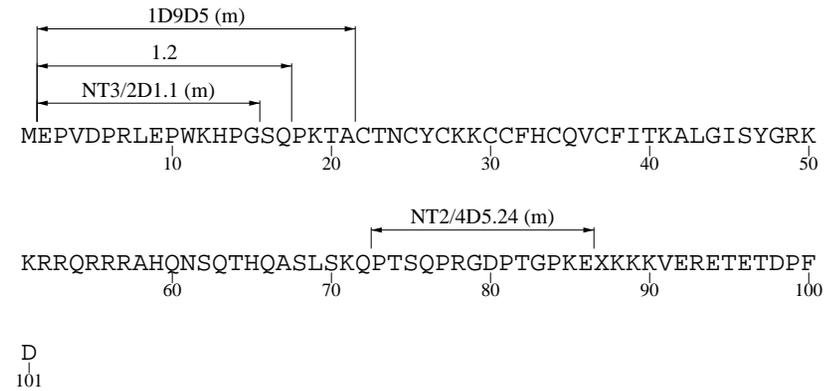
Integrase Antibody Map



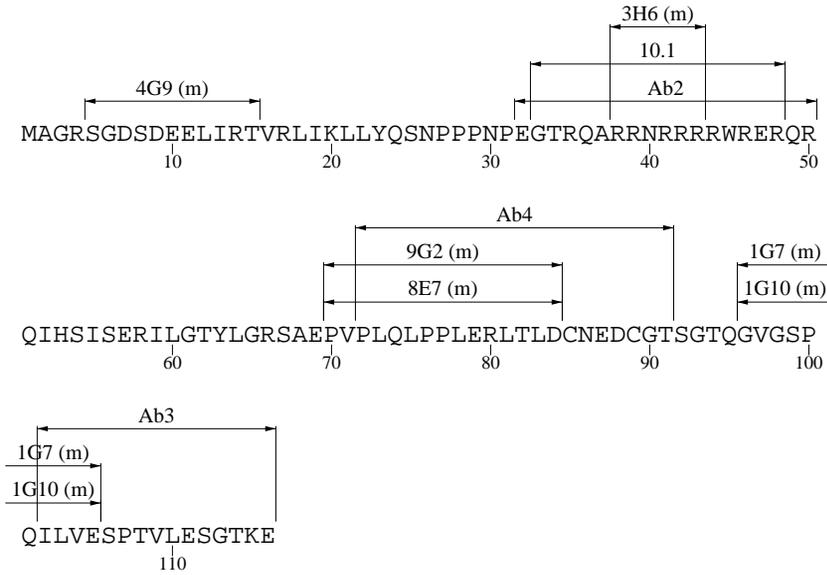
Vif Antibody Map



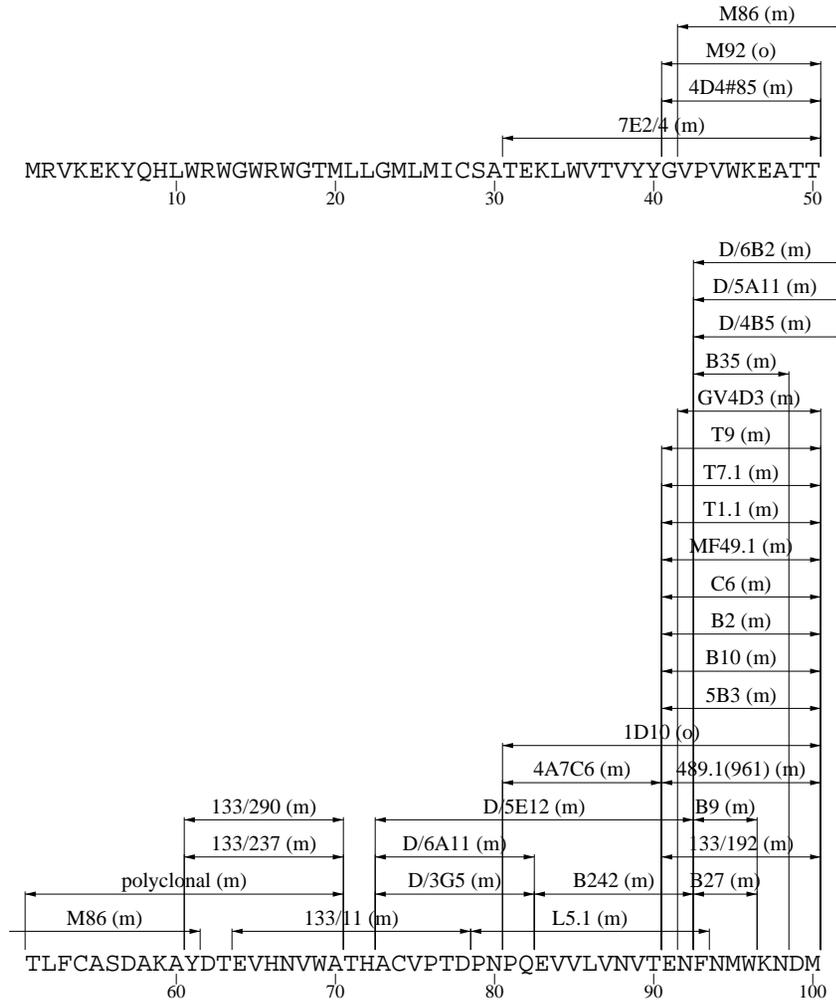
Tat Antibody Map

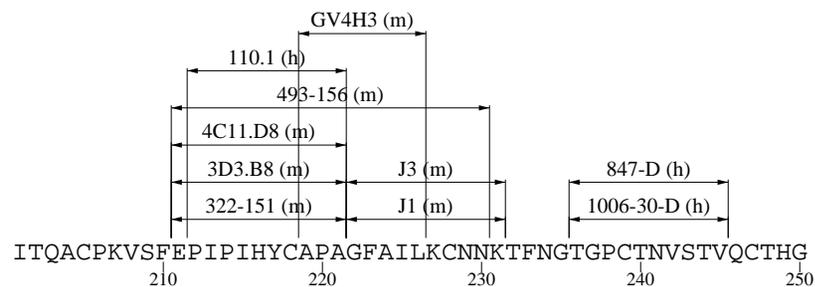
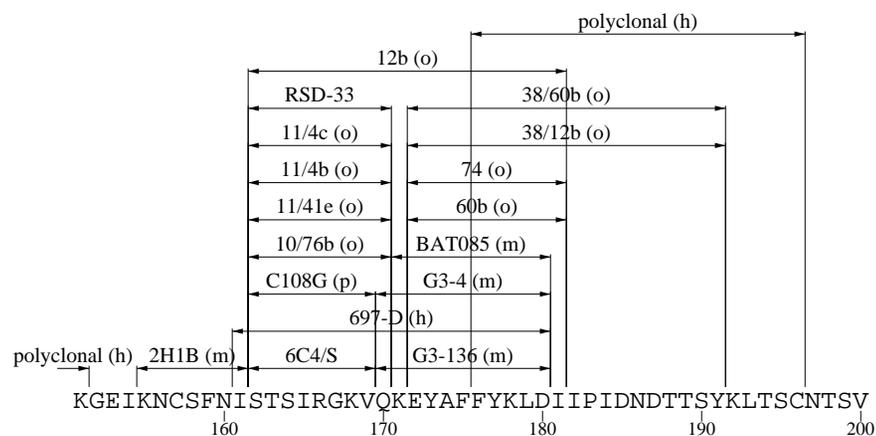
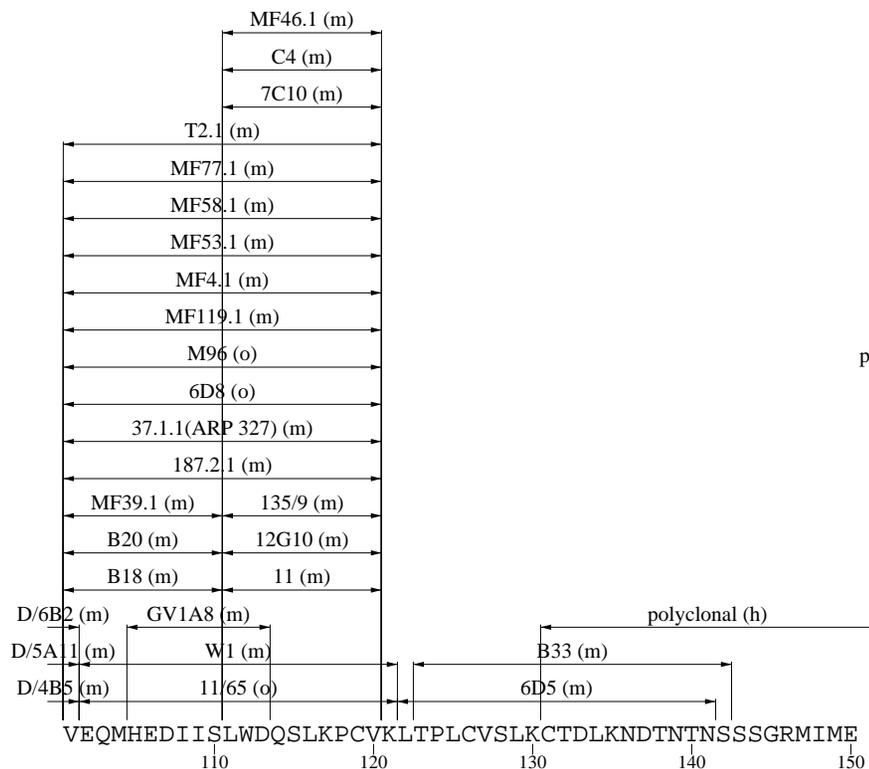


Rev Antibody Map

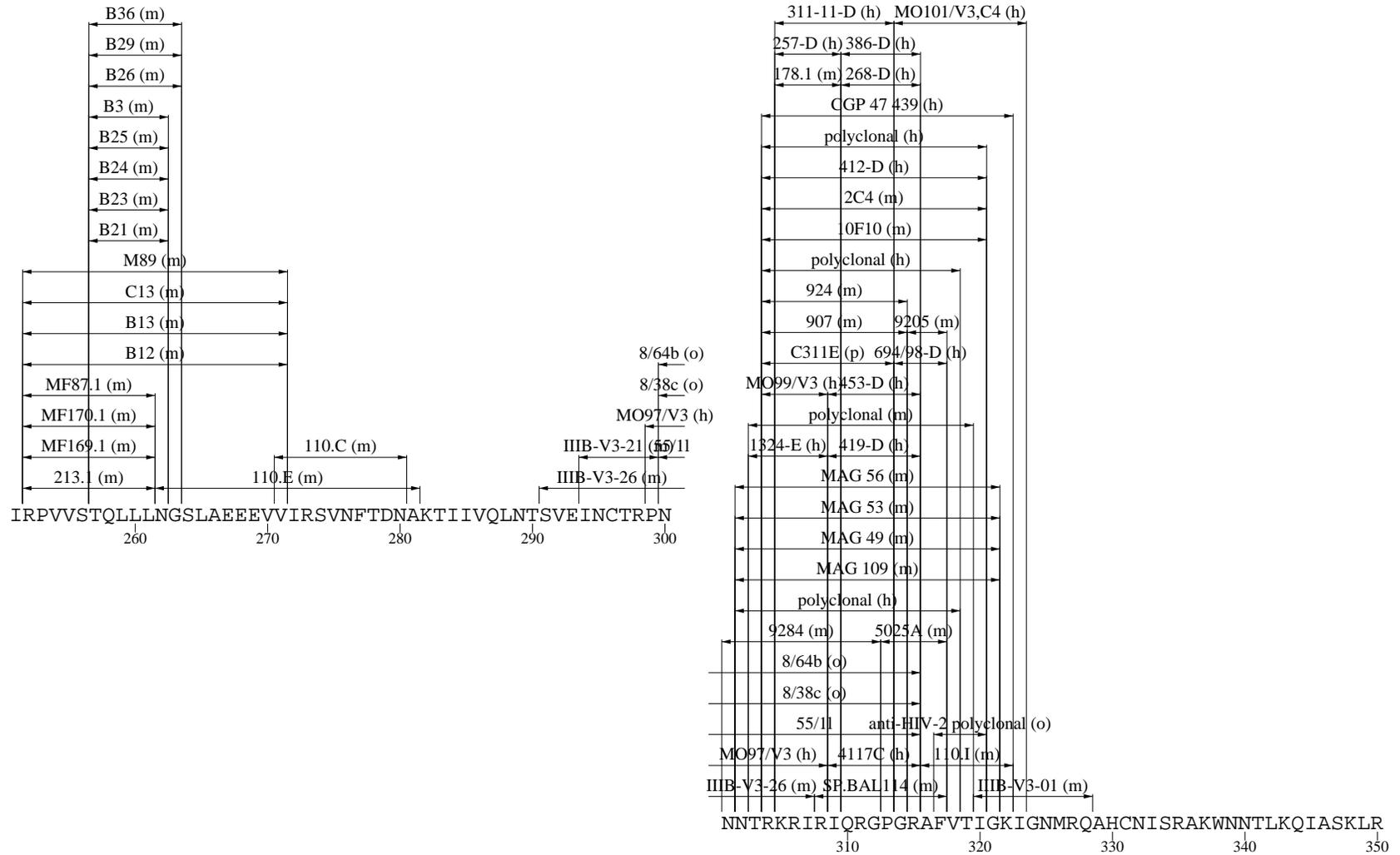


gp160 Antibody Map

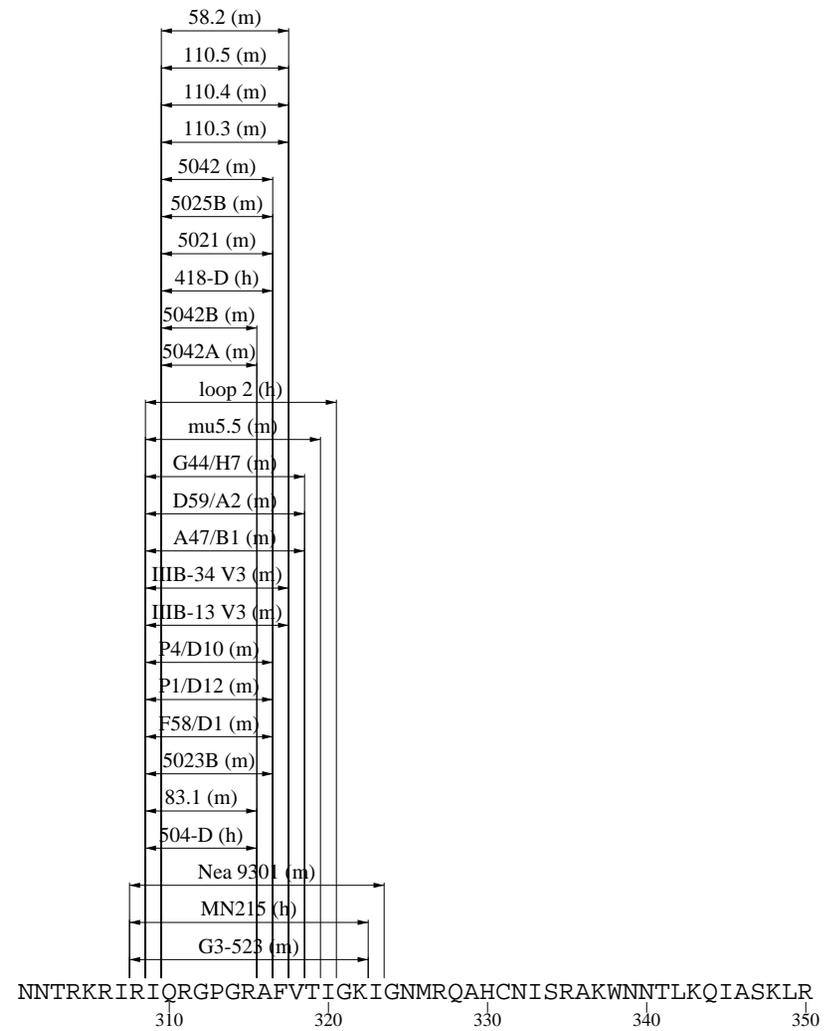
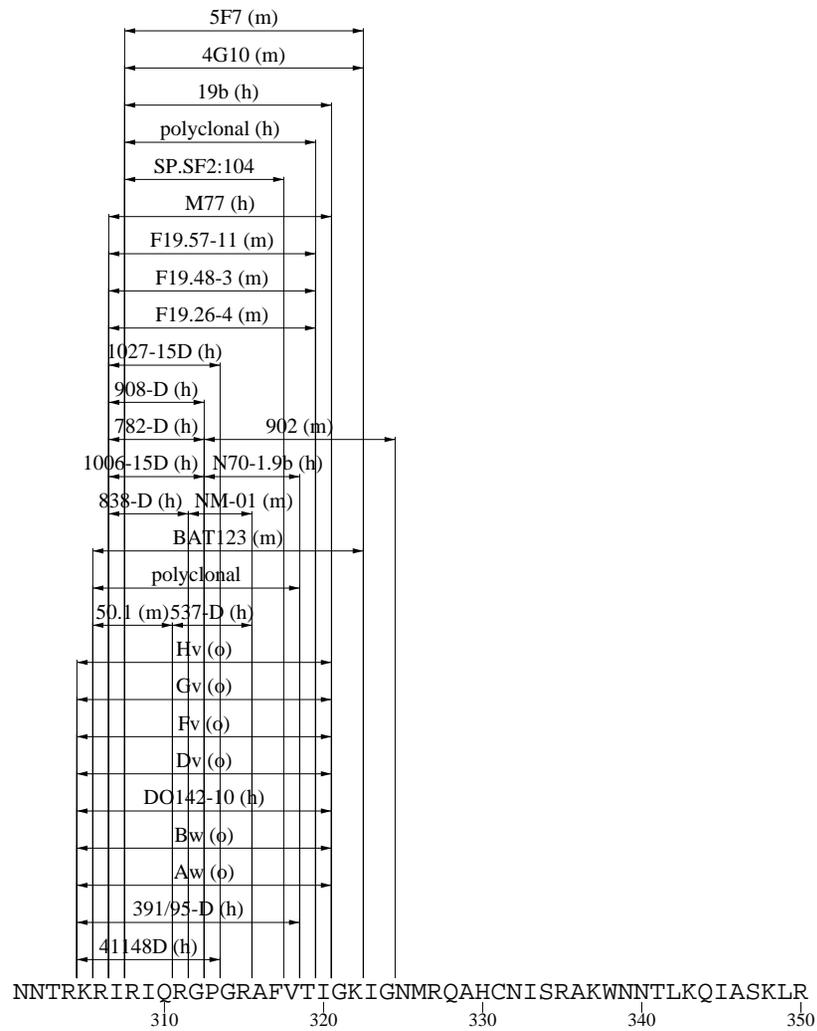




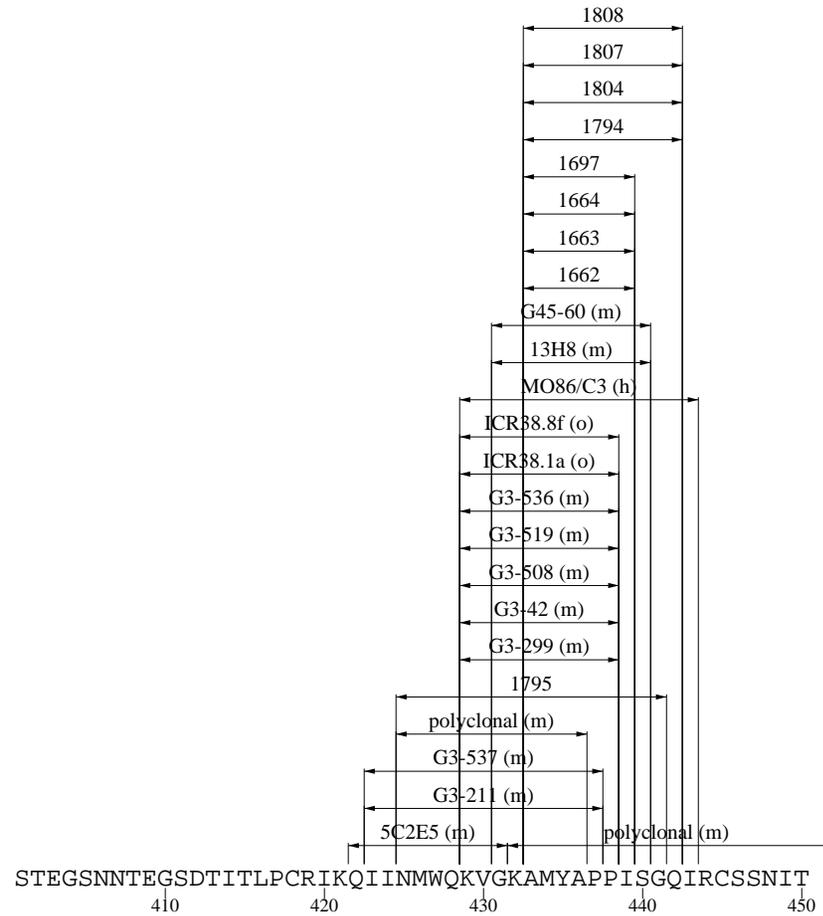
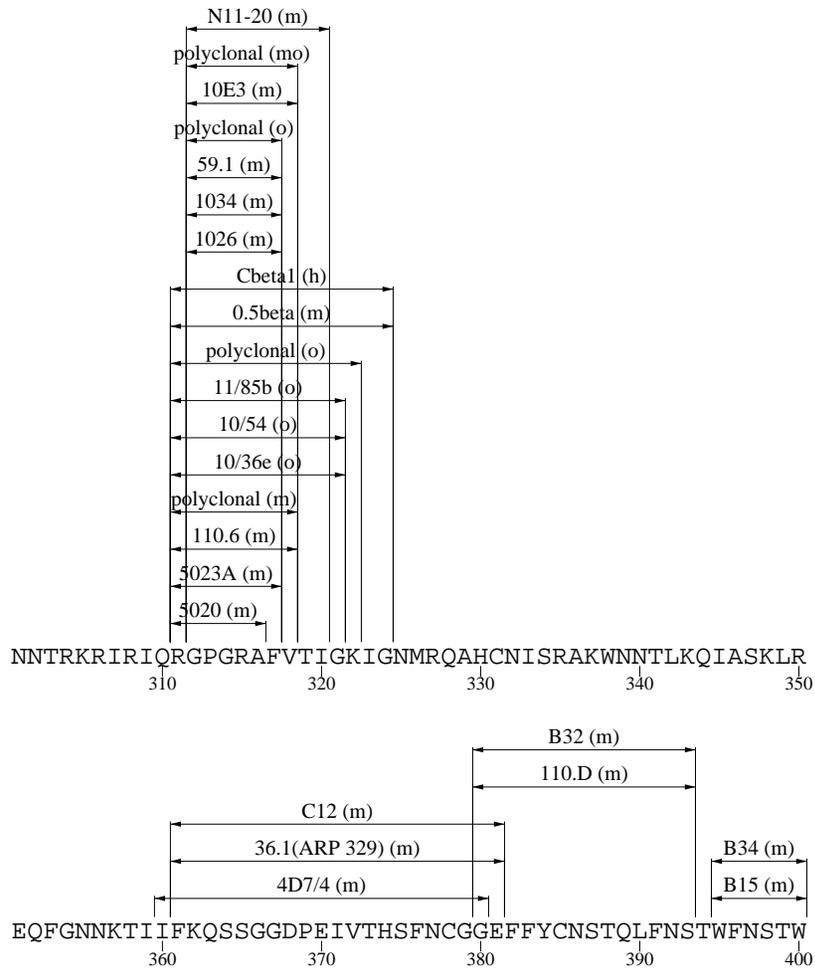
Antibody Protein Maps



B Cell

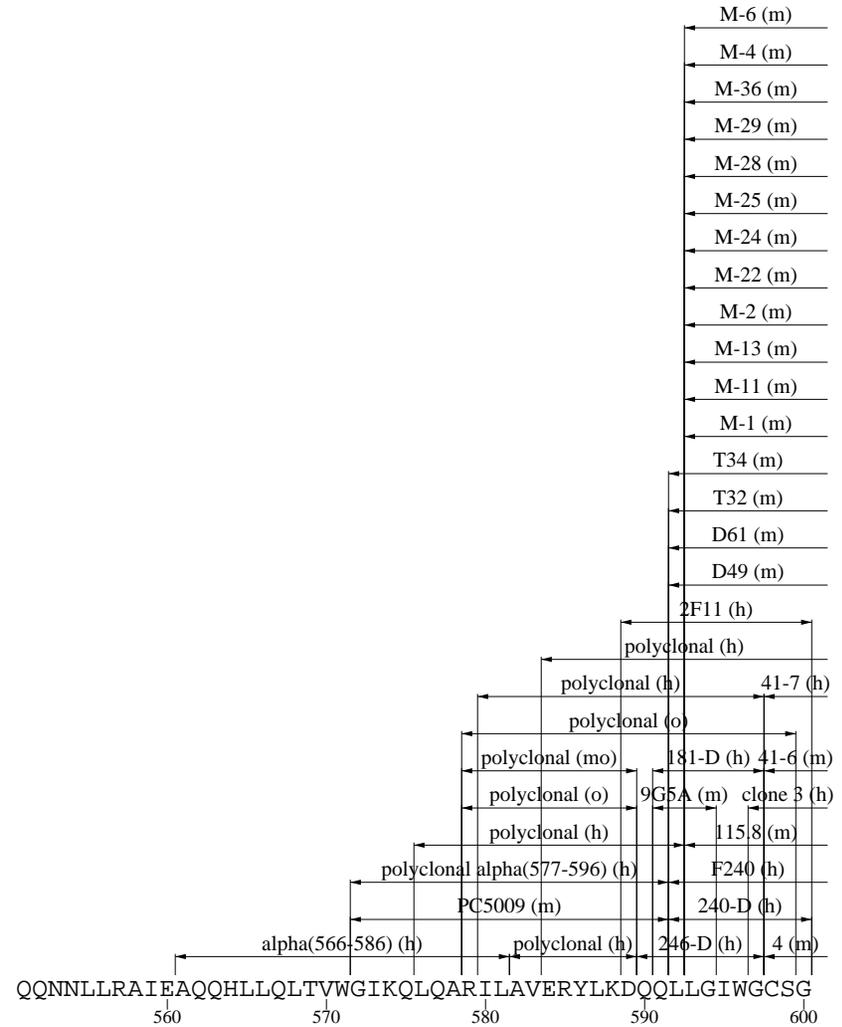
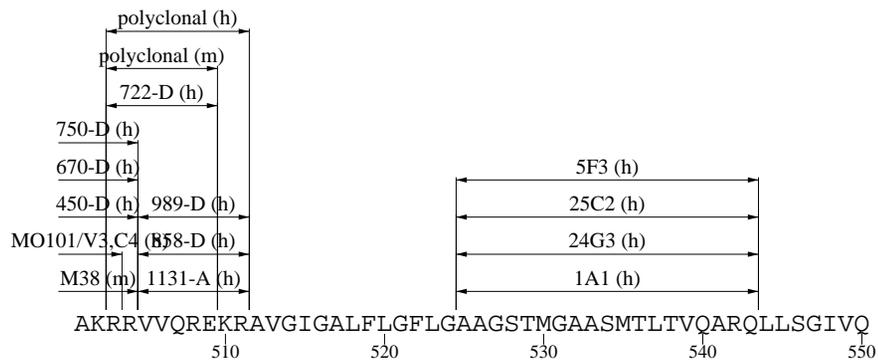
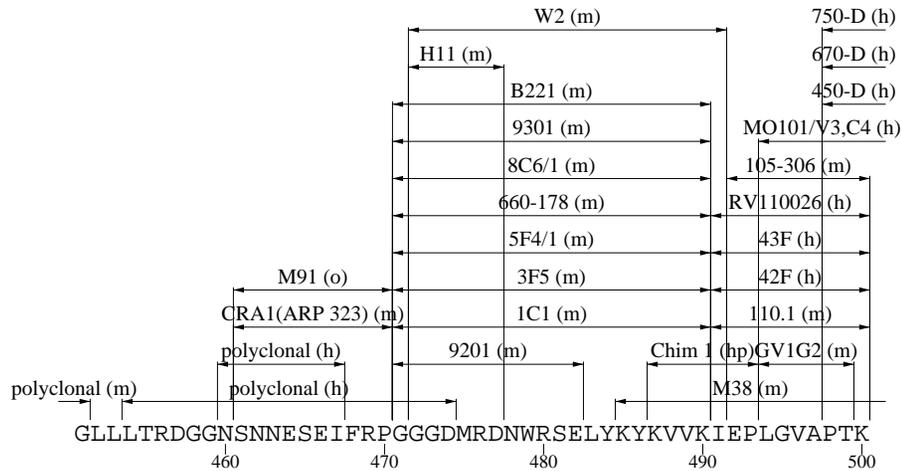


Antibody Protein Maps



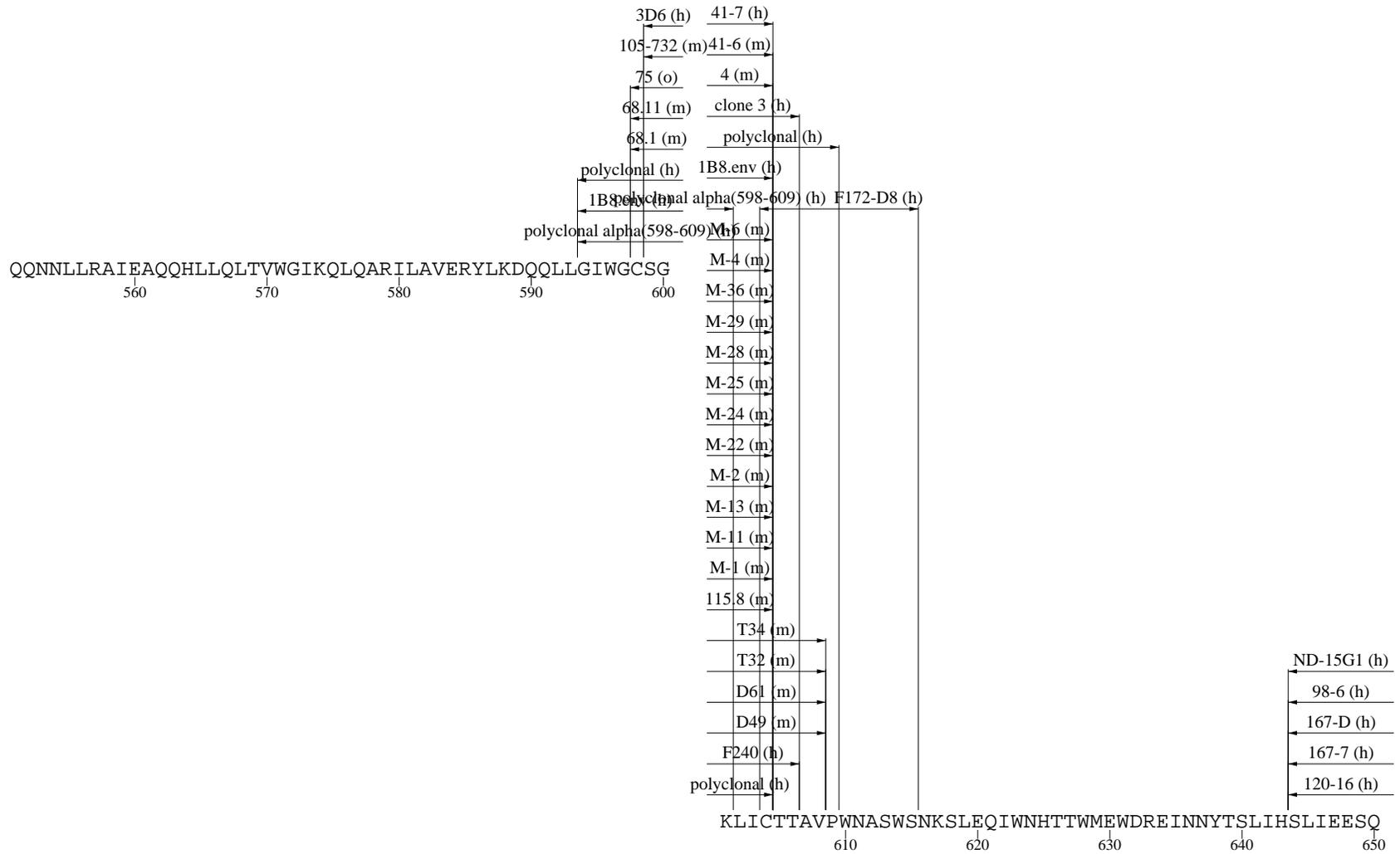
B Cell

Antibody Protein Maps

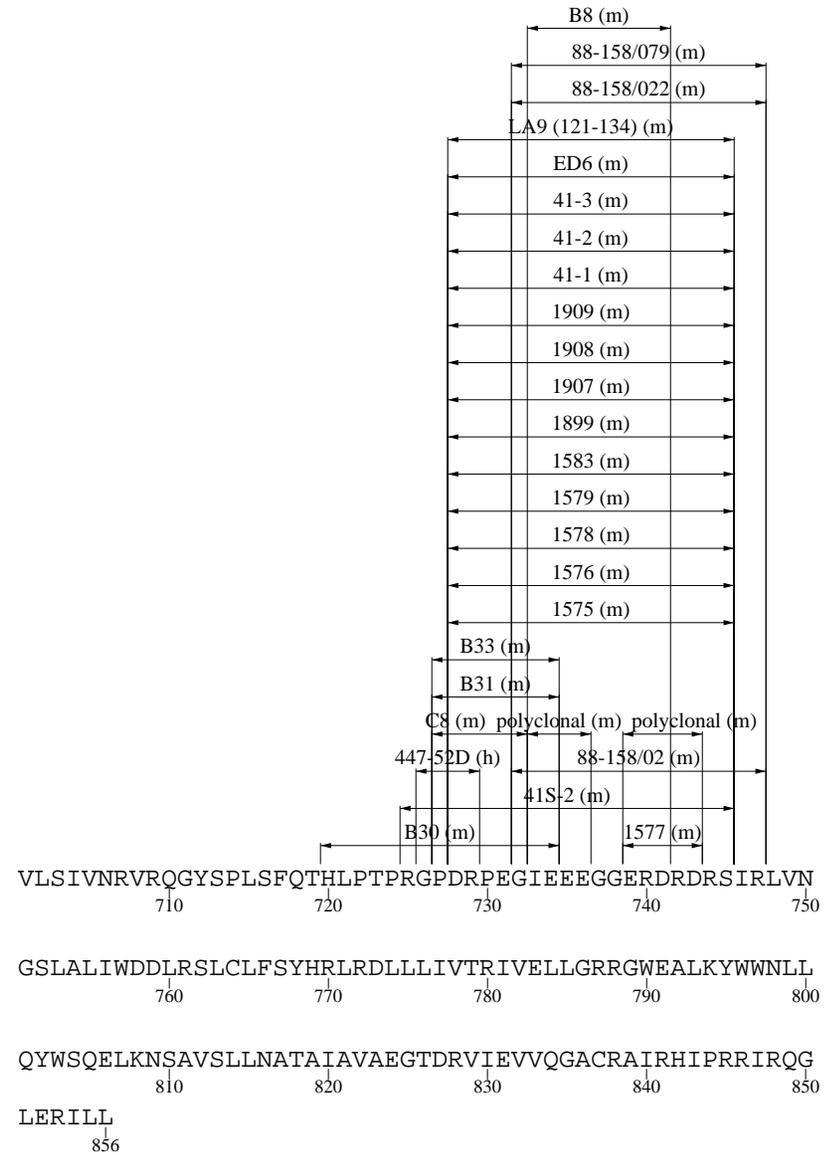
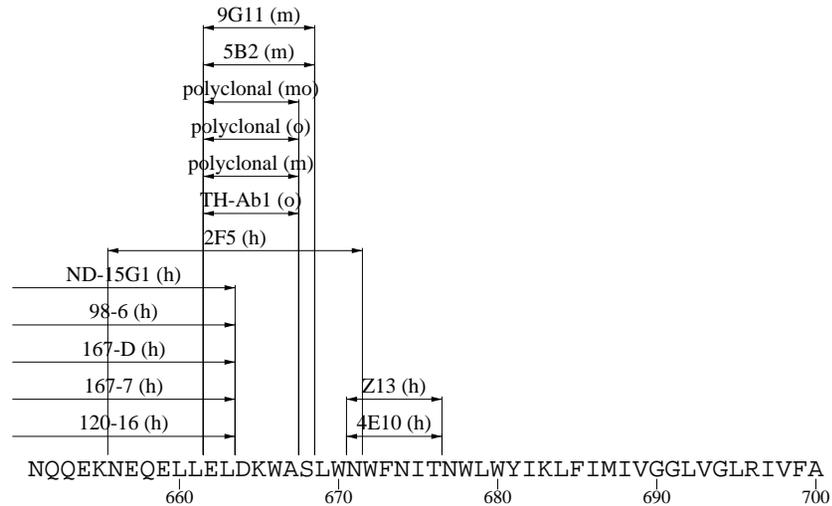
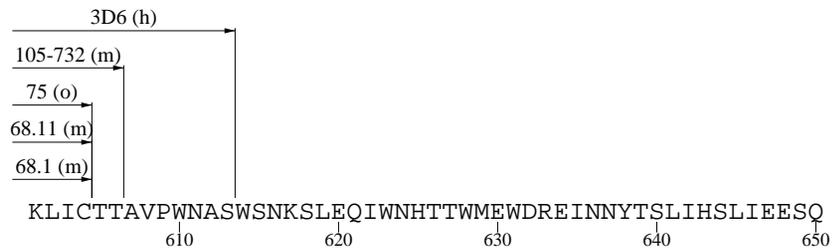


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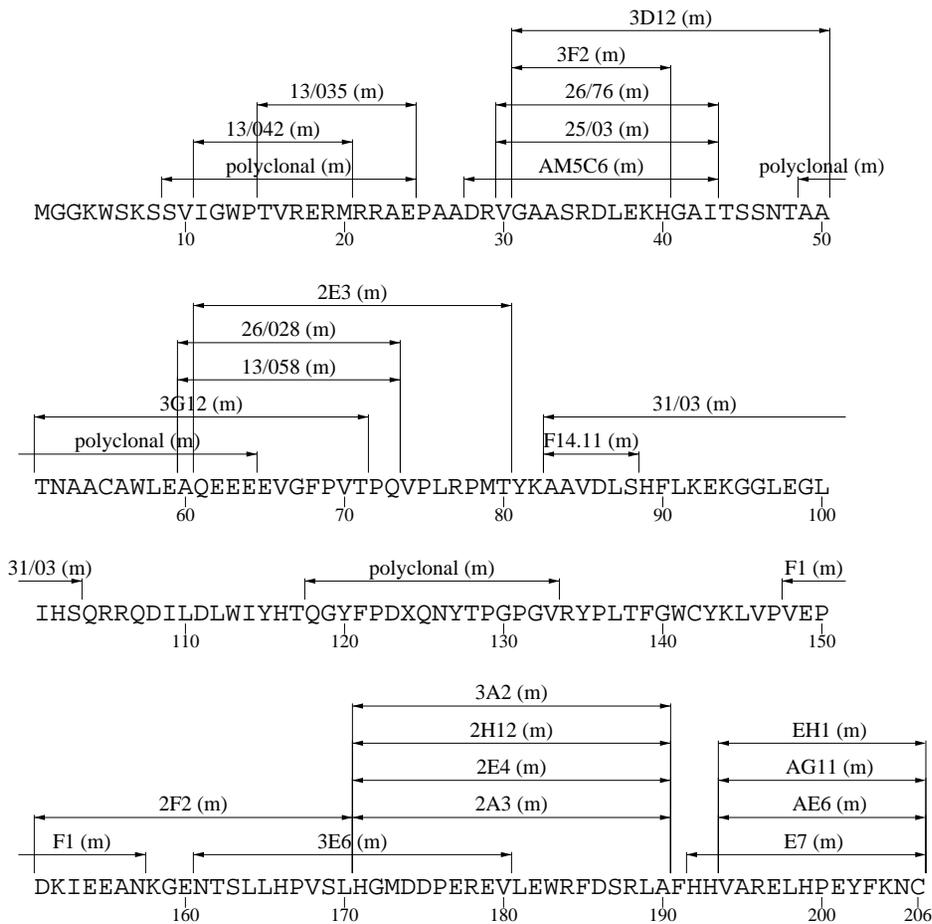
Antibody Protein Maps



B Cell



Nef Antibody Map



Part IV-D: Antibody References

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- ii) recombinant gp120 lacking the V1, V2, V3 loops; iii) a panel of 20 mer peptides; iv) a panel of gp120 mutants; and v) oligomeric versus monomeric gp120. The binding ratio of native versus denatured monomeric gp120 is included in the table in this database. These numbers should be considered with the following points in mind: a continuous epitope may be partially exposed on the surface; and a preparation of rgp120 is not homogeneous and contains fully folded, partly denatured, and some completely unfolded species, so the conformation of what is considered to be a native protein will not only reflect fully folded gp120. The authors suggest that a fivefold increase in the affinity for a MAb binding to denatured versus native gp120 indicates that the epitope is inaccessible in the native form. We also have included here information extracted from Moore et al's list of the gp120 mutations that reduced the binding of a particular MAb. In mapping of exposed regions of gp120, C2, C3, and C5 domain epitopes were found to bind preferentially to denatured gp120. V1, V2 and V3, part of C4, and the extreme carboxy terminus of C5 were exposed on the native monomer. In the oligomeric form of the molecule, only V2, V3 and part of C4 are well exposed as continuous epitopes.
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