

PQVPLRPMTYKAAVDLSHFL

QUERY

PQVPLRPMTYKAAVDLSHFL

CONSENSUS_A -----g-f-----
 A.FR.HIV232956 -----F-G-L-----
 A.FR.HIV232957 -----F-G-F--F--
 A.FR.HIV232959 -----F-G-F--F--
 A.KE.Q23-CXC-CG -----G-----
 A.SE.SE6594 -----
 A.SE.SE7253 -H-----G-L-----
 A.SE.SE7535 -----G-L-----
 A.SE.SE8131 -----G-L-----
 A.SE.SE8538 -----G-F-----
 A.SE.SE8891 -----G-----
 A.UG.92UG037 -----F--GF--
 A.UG.U455 -----F--F--

 CONSENSUS_B -----
 B.-.E90NEF -----G-----
 B.-.HIV232997 -----L-----
 B.-.HIV233002 -----L-----
 B.-.HIV233009 -----G-L-----
 B.-.HIV233016 -K-----V--M--
 B.-.HIV233020 -----
 B.-.HIV233023 --I-----
 B.-.HIV233029 -----G-L-----
 B.-.HIV233030 -----G-----
 B.-.HIV233032 -----G-----
 B.-.HIV233037 -----G-L-----
 B.-.HIV233038 -----
 B.-.HIV233043 -----G-----
 B.-.HIV233045 -----G-L-----
 B.-.HIV233046 -----G-L-----
 B.AU.1062-1-NEF -----
 B.AU.93JW-3 -----P--
 B.AU.93LW-3 -----
 B.AU.AF064660 -----G-F--N--
 B.AU.AF064667 -----FR-----
 B.AU.AF064676 -----L-I-----
 B.AU.MBC200 -----
 B.AU.MBC925 -----
 B.CN.AF033570 -----G-L-----
 B.CN.AF033572 -----G-L--N--
 B.CN.PRC8 -----F--L-----
 B.CN.RL42 -----G-L-----
 B.DE.D31 -----
 B.DE.HAN -----G-L-----
 B.DE.HEI28CS -----x-G-x-----
 B.DE.HEI3BL -----G-L-----
 B.DE.HEI4BL -----
 B.DE.HIVU52491 -----G-L-----
 B.DE.NEFCC -----SR--R-----
 B.DE.NEFCCG ---x-----G-L-----
 B.DE.NH53 -----
 B.ES.89SP061 -----G-L-----
 B.ES.AF082355 -----
 B.ES.AF082357 -----G-L-----

B.ES.AF082358 -----G-L-----
 B.ES.AF082359 -----F-----
 B.ES.AF082363 -----M-I-----
 B.ES.AF082364 -----
 B.ES.AF082366 -----G-L-----
 B.ES.AF082368 -----G-L-----
 B.ES.AF082370 -----G-----
 B.ES.AF082375 -----F--F--
 B.ES.AF082376 -----G-----
 B.ES.AF082377 -----
 B.ES.AF082378 -----
 B.ES.AF082380 -----
 B.ES.AF082383 -----G-F-----
 B.ES.AF082386 -----
 B.FR.HIV232961 -----
 B.FR.HIV232962 -----G-----
 B.FR.HIV232963 -----F-G-L-----
 B.FR.HIV232964 -----S--L-----
 B.FR.HIV232965 -----
 B.FR.HXB2 -----
 B.FR.NE100 -----RR--I-----
 B.FR.SWB884 -----RR--I-----
 B.GA.OYI -----G-L-----
 B.GB.001GH-93(1) -----G-M-----
 B.GB.002EM-93(1) -----G-LN-----
 B.GB.003PW-93(1) -----I--L-----
 B.GB.005PF1-93(1) -----
 B.GB.006DC-93(1) -----G-F-----
 B.GB.010JW-93(1) -----G-L-----
 B.GB.011JR-93(4) -----G-L-----
 B.GB.012WM-93(1) -----F-----
 B.GB.013PP-94(2) --I--Q--L-----
 B.GB.016GB-93(1) -----V--R--R-F--
 B.GB.023PA-93(1) L-----F-----
 B.GB.025JN-93(1) -----G-F--Y--
 B.GB.027SL-93(1) -----
 B.GB.028JH-94(1) -----G-L-----
 B.GB.030JG-93(1) -----L-----
 B.GB.031DA-93(1) ---V---G-L-----
 B.GB.032AN-93(1) -----R--L-----
 B.GB.037BS-94(2) -----R-----
 B.GB.039NM-94(1) --I-----G-L-----
 B.GB.044C1-94(2) -----G-----
 B.GB.046JM-94(1) -----L-----
 B.GB.048AD-94(1) -----G-L-----
 B.GB.056RP-94B(1) -----I--G-F-----
 B.GB.057DR-94(1) -----G-----
 B.GB.065RK-94(1) -----
 B.GB.067MM-94(2) -----G-F--Y--
 B.GB.068JB-94(1) L-----
 B.GB.098MS-94(1) -----
 B.GB.103CD-94(1) -----G-L-----
 B.GB.104RT-94(1) -----G-----
 B.GB.105AS-94(1) -----G-L--T--
 B.GB.112CR-94(2) -----I--G-L-----
 B.GB.117CH-94(2) -----
 B.GB.122PS-95(1) -----G-L-----
 B.GB.124PD-95(1) -----

B.GB.127RG-96(1) -----D--G-----
 B.GB.130WDC-95(1) -----L-----
 B.GB.131MVS-95(1) -----I-----
 B.GB.143PL-95(1) -H-----L-----
 B.GB.151DH-95(1) -----
 B.GB.157GT-95(1) -----N--G-----
 B.GB.160KO-95(1) -----
 B.GB.161KC-95(1) -----G-L-----
 B.GB.162BB-95(1) -----
 B.GB.163NG-95(1) -----I--R--L-----
 B.GB.164SZ-95(1) -----G-I-----
 B.GB.165DH-95(1) -----G-L-----
 B.GB.166PW-95(1) -----G-L-----
 B.GB.167RW-95(1) -----F-G-L-----
 B.GB.168MB-95(1) -----G-----
 B.GB.CAM1 -----L-I-----
 B.GB.GLNEF1 -----
 B.GB.MANC -----F-G-L-----
 B.GB.NEF2 -----V-----
 B.GB.NEF3 -----
 B.GB.NEF5 -----
 B.IN.HIVP35A -----G-L-----
 B.IT.AF011471 --E-----
 B.IT.AF011474 -----G-F-----
 B.IT.AF011477 -----RG-L-----
 B.IT.AF011478 -----RGxL-----
 B.IT.AF011480 -----x-----
 B.IT.AF011482 T-----L-----
 B.IT.AF011483 -----G-L-----
 B.IT.AF011486 -----Q--L-----
 B.IT.AF011488 x-----R--R-----
 B.IT.AF011492 -----G-L-----
 B.IT.AF047080 -----S-----
 B.IT.AF047081 -----G-----
 B.IT.B.IT-L1 -----x--L-----
 B.IT.B.IT-L2 -----
 B.IT.B.IT-L3 -----HR--I-----
 B.IT.B.IT-L4 -----M-----
 B.IT.B.IT-L5 ---x-----S---x--
 B.IT.B.IT-R1 -----L-----
 B.IT.B.IT-R2 -----G-L-----
 B.IT.B.IT-R3 -----Q--xN-----
 B.IT.B.IT-R4 -----G-----
 B.IT.B.IT-R5 -----x-CR--I-----
 B.KR.AF063915 -----G-L-----
 B.KR.AF063916 -----
 B.KR.AF063919 -----G-F-----
 B.KR.AF063921 -----I-----
 B.KR.AF063926 -----G-L-----
 B.KR.AF063927 -----
 B.KR.AF063931 -----L-----
 B.KR.HIV298019 -----
 B.KR.HIV298022 -----G-----
 B.KR.HIV298024 -----G-L-----
 B.KR.HIV298025 -----
 B.KR.HIV298027 -----G-L-----
 B.KR.HIV298029 -----G-L-----
 B.KR.HIV298030 -----S-----

B.KR.HIVZ98032	-----G-S-----	B.US.NEF179C	-----G-L-----		
B.KR.HIVZ98034	-----D--SS-----	B.US.NEF226B	-----V-----	CONSENSUS_F	-----
B.NL.3202A21	-----G-L-----	B.US.P102A13	-----	F.CM.HIV232985	-----
B.NL.NEFA	-----L-----	B.US.P233A17	-----G-L-----	F.CM.HIV232986	-----L-----
B.NL.NEFD	-----G-L-----	B.US.P248A01	-----	F.FR.HIV232987	-----F-----
B.NL.NEFE	-----F-----	B.US.P357A01	-----G-L-----		
B.SE.AF047082	-----L-----	B.US.P896	-----	CONSENSUS_F1	-----?-----
B.SE.AF047083	-----G-----	B.US.PC-93(1)	-----G-----	F1.BE.VI850	-----V-----
B.SE.AF047085	-----F-----	B.US.PRISO(1)	-H-----	F1.BR.93BR020.1	-----G-----
B.TH.28-19	-----G-L-----	B.US.RF	-----F-----	F1.FI.FIN9363	-----G-F---Q-x
B.TH.AF082838	-----G-L-----	B.US.RP12	-----	F1.FR.MP411	-----F-----
B.TH.AF082839	-----	B.US.RR1	-----		
B.TH.AF082841	-----F-----	B.US.SC	-----	CONSENSUS_F2	-----?-----
B.TW.LM49	-----D-G-I-----	B.US.SF2	-----L-I-----	F2.CM.MP255	-----
B.US.HIV1U03375	-----G-L-----	B.US.U16917	-----S---I---	F2.CM.MP257	-----L-----
B.US.005PF-96(1)	-----	B.US.WEAU160	-----H---#---		
B.US.AD-93(1)	-----G-L-----	B.US.WR27	-----	CONSENSUS_G	-----f---F--
B.US.AD8	-----	B.US.YU2	-----H---M-----	G.BE.DRCBL	-----F---F--
B.US.BC	-----I-----I---			G.FI.HH8793	-----V-----F---F--
B.US.BIB	-----G-R---W---	CONSENSUS_C	-----g-f---f---	G.ML.HIV232990	-----L---F--
B.US.BJ-93(1)	-----	C.BR.92BR025	-----V---F---	G.NG.92NG083	-----F---F--
B.US.BO1	-----	C.BW.96BW01B21	-----G-F---GF--	G.NG.HIV232991	-----L---G-F---F--
B.US.BRVA	-----	C.BW.96BW0402	-----F---F---	G.NG.HIV232992	-----G-F---F--
B.US.BT-94(1)	-R-----	C.BW.96BW0502	-----G-F---GF--	G.SE.SE6165	-----F-G-F---F--
B.US.CD1	-----	C.BW.96BW1104	-----FG---F---		
B.US.D8511	-----G-L-----	C.BW.96BW1210	-----G-F---F---	CONSENSUS_H	-----g-f-----
B.US.DH1	-----G-L-----	C.BW.96BW15B03	-----G---F---	H.BE.VI991	-----G-F-----
B.US.DH123	--I-----L-----	C.BW.96BW16B01	-----E-F---F---	H.BE.VI997	-----L-----
B.US.DJ-93(1)	-----	C.BW.96BW17A09	-----F---F---	H.CD.HIV232994	-----E-F-F-F---
B.US.E1	-----G-L-----	C.ET.ETH2220	-----F---L---	H.CD.HIV232995	-----V---G-L-F---
B.US.E81NEF	-----G-----	C.FR.HIV232966	-----F-G-F---F---	H.CF.90CF056	-----G-F-----
B.US.E88NEF	-----G-----	C.FR.HIV232967	-----F-G-F---GF--		
B.US.EP-94(1)	-----W--L-----	C.FR.HIV232968	-----S---F---F---	CONSENSUS_J	--?-----G-?---F--
B.US.FA-93(1)	-----G-----	C.FR.HIV232969	-----S-S-F---F---	J.SE.SE9173	--x-----G-F---F--
B.US.HIV1U16893	-----L-----	C.FR.HIV232970	-----S---F---F---	J.SE.SE9280	--I-----G---F--
B.US.HIV1U24455	-----G-----	C.FR.HIV232971	-----F---GF---		
B.US.HIV1U26074	-----	C.FR.HIV232972	-----F-FGF---	CONSENSUS_K	-----?--?--F--GF--
B.US.HIV1U26098	-----	C.FR.HIV232973	-----W---	K.CD.EQTB11C	-----F-G-F---GF--
B.US.HIV1U26112	-----G-L-----	C.FR.HIV232976	-----F---F---	K.CM.MP535	-----F---F---GF--
B.US.HIV1U26119	-----	C.FR.HIV232977	-----W---	N.CM.YBF30	-----I---Q-F---F--
B.US.HIV1U26141	-----	C.FR.HIV232978	-----F---F---		
B.US.HIVU44444	-----I-----	C.FR.HIV232979	-----G-F---F---	CONSENSUS_O	-----?--?--F---F--
B.US.HIVU44450	-----G-L-----	C.FR.HIV232980	-----F---	O.CM.ANT70C	-----G-F---F--
B.US.HIVU44456	-----G-----	C.FR.HIV232986	-----G-F---F---	O.CM.MVP5180	-----F---F---F--
B.US.HIVU44465	-----G-L-----	C.IN.21068	-----F-G-L---F---	CRF01_AE.CF.90CF402	-----G-F---F--
B.US.HIVU44468	-----	C.IN.301904	-----F-E---F---	CRF01_AE.FR.232982	-----G-F---F--
B.US.HP87B1	-----G-L-----	C.IN.301999	-----F-G-F---F---	CRF01_AE.FR.232983	-----G-F---F--
B.US.HS-93(1)	-----L-----	C.IN.94IN11246	-----F-G-F---F---	CRF01_AE.FR.232984	-----F-E-F---F--
B.US.JRCSF	-----I-----	C.IN.HIVY15117	-----G-F---F---	CRF01_AE.TH.1-2	-----F-E-F---F--
B.US.JRFL	-----G-----	C.IN.HIVY17884	-----F-G-F---F---	CRF01_AE.TH.1-3	-----F-E-F---F--
B.US.LM1	-----G-L-----	C.IN.HIVY17891	-----F-G-F---F---	CRF01_AE.TH.11-25	-H-----F-G-F---F--
B.US.LT-87-1(1)	-----G-L-----	C.IN.HIVY17892	-----F-G-F---F---	CRF01_AE.TH.11-31	-----F-G-F---F--
B.US.MB-94(1)	-----V--G-----			CRF01_AE.TH.122-21	-----G-F---F--
B.US.MNCG	-----L-----	CONSENSUS_D	-----e-----	CRF01_AE.TH.18-47	-----F---F--
B.US.NC7	-----GI-----	D.CD.84ZR085	-----	CRF01_AE.TH.235-3	-----G-F---F--
B.US.NEF	-----	D.CD.ELI	-----E-L-----	CRF01_AE.TH.235-32	-----G-F---F--
B.US.NEF164B	-----I-M---	D.CD.NDK	-----E-----	CRF01_AE.TH.24-54	-----G-F---F--
B.US.NEF166E	-----G-L-----	D.UG.94UG1141	-----E-----	CRF01_AE.TH.240-12	-----G-F-F-F--

CRF01_AE.TH.26-3	-----G-F--F--
CRF01_AE.TH.35-6	-----G-F--F--
CRF01_AE.TH.6-9	-----G-F--F--
CRF01_AE.TH.73-44	-----F-G-F--F--
CRF01_AE.TH.74-26	-----F--F--F--
CRF01_AE.TH.89-30	-----F-G-F--F--
CRF01_AE.TH.9-3	-----G-F--F--
CRF01_AE.TH.93TH253	-----G-F--F--
CRF01_AE.TH.98-4	-----G-F--F--
CRF01_AE.TH.CM240	-----G-F--F--
CRF01_AE.TH.TH022	-----G-F--F--
CRF01_AE.TH.TH047	-----F-E-F--F--
CRF02_AG.FR.DJ263	-----F--GF--
CRF02_AG.FR.DJ264	-----G-F--GF--
CRF02_AG.NG.IBNG	-----G--
CRF03_AB.RU.KAL1532	-----G-F--
CRF04_cpx.CY.94CY03	-----F-G-L--
CRF04_cpx.GR.97PVCH	-----F--L--
CRF04_cpx.GR.97PVMY	-----
AC.IN.21301	-----G-L--F--
AC.RW.92RW009	-----F--
AC.SE.SE9488	-----G-L--
AC.ZM.ZAM184	-----G--
ACD.SE.SE8603	-----F--
AD.SE.SE6954	-----G--
AD.SE.SE7108	-----
ADHU.NO.NOGIL3	-----
ADU.CD.MAL	-----G-F--
AF.GA.HIV232981	-----G-F--
AG.NG.G3	Q-----F--F--
AG.SE.SE7812	-----
AGHU.GA.VI354	--L-----F-G-F--GF--
AGJ.AU.BFP90	---V-----F--F--
AGJ.ML.95ML84	-----F-G-F--F--
AGU.CD.Z321	-----F-G-F--F--
BF.BR.93BR029.4	-----G-L--
DF.BE.VI961	-----F-G-L--
GH.GA.HIV232993	-----G-F--GF--
GU.FR.HIV232974	-----G-F--
U.CD.VI1126	-----G-F--I
U.CM.HIV232988	-----F--GF--
U.FR.HIV232958	-----G-F--GF--
U.FR.HIV232960	-----G-F--GF--
CONSENSUS_CPZ	----?------?-F--??--
CPZ.GA.CPZGAB	----T-----F--
CPZ.US.CPZUS	-----Q-F--GF--

SPAIFQSSMTKILEPFRKQ

QUERY **SPAIFQSSMTKILEPFRKQ**
 CONSENSUS_A -----sk-
 A.KE.Q23-CXC-CG -----SK-
 A.SE.SE6594 -----SK-
 A.SE.SE7253 -----LK-
 A.SE.SE7535 -----ER-
 A.SE.SE8131 -----SK-
 A.SE.SE8538 --S-----SK-
 A.SE.SE8891 -----I-----V--
 A.UG.92UG037 -----A-----SK-
 A.UG.U455 --S-----S-H

 CONSENSUS_B -----
 B.-.NL43E9 -----C-----
 B.AU.MBC18 -----R-----R--
 B.AU.MBC200 -----
 B.AU.MBC925 -----C-----
 B.AU.MBCC54 -----
 B.AU.MBCC98 -----Y-----
 B.AU.MBCD36 -----
 B.CN.RL42 -----C-----
 B.DE.D31 -----
 B.DE.HAN -----
 B.FR.HXB2 -----
 B.GA.OYI -----
 B.GB.CAM1 -----
 B.GB.MANC -----
 B.NL.3202A21 -----C-----
 B.TW.LM49 -----R-----
 B.US.AD8 -----
 B.US.BC -----
 B.US.DH123 -----
 B.US.JRCSF -----
 B.US.JRFL -----
 B.US.MNCG -----
 B.US.NY5CG -----C-----
 B.US.P896 -----
 B.US.RF -----K---
 B.US.SF2 -----
 B.US.WEAU160 -----
 B.US.WR27 --T--P---Q-----P-
 B.US.YU2 -----T-----

 CONSENSUS_C -----a--
 C.BR.92BR025 --S----T-----A--
 C.BW.96BW01B03 -----AL-
 C.BW.96BW0402 -----I-----TK-
 C.BW.96BW0502 -----L--
 C.BW.96BW1104 --S-----AK-
 C.BW.96BW1210 -----A--
 C.BW.96BW15B03 --S-----AR-
 C.BW.96BW1626 -----A--
 C.BW.96BW17A09 -----A--
 C.ET.ETH2220 --P-----PQ-----AP-
 C.IN.21068 -----N--R-----A--

C.IN.301904 -----R-----AR-
 C.IN.301905 -----C--R-----A--
 C.IN.301999 -----A-----A--
 C.IN.94IN11246 ----------GR-

 CONSENSUS_D -----
 D.CD.84ZR085 -----I-----
 D.CD.ELI -----
 D.CD.NDK -----
 D.CD.Z2Z6 -----
 D.UG.94UG1141 -----

 CONSENSUS_F1 -----c-----ak-
 F1.BE.VI850 -----C-----MK-
 F1.BR.93BR020.1 -----Y-----D--AK-
 F1.FI.FIN9363 -----C-----TR-
 F1.FR.MP411 ----------AK-

 CONSENSUS_F2 -----?--?-----??-
 F2.CM.MP255 -----C-----AK-
 F2.CM.MP257 -----I-----E-

 CONSENSUS_G -----tk-
 G.BE.DRCBL -----T--
 G.FI.HH8793 -----IK-
 G.NG.92NG083 -----S-TK-
 G.SE.SE6165 -----R-----AN-

 CONSENSUS_H -----
 H.BE.VI991 -----
 H.BE.VI997 -----
 H.CF.90CF056 -----A--E--

 CONSENSUS_J -----C-----K---ER-
 J.SE.SE9173 -----C-----K---ER-
 J.SE.SE9280 -----C-----K---ER-

 CONSENSUS_K -----?-----?K-
 K.CD.EQTB11C -----C-----RK-
 K.CM.MP535 -----H-----IK-
 N.CM.YBF30 -----T-----EKH

AGJ.AU.BFP90 -----I-----IK-
 AGJ.ML.95ML8 -----I-----TK-
 AGU.CD.Z321 -----TK-
 BF.BR.93BR029.4 -----
 CRF01_AE.CF.90CF40 -----AR-
 CRF01_AE.TH.93TH25 -----IK-
 CRF01_AE.TH.CM240 -----IK-
 CRF01_AE.TH.TH022 -----C-T-----TK-
 CRF01_AE.TH.TH047 -----IK-
 CRF02_AG.FR.DJ263 -----A--N---HY-IK-
 CRF02_AG.FR.DJ264 -----A-----IK-
 CRF02_AG.NG.IBNG -----A-----TK-
 CRF03_AB.RU.KAL153 -----
 CRF04_CPX.CY.94CY0 -----C-----FK-
 CRF04_CPX.GR.97PVC -----Y-----TR-
 CRF04_CPX.GR.97PVM -----C-----TK-
 DF.CD.VI961 -----C-----
 U.CD.VI1126 -----Y-----TK-

 CONSENSUS_CPZ -----?----?k?
 CPZ.CD.CPZANT -----A-----A---DKY
 CPZ.GA.CPZGAB --S-----EK-
 CPZ.US.CPZUS -----D-----H

A*0206 X[V]XXXXXX[V]
A*0206 X[V]XXXXXX[V]
A*0206 X[V]XXXXXXXX[V]
A*0207 X[L][D]XXXXX[L]
A*0207 X[L][D]XXXXX[L]
A*0207 X[L][D]XXXXXX[L]
A*0214 X[VQL]XXXXXX[LV]
A*0214 X[VQL]XXXXXX[LV]
A*0214 X[VQL]XXXXXXXX[LV]
A3 X[LVM]XXXXXX[KYF]
A3 X[LVM]XXXXXX[KYF]
A3 X[LVM]XXXXXXXX[KYF]
B*5101 X[APG]XXXXXX[FI]
B*5101 X[APG]XXXXXX[FI]
B*5101 X[APG]XXXXXXXX[FI]
B*5102 X[PAG]XXXXXX[IV]
B*5102 X[PAG]XXXXXX[IV]
B*5102 X[PAG]XXXXXXXX[IV]
B*5103 X[APG]XXXXXX[VIF]
B*5103 X[APG]XXXXXX[VIF]
B*5103 X[APG]XXXXXXXX[VIF]

A3 X[LVM]XXXXXXXX[KYF]
B*5101 X[APG]XXXXXXXX[FI]
B*5101 X[APG]XXXXXX[FI]
B*5101 X[APG]XXXXXXXX[FI]
B*5102 X[PAG]XXXXXXXX[IV]
B*5102 X[PAG]XXXXXX[IV]
B*5102 X[PAG]XXXXXXXX[IV]
B*5103 X[APG]XXXXXXXX[VIF]
B*5103 X[APG]XXXXXX[VIF]
B*5103 X[APG]XXXXXXXX[VIF]

This table lists epitopes that are experimentally observed to be presented by a HLA type carried by the patient, but the defined epitope has substitutions relative to the peptides from your reference strains and so might be missed by your reagents: in HXB2 for Gag, Pol; MN for Env; BRU for Nef, relative to most B clade Sequences in the database:

Protein	Epitope in Database	Epitope in Ref. strain	Epitope in Consensus B	HLA	Notes
p17(77–85)	SLFNTVATL	SLYNTVATL	SLYNTVATL	A*0201	
RT(179–187)	VIYQYMMDL	VIYQYMDDL	VIYQYMDDL	A2	
RT(179–187)	VIYQYMMDL	VIYQYMDDL	VIYQYMDDL	A2, A*0202	
RT(308–317)	EILKEPVGHV	EILKEPVHGV	EILKEPVHGV	A*0201	
gp160(78–86)	DPNPQEVVL	DPNPQEVEL	DPNPQEVVL	B35, B51	
gp160(121–129)	KLTPLCVSL	KLTPLCVTL	KLTPLCVTL	A2	
gp160(156–165)	NCSFNISTSI	NCSFNITTSI	NCSFNITTSI	Cw*08	
gp160(156–165)	NCSFNISTSI	NCSFNITTSI	NCSFNITTSI	Cw8	
gp160(192–200)	KLTSNTSV	RLISNTSV	RLISNTSV	A2	
gp160(192–200)	TLTSNTSV	RLISNTSV	RLISNTSV	A2	
gp160(192–200)	TLTSNTSV	RLISNTSV	RLISNTSV	A2.1	
gp160(239–247)	CTNVSTVQC	CKNVSTVQC	CTNVSTVQC	Cw8	
gp160(311–320)	RGPGRAFVTI	IGPGRAFYTT	IGPGRAFYTT	A*0201	
gp160(311–320)	RGPGRAFVTI	IGPGRAFYTT	IGPGRAFYTT	A2	
gp160(311–320)	MGPKRAFYAT	IGPGRAFYTT	IGPGRAFYTT	A2	
gp160(369–375)	PEIVTHS	PEIVMHS	PEIVMHS	A2	
gp160(377–387)	NSGGEFFYSNS	NCGGEFFYCNT	NCGGEFFYCNT	A2	
gp160(416–424)	LPCRKQII	LQCKIKQII	LPCRKQII	B*5101	
gp160(557–565)	RAIEAQQHL	RAIEAQQHM	RAIEAQQHL	B*5101	
gp160(557–565)	RAIEAQQHL	RAIEAQQHM	RAIEAQQHL	B51	
gp160(700–708)	AVLSVVNRV	AVLSIVNRV	AVLSIVNRV	A2	
gp160(747–755)	RLVNGSLAL	RLVHGFLAI	RLVDGFLAL	A2	
gp160(770–778)	RLRDLLIV	HHRDLLIA	RLRDLLIV	A*0201	
gp160(770–780)	RLRDLLIVTR	HHRDLLIAAR	RLRDLLIVTR	A*0301	
gp160(770–780)	RLRDLLIVTR	HHRDLLIAAR	RLRDLLIVTR	A3	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A*0201	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2.1	
gp160(814–822)	LLNATDIAV	LLNATAIAV	LLNATAIAV	A2	
gp160(835–843)	RAYRAILHI	RAGRAILHI	RACRAILHI	B*5101	
Nef(136–145)	PLTFGWCFKL	PLTFGWYKL	PLTFGWCFKL	A2	
Nef(190–198)	AFHHVAREK	AFHHVAREL	AFHHMAREL	A3	

Table 1: **p17**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85 Clade A)	SLFNTVATL	HIV-1 infection	human(A*0201)	[Dorrell (1999)]
		<ul style="list-style-type: none"> • Epitope SL9: CTL responses in three individuals with non-clade B infections were studied, 2 with subtype A infections, 1 with subtype C – their infections all originated in East Africa • This epitope is most commonly SLYNTVATL in B subtype, and CTL from the C subtype infection did not recognize B clade gag or the 3Y form of the epitope, but do recognize the predominant A and C clade form, SLFNTVATL 			

Table 2: **RT**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(179–187)	RT()	VIYQYMMDL	HIV-1 exposure	human(A2)	[Rowland-Jones (1998a)]
		<ul style="list-style-type: none"> • A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-defined B clade epitopes that tended to be conserved in A and D clades – such cross-reactivity could protect against both A and D and confer protection in Nairobi where both subtypes are circulating • The A and D consensus sequences are both VIYQYMMDL 			
RT(179–187)	Pol()	VIYQYMMDL	HIV-1 exposure	human(A2, A*0202)	[Rowland-Jones (1998b)]
		<ul style="list-style-type: none"> • HIV-specific CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A, B and D clade viruses 			
RT(308–317)	RT()	EILKEPVGHV	HIV-1 infection	human(A*0201)	[van der Burg (1997), Menendez-Arias (1998)]
		<ul style="list-style-type: none"> • Recognized by CTL from a long-term survivor, SPIETVPVKL was also recognized • Recognized by CTL from a progressor, EELRQHLLRW and TWETWWTEYW were also recognized 			

Table 3: **gp160**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(78–86)	gp120(77–85 SF2) • Binds HLA-B*3501 and B*5101 – binds and kills gp120-vaccinia virus infected cells carrying B35 or B51	DPNPQEVVL	HIV-1 infection	human(B35, B51)	[Shiga (1996)]
gp160(121–129)	gp120(121–129) • This study compares the ability of macrophages and dendritic cells to stimulate primary responses in CD8+ lymphocytes isolated from HLA-appropriate HIV-uninfected donors using peptide-pulsed APC – the dendritic cells performed better as APC for the stimulation of primary responses • Strong CTL responses were elicited by the epitopes DRFYKTLRA and GEIYKRWII when presented by either immature or mature dendritic cells – macrophages were not able to prime a CTL response against DRFYKTLRA • A weak response to KLTPLCVSL was stimulated using macrophages as the APC • No detectable response was observed for the following previously-defined HIV epitopes: KIRLRPGGK, ILKEPVHGV, IRLRPGGK, GPKVKQWPL	KLTPLCVSL	<i>in vitro</i> stimulation	human(A2)	[Zarling (1999)]
gp160(156–165)	gp120(156–165) • Recognized by CTL clone LWF A5, isolated from a lab worker exposed to HIV-1 in 1985 • The processing of this epitope is TAP1/2-dependent, as are most Env epitopes, and it contains two N-linked glycosylation sites that are glycosylated in Env • Only peptide that has been deglycosylated, a process that changes asparagine (N) to aspartic acid (D) was recognized: the aspartic acid at position 5 was critical, position 1 could be either D or N • This peptide also contains a Cys involved in a disulfide linkage but reducing conditions did not effect recognition by CTL clone LWF A5 • The HIV-1 Env epitopes are typically processed by a TAP1/2 dependent mechanism, which involves cotranslational translocation into the ER, glycosylation, export back into the cytosol, and deglycosylation for processing, and retransport into the ER for the association with class I molecules • The particular pathway of generating an epitope may have an impact on the presentation of that epitope, quantitatively as well as qualitatively	NCSFNISTSI	HIV-1 infection	human(Cw*08)	[Ferris (1999)]
gp160(156–165)	gp120(156–165 IIIB) • HIV IIIB proteins were used to define the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIIB • NCSFNITTSI, a variant found in HIV-1 MN, was not recognized, thus this epitope was type-specific • NCSFNISTSI contains two potential N-linked glycosylation sites and cysteine residue, possibly related to the requirement for a high sensitizing dose of peptide for CTL activity	NCSFNISTSI	HIV-1 infection	human(Cw8)	[Sipsas (1997)]
gp160(192–200)	gp120(192–199 HXB2R) • Epitope predicted on HLA binding motif, and studied in the context of inclusion in a synthetic vaccine	KLTSNNTSV	HIV-1 infection	human(A2)	[Brander (1995)]

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(192–200)	gp120(197–205) • Crystallization of HLA-A2 molecules complexed with antigenic peptides – refers to Dadaglio <i>et al</i> 1991	TLTSCNTSV	no CTL shown	human(A2)	[Garboczi (1992)]
gp160(192–200)	gp120(199–207) • This epitope was recognized by PBMC from 6/14 HIV+ asymptomatic patients • This epitope was used along with pol CTL epitope ALQDSGLEV and a tetanus toxin T helper epitope for a synthetic vaccine • This vaccine failed to induce a CTL response, although a helper response was evident	TLTSCNTSV	peptide immunization and HIV-1 infection	human(A2.1)	[Brander (1996)]
gp160(239–247)	gp120(241–249 LAI) • HIV IIIB proteins were used to define the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIIB • CTNVSTVQC contains a potential N-linked glycosylation site and cysteine residues, possibly related to a requirement for a high sensitizing dose of peptide for CTL activity	CTNVSTVQC	HIV-1 infection	human(Cw8)	[Sipsas (1997)]
gp160(311–320)	gp160(318–327 IIIB) • This immunogenic peptide does not have the known binding motif for A2.1 • The same optimal peptide for this human HLA-A2.1 epitope was observed for a murine H-2 D ^d epitope	RGPGRAFVTI	CTL line from HIV-donor	human(A*0201)	[Alexander-Miller (1996)]
gp160(311–320)	gp160(318–327 IIIB) • Individual was immunized with rec vaccinia gp160 IIIB and boosted with purified gp160 • Lysis only occurs with IIIB P18 peptide pulsed onto autologous targets; MN, RF, SIMI P18 peptides fail to stimulate CTL • Restimulating immune cells from gp160 IIIB vaccinees with MN, RF, or SIMI P18 did not enhance the MN, RF, or SIMI specific CTL response	RGPGRAFVTI	vaccinia IIIB gp160	human(A2)	[Achour (1996)]
gp160(311–320)	gp160(318–327 SIMI) • Individual was immunized with rec vaccinia gp160 SIMI and boosted with purified recombinant gp160 SIMI • P18 MN and RF peptides were able to stimulate the HIV-specific CTL that arose in response to the SIMI vaccination, thus the P18 MN peptide (IGPGRAFYT) and the P18 RF peptide (KGPRVIYAT) could cross-react • The P18 IIIB peptide does not cross-react (RGPGRAFVTI in the epitope region) • gp160 SIMI primed immune cells could generate a significantly broader specificity when stimulated with P18 MN or P18RF peptides, but not P18 IIIB	MGPKRAFYAT	vaccinia SIMI gp160	human(A2)	[Achour (1996)]
gp160(369–375)	gp120(374–380 BRU) • Defined through blocking CTL activity, and Env deletions	PEIVTHS	HIV-1 infection	human(A2)	[Dadaglio (1991)]

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(377–387)	gp120(377–387)	NSGGEFFYSNS		human(A2)	[Hickling (1990)]
					<ul style="list-style-type: none"> • Peptides recognized by class I restricted CTL can bind to class II
gp160(416–424)	Env(413–421 SF2)	LPCRRIKQII	HIV-1 infection	human(B*5101)	[Tomiya (1999)]
					<ul style="list-style-type: none"> • HLA-B27, -B51, and -B57 are associated with slow progression to AIDS, while HLA-B35, -B8, -B24 are associated with a rapid progression to AIDS (Nat. Med. 2:405, 1996; Lancet 22:1187, 1986; Hum Immunol 22:73, 1988; Hum Immunol 44:156, 1995) • 15% of Japanese populations carry HLA-B51 while HLA-B27 and -B57 are detected in less than 0.3% • Of the 172 HIV-1 peptides with HLA-B*5101 anchor residues, 33 bound to HLA-B*5101, seven of these peptides were reactive with CTL from 3 B*5101 positive individuals, and six were properly processed • Four of the six epitopes were highly conserved among B subtype sequences, LPCRRIKQII is not conserved
gp160(557–565)	gp41(557–565 IIIB)	RAIEAQQHL	HIV-1 infection	human(B*5101)	[Brander & Goulder(2001)]
					<ul style="list-style-type: none"> • C. Brander notes this is a B*5101 epitope
gp160(557–565)	gp41(557–565 IIIB)	RAIEAQQHL	HIV-1 infection	human(B51)	[Sipsas (1997)]
					<ul style="list-style-type: none"> • HIV IIIB proteins were used to define the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIIB • KAIEAQQHL, a variant found in HIV-1 NY5CG, was also recognized • RAIEAQQHM, a variant found in HIV-1 JRCSF, was also recognized • RAIDAQQHL, a variant found in HIV-1 ETR, was also recognized • RAIKAQQHL, a variant found in HIV-1 CDC42, was also recognized
gp160(700–708)	gp41(705–714)	AVLSVVNRV	HIV-1 infection	human(A2)	[Ferris (1999)]
					<ul style="list-style-type: none"> • This epitope is processed by a TAP1/2 dependent mechanism
gp160(747–755)	gp41(747–755)	RLVNGSLAL	HIV-1 infection	human(A2)	[Parker (1992)]
					<ul style="list-style-type: none"> • Studied in the context of HLA-A2 peptide binding
gp160(770–778)	Env(679–777)	RLRDLLLIV	HIV-1 infection	human(A*0201)	[Kmieciak (1998)]
					<ul style="list-style-type: none"> • CTL responses in six patients to four Env epitopes were studied: D2: LLNATAIAV, 5.3: RLRDLLLIV, D1: KLTPLCVTL, and 4.3: QMHEDIISL – all have A2 anchor residues • The C terminal epitopes (D2 and 5.3) were highly variable and the variability was considered responsible for limited CTL response, while D1 and 4.3, N-terminal epitopes, were much more conserved and gave evidence of high levels of CTL response <i>in vitro</i> • Peptides 5.3 and D2 bound to HLA A*0201 with low affinity and were variable, particularly D2;

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(770–780)	gp41(768–778 NL43) • CD8+ T cell clone	RLRDLLLVTR	HIV-1 infection	human(A*0301)	[Takahashi (1991)]
gp160(770–780)	gp41(768–778 NL43) • The consensus peptide of clade B is RLRDLLLVTR • The consensus peptide of clades A, C and E is RLRDFILIVTR and it is less reactive • The consensus peptide of clade D is SLRDLLLVTR and it is less reactive	RLRDLLLVTR	HIV-1 infection	human(A3)	[Cao (1997)]
gp160(813–822)	gp41(814–823 LAI) • Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823 • Noted to be A*0201 in Brander <i>et al.</i> , 1999 database	SLLNATDIAV	MN rec gp160	human(A*0201)	[Dupuis (1995)]
gp160(813–822)	gp41(814–823) • Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-specific CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • SLLNATDIAV is a conserved HLA-A2 epitope included in this study – 4/6 patients had this sequence as their HIV direct sequence, and 3 of these had a detectable CTL response – the other two had either the sequence SLFNATDIAV or SLLNTTDIVV and no detectable CTL response • CTL demonstrated against peptide-coated target, epitope is naturally processed and enhancible with vaccine	SLLNATDIAV	HIV-1 infection	human(A2)	[Kundu (1998b)]
gp160(813–822)	Env(814–823 Clade B) • Ten HIV-1+ HLA A2 asymptomatic individuals were given two courses of HIV-1 MN rgp160 vaccine over a 2 year period • Two hundred and fifty three HIV-1 peptides of 9 or 10 aa possessing the HLA-A2.1 binding motif (Leu at position 2, Val at the C terminus) were identified in gp160, of which 25 had a high or intermediate binding affinity • Eleven peptides were studied that had high HLA-A2 binding affinity – a CTL response was detected to 9/11 peptides in at least 1 individual • CTL responses after reimmunization may include recall responses – only individuals with vaccine cross-reactive sequences prior to vaccination showed detectable CTL responses • CTL to overlapping peptides in this region gave a positive response in the greatest number of patients • ALTERNATIVE EPITOPES: LLNATDIAV and LLNATDIAVA – CTL were induced by vaccine in those that had the sequence SLLNATAIAVA in their own infection, but not in those with: NLLNTIAIAVA or NLFNTTIAIAVA or SLLNATAITVA	SLLNATDIAV	HIV-1 MN rgp160	human(A2.1)	[Kundu (1998a)]
gp160(814–822)	gp41(815–823 LAI) • Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823	LLNATDIAV	MN rec gp160	human(A2)	[Dupuis (1995)]

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(835–843)	Env(834–842 SF2)	RAYRAILHI	HIV-1 infection	human(B*5101)	[Tomiyama (1999)]
					<ul style="list-style-type: none"> • HLA-B27, -B51, and -B57 are associated with slow progression to AIDS, while HLA-B35, -B8, -B24 are associated with a rapid progression to AIDS (Nat. Med. 2:405, 1996; Lancet 22:1187, 1986; Hum Immunol 22:73, 1988; Hum Immunol 44:156, 1995) • 15% of Japanese populations carry HLA-B51 while HLA-B27 and -B57 are detected in less than 0.3% • Of the 172 HIV-1 peptides with HLA-B*5101 anchor residues, 33 bound to HLA-B*5101, seven of these peptides were reactive with CTL from 3 B*5101 positive individuals, and six were properly processed • This peptide could stimulate CTL from one person, however this CTL clone did not recognize B*5101 positive target cells infected with HIV-1 recombinant vaccinia expressing Env, so it was not confirmed that this peptide was a properly processed epitope

Table 4: **Nef**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(136–145)	Nef(136–145)	PLTFGWCFKL	HIV-1 infection	human(A2)	[Durali (1998)]
					<ul style="list-style-type: none"> • Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • Patient B18 had the greatest breadth and diversity of response, and recognized Gag SLYNTVATL and Nef PLTFGWCFKL
Nef(190–198)	Nef(190–198 LAI)	AFHHVAREK	HIV-1 infection	human(A3)	[Hadida (1995)]
					<ul style="list-style-type: none"> • Naturally occurring L to K anchor substitution abrogates A2 binding, but permits HLA-A3 binding

Table 5: **All Defined Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(72–79)	Nef()	VPLRPMTY	HIV-1 exposed seronegative	human(B35)	[Kaul (2000)]
					<ul style="list-style-type: none"> • 11/16 heavily HIV exposed but persistently seronegative sex-workers in Nairobi had HIV-specific CD8 gamma-IFN responses in the cervix – systemic CD8+ T cell responses tended to be to the same epitopes but at generally lower levels than cervical CD8+ T cell responses • Low risk individuals did not have such CD8+ cells • CD8+ epitopes T cell DTVLEDINL (3 individuals), SLYNVATL (4 individuals), LSPRTLNAW (3 individuals) and YPLTFGWCF (4 individuals) were most commonly recognized by the HIV-resistant women
Nef(72–79)	Nef()	VPLRPMTY	HIV-1 infection	human(B35)	[Wilson (2000)]
					<ul style="list-style-type: none"> • Three individuals with highly focused HIV-specific CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-specific CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-specific T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been defined earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWILGGLNK • The subject with A*0201 had a moderately strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL
Nef(72–91)	Nef(71–90 SF2)	PQVPLRMTYKAAVDLSHFL	HIV-1 infection	human()	[Lieberman (1997a)]
					<ul style="list-style-type: none"> • Of 25 patients, most had CTL specific for more than 1 HIV-1 protein • Eleven subjects had CTL that could recognize vaccinia-expressed LAI Nef • Three of these 11 had CTL response to this peptide • The responding subjects were HLA-A3, A32, B51, B62; HLA-A11, A24, B8, B53
Nef(72–91)	Nef(71–90 SF2)	PQVPLRMTYKAAVDLSHFL	HIV-1 infection	human()	[Lieberman (1997b)]
					<ul style="list-style-type: none"> • CTL expanded <i>ex vivo</i> were later infused into HIV-1 infected patients
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV infection	human()	[Garcia (1997)]
					<ul style="list-style-type: none"> • The anti-Nef CTL line P1 specific for this epitope is able to kill target cells via two mechanisms • First: Ca²⁺-dependent, perforin-dependent Nef-specific lysis • Second: Ca²⁺-independent, CD95-dependent apoptosis that could also kill non-specific targets • Findings indicate that the two mechanisms are not mutually exclusive in human CTL, as they are in mice • CTL mediated CD95-dependent apoptosis may play a role in pathogenesis

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(73-82)	Nef(73-82 NL43) <ul style="list-style-type: none"> • 81 Tyr is critical for binding to A3.1 • C. Brander notes that this is an A*0301 epitope in the 1999 database 	QVPLRPMTYK	HIV-1 infection	human(A*0301)	[Koenig (1990)]
Nef(73-82)	Nef(73-82 LAI) <ul style="list-style-type: none"> • C. Brander notes this is an A*0301 epitope 	QVPLRPMTYK		human(A*0301)	[Brander & Goulder(2001)]
Nef(73-82)	Nef(73-82) <ul style="list-style-type: none"> • Soluble factors in supernatant from both an HIV-specific cloned CTL line and an EBV (Epstein-Barr-virus) CTL line inhibit viral replication, but do not block viral entry in CD4+ T lymphocytes, by a noncytotoxic mechanism 	QVPLRPMTYK	HIV-1 infection	human(A11)	[Le Borgne (2000)]
Nef(73-82)	Nef(73-82 LAI) <ul style="list-style-type: none"> • Development of a retroviral vector (pNeoNef) to generate autologous CTL targets • [Hunziker (1998)] suggests that HLA-A2 does not in fact present this epitope • The initial assignment of HLA-A2 presentation for this epitope was based on a serological HLA typing. Subsequently, the authors revisited the issue with genetic HLA typing and found that HLA-A11 was the correct presenting molecule (Dr. Florence Buseyne, Pers. Comm., 2000) 	QVPLRPMTYK	HIV-1 infection	human(A11)	[Robertson (1993)]
Nef(73-82)	Nef(73-82 LAI) <ul style="list-style-type: none"> • Mutational variation in HIV epitopes in individuals with appropriate HLA types can result in evasion of CTL response • [Goulder (1997a)] is a review of immune escape that summarizes this study 	QVPLRPMTYK	HIV-1 infection	human(A11)	[Couillin (1994), Goulder (1997a)]
Nef(73-82)	Nef(73-82 LAI) <ul style="list-style-type: none"> • Mutations found in this epitope in HLA-A11 positive and negative donors were characterized 	QVPLRPMTYK	HIV-1 infection	human(A11)	[Couillin (1995)]
Nef(73-82)	()	QVPLRPMTYK		(A11)	[Brander & Goulder(2001), Buseyne(1999)]
Nef(73-82)	Nef(73-82 LAI) <ul style="list-style-type: none"> • Mutations in Nef that flank this epitope, Thr71Lys and Ala83Gly, may account for an observed loss of CTL reactivity, with escape due to the introduction of proteasome processing defects 	QVPLRPMTYK	HIV-infection	human(A3)	[Chassin (1999)]

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV-1 infection	human(A3)	[Durali (1998)]
					<ul style="list-style-type: none"> • Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • One of the patients was shown to react to this epitope: QVPLRPMTYK
Nef(73–82)	Nef(73–82 LAI)	QVPLRPMTYK	HIV-1 infection	human(A3)	[Goulder (1997b), Goulder (1997a)]
					<ul style="list-style-type: none"> • Identical twin hemophiliac brothers were both infected with the same batch of factor VIII • Both had a response to this epitope • [Goulder (1997a)] is a review of immune escape that summarizes this study
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV-1 infection	human(A3)	[Lubaki (1997)]
					<ul style="list-style-type: none"> • Eighty two HIV-1-specific CTL clones from 5 long-term non-progressors were isolated and analyzed for breadth of response • A sustained Gag, Env and Nef response was observed, and clones were restricted by multiple HLA epitopes, indicating a polyclonal response • An A3+ subject had a strong response to this epitope, with 10/11 CTL clones being specific for this epitope, isolated at two time points, 1 year apart
Nef(73–82)	Nef(73–82 BRU)	QVPLRPMTYK	HIV-1 infection	human(A3, A11, B35)	[Culmann (1991)]
					<ul style="list-style-type: none"> • Nef CTL clones from HIV+ donors
Nef(73–82)	Nef(73–82 LAI)	QVPLRPMTYK	HIV-1 infection	human(A3.1)	[Koenig (1995)]
					<ul style="list-style-type: none"> • Alanine substitutions L76A, R77A, M79A, T80A significantly decreased immunogenicity of peptide • Nef CTL clones (4N225) were infused into an HIV-1 infected volunteer to evaluate effects of infusion on viral load/patient health • Infusion led to outburst of escape variants which resulted in higher viral load/accelerated disease progression
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV-1 infection	human(A3.1)	[Betts (2000)]
					<ul style="list-style-type: none"> • Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninety five optimally defined peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was A3, and responded to QVPLRPMTYK as well as two other A3.1 epitopes

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV-1 infection	human(B*0301)	[Wilson (2000)]
					<ul style="list-style-type: none"> • Three individuals with highly focused HIV-specific CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-specific CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-specific T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been defined earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWILGGLNK • The subject with A*0201 had a moderately strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVVWV, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL
Nef(73–82)	Nef(73–82 LAI)	QVPLRPMTYK		human(B27)	[Culmann(1998)]
					<ul style="list-style-type: none"> • Optimal epitope mapped by peptide titration
Nef(73–82)	Nef(73–82 LAI)	SVPLRPMTYK	HIV-1 infection	human(B35 or C4)	[Buseyne (1993)]
					<ul style="list-style-type: none"> • Vertical transmission of HIV ranges from 13% to 39% • Primary assays showed cytotoxic activity against at least one HIV protein was detected in 70% of infected children • Epitopes recognized in five children were mapped using synthetic peptides and secondary cultures • Patient EM13, who had a CTL response to three epitopes in Nef, was infected via blood transfusion after birth and went from CDC stage P2A to P2E during the study
Nef(74–81)	Nef(74–82)	VPLRPMTY		human(A3)	[Carreno (1992)]
					<ul style="list-style-type: none"> • Included in HLA-A3 binding peptide competition study
Nef(74–81)	Nef(73–82 LAI)	VPLRPMTY	HIV-1 or HIV-2 infection	human(B*3501)	[Brander & Goulder(2001)]
					<ul style="list-style-type: none"> • C. Brander notes this is a B*3501 epitope
Nef(74–81)	Nef(75–82)	VPLRPMTY	no CTL shown	human(B*3501)	[Smith (1996)]
					<ul style="list-style-type: none"> • Crystal structure of VPLRPMTY-class I B allele HLA-B*3501 complex
Nef(74–81)	Nef(73–82 LAI)	VPLRPMTY	HIV-1 or HIV-2 infection	human(B35)	[McMichael & Walker(1994), Culmann (1991)]
					<ul style="list-style-type: none"> • Review of HIV CTL epitopes – defined by B35 motif found within a larger peptide

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(74–81)	Nef(73–82 LAI)	VPLRPMTY	HIV-1 or -2 infection	human(B35)	[Rowland-Jones (1995)]
					<ul style="list-style-type: none"> • VPLRPMTY also recognized by CTL from HIV-2 seropositives; epitope is conserved
Nef(74–81)	Nef()	VPLRPMTY	HIV-1 exposure	human(B35)	[Rowland-Jones (1998a)]
					<ul style="list-style-type: none"> • A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-defined B clade epitopes that tended to be conserved in A and D clades – such cross-reactivity could protect against both A and D and confer protection in Nairobi where both subtypes are circulating • The A and D subtype consensus are identical to the B clade epitope
Nef(74–81)	Nef(75–82)	VPLRPMTY	none	human(B35)	[Lalvani (1997)]
					<ul style="list-style-type: none"> • A peptide-based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-specific CTLp counts could be obtained via staining with peptide-Class I tetramers • This peptide was one of the B35 presented test peptides used in control experiments showing that the assay gave no activity using lymphocytes from 21 healthy B35 seronegative donors
Nef(74–81)	Nef()	VPLRPMTY	HIV-1 exposure	human(B35)	[Rowland-Jones (1998b)]
					<ul style="list-style-type: none"> • HIV-specific CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A, B, and D clade viruses
Nef(74–81)	Nef()	VPLRPMTY		human(B35)	[Rowland-Jones (1999)]
					<ul style="list-style-type: none"> • CTL responses in seronegative highly HIV-exposed African female sex workers in Gambia and Nairobi were studied – these women had no delta 32 deletion in CCR5 • In Gambia there is exposure to both HIV-1 and HIV-2, CTL responses to B35 epitopes in exposed, uninfected women are cross-reactive, • HIV-2 version of this epitope is conserved: VPLRPMTY, and CTLs are cross-reactive – one of five B35 CTL epitopes that are cross-reactive, see also [Rowland-Jones (1995)]
Nef(74–82)	Nef(73–82)	VPLRPMTYK	no CTL shown	human(A11)	[Zhang (1993)]
					<ul style="list-style-type: none"> • Exploration of A11 binding motif

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(75–82)	Nef(75–82 LAI)	PLRPMTYK	HIV-1 infection	human(A*1101)	[McMichael & Walker(1994)] <ul style="list-style-type: none"> • Review of HIV CTL epitopes • C. Brander notes that this is an A*1101 epitope in the 1999 database
Nef(75–82)	Nef(75–82 LAI)	PLRPMTYK	HIV-1 infection	human(A*1101)	[Brander & Goulder(2001)] <ul style="list-style-type: none"> • C. Brander notes this is an A*1101 epitope
Nef(77–85)	Nef(77–85 LAI)	RPMTYKAAL	HIV-1 infection	human(B*0702)	[Bauer (1997)] <ul style="list-style-type: none"> • Structural constraints on the Nef protein may prevent escape • Noted in Brander 1999, this database, to be B*0702
Nef(77–85)	Nef(77–85 LAI)	RPMTYKAAL	HIV-1 infection	human(B*0702)	[Brander & Goulder(2001)] <ul style="list-style-type: none"> • C. Brander notes this is a B*0702 epitope
Nef(82–91)	Nef(82–91 LAI)	KAAVDLSHFL	HIV-1 infection	human(C*0802)	[Nixon (1999)] <ul style="list-style-type: none"> • A patient who made a mono-specific CTL response to this Nef specific epitope was given effective anti-retroviral therapy within 90 days of infection, reducing the antigenic stimulus • Within 7 days of therapy, his CTLp frequency dropped from 60 to 4 per million PBMC, as his viremia dropped • The patient went from having an activated effector population (detected by CTLp and clone specific RNA) to a non-activated quiescent population (detected by the CTL-clone specific DNA)
Nef(82–91)	Nef(82–91 LAI)	KAAVDLSHFL	HIV-1 infection	human(C*0802(Cw8))	[Brander & Goulder(2001)] <ul style="list-style-type: none"> • C. Brander notes this is a C*0802(Cw8) epitope
Nef(84–91)	Nef(84–91 LAI)	AVDLSHFL	HIV-1 infection	human(Bw62)	[Culmann-Penciolelli (1994)]
Nef(84–91)	Nef(84–91)	AVDLSHFL	HIV-1 infection	human(Bw62)	[Betts (2000)] <ul style="list-style-type: none"> • Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninety five optimally defined peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals that didn't respond to SLYNTVATL reacted with seven other epitopes including this epitope

Table 6: **All Defined Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(72–79)	Nef()	VPLRPMTY	HIV-1 exposed seronegative	human(B35)	[Kaul (2000)]
					<ul style="list-style-type: none"> • 11/16 heavily HIV exposed but persistently seronegative sex-workers in Nairobi had HIV-specific CD8 gamma-IFN responses in the cervix – systemic CD8+ T cell responses tended to be to the same epitopes but at generally lower levels than cervical CD8+ T cell responses • Low risk individuals did not have such CD8+ cells • CD8+ epitopes T cell DTVLEDINL (3 individuals), SLYNVATL (4 individuals), LSPRTLNAW (3 individuals) and YPLTFGWCF (4 individuals) were most commonly recognized by the HIV-resistant women
Nef(72–79)	Nef()	VPLRPMTY	HIV-1 infection	human(B35)	[Wilson (2000)]
					<ul style="list-style-type: none"> • Three individuals with highly focused HIV-specific CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-specific CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-specific T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been defined earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWILGGLNK • The subject with A*0201 had a moderately strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL
Nef(72–91)	Nef(71–90 SF2)	PQVPLRMTYKAAVDLSHFL	HIV-1 infection	human()	[Lieberman (1997a)]
					<ul style="list-style-type: none"> • Of 25 patients, most had CTL specific for more than 1 HIV-1 protein • Eleven subjects had CTL that could recognize vaccinia-expressed LAI Nef • Three of these 11 had CTL response to this peptide • The responding subjects were HLA-A3, A32, B51, B62; HLA-A11, A24, B8, B53
Nef(72–91)	Nef(71–90 SF2)	PQVPLRMTYKAAVDLSHFL	HIV-1 infection	human()	[Lieberman (1997b)]
					<ul style="list-style-type: none"> • CTL expanded <i>ex vivo</i> were later infused into HIV-1 infected patients
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV infection	human()	[Garcia (1997)]
					<ul style="list-style-type: none"> • The anti-Nef CTL line P1 specific for this epitope is able to kill target cells via two mechanisms • First: Ca²⁺-dependent, perforin-dependent Nef-specific lysis • Second: Ca²⁺-independent, CD95-dependent apoptosis that could also kill non-specific targets • Findings indicate that the two mechanisms are not mutually exclusive in human CTL, as they are in mice • CTL mediated CD95-dependent apoptosis may play a role in pathogenesis

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(73-82)	Nef(73-82 NL43) • 81 Tyr is critical for binding to A3.1 • C. Brander notes that this is an A*0301 epitope in the 1999 database	QVPLRPMTYK	HIV-1 infection	human(A*0301)	[Koenig (1990)]
Nef(73-82)	Nef(73-82 LAI) • C. Brander notes this is an A*0301 epitope	QVPLRPMTYK		human(A*0301)	[Brander & Goulder(2001)]
Nef(73-82)	Nef(73-82) • Soluble factors in supernatant from both an HIV-specific cloned CTL line and an EBV (Epstein-Barr-virus) CTL line inhibit viral replication, but do not block viral entry in CD4+ T lymphocytes, by a noncytotoxic mechanism	QVPLRPMTYK	HIV-1 infection	human(A11)	[Le Borgne (2000)]
Nef(73-82)	Nef(73-82 LAI) • Development of a retroviral vector (pNeoNef) to generate autologous CTL targets • [Hunziker (1998)] suggests that HLA-A2 does not in fact present this epitope • The initial assignment of HLA-A2 presentation for this epitope was based on a serological HLA typing. Subsequently, the authors revisited the issue with genetic HLA typing and found that HLA-A11 was the correct presenting molecule (Dr. Florence Buseyne, Pers. Comm., 2000)	QVPLRPMTYK	HIV-1 infection	human(A11)	[Robertson (1993)]
Nef(73-82)	Nef(73-82 LAI) • Mutational variation in HIV epitopes in individuals with appropriate HLA types can result in evasion of CTL response • [Goulder (1997a)] is a review of immune escape that summarizes this study	QVPLRPMTYK	HIV-1 infection	human(A11)	[Couillin (1994), Goulder (1997a)]
Nef(73-82)	Nef(73-82 LAI) • Mutations found in this epitope in HLA-A11 positive and negative donors were characterized	QVPLRPMTYK	HIV-1 infection	human(A11)	[Couillin (1995)]
Nef(73-82)	()	QVPLRPMTYK		(A11)	[Brander & Goulder(2001), Buseyne(1999)]
Nef(73-82)	Nef(73-82 LAI) • Mutations in Nef that flank this epitope, Thr71Lys and Ala83Gly, may account for an observed loss of CTL reactivity, with escape due to the introduction of proteasome processing defects	QVPLRPMTYK	HIV-infection	human(A3)	[Chassin (1999)]

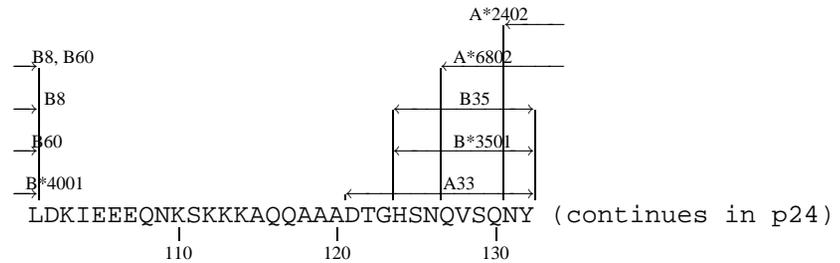
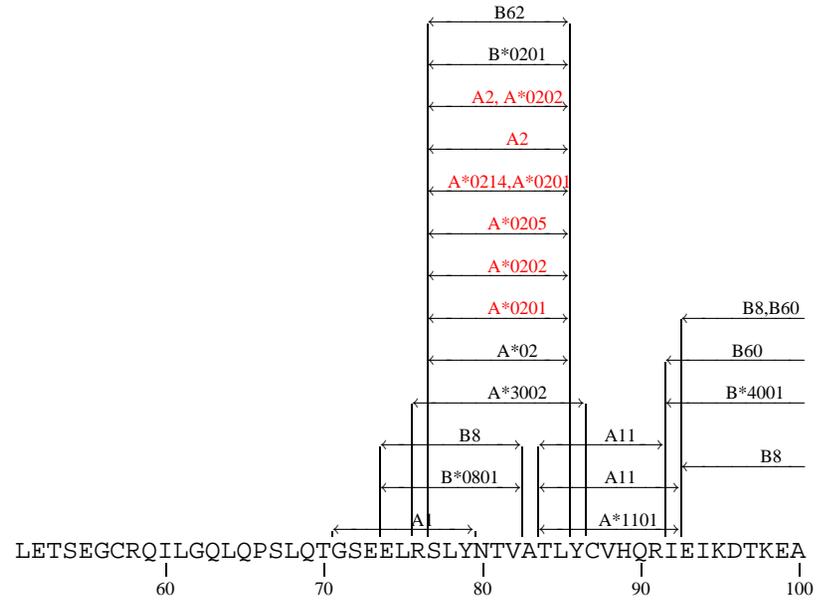
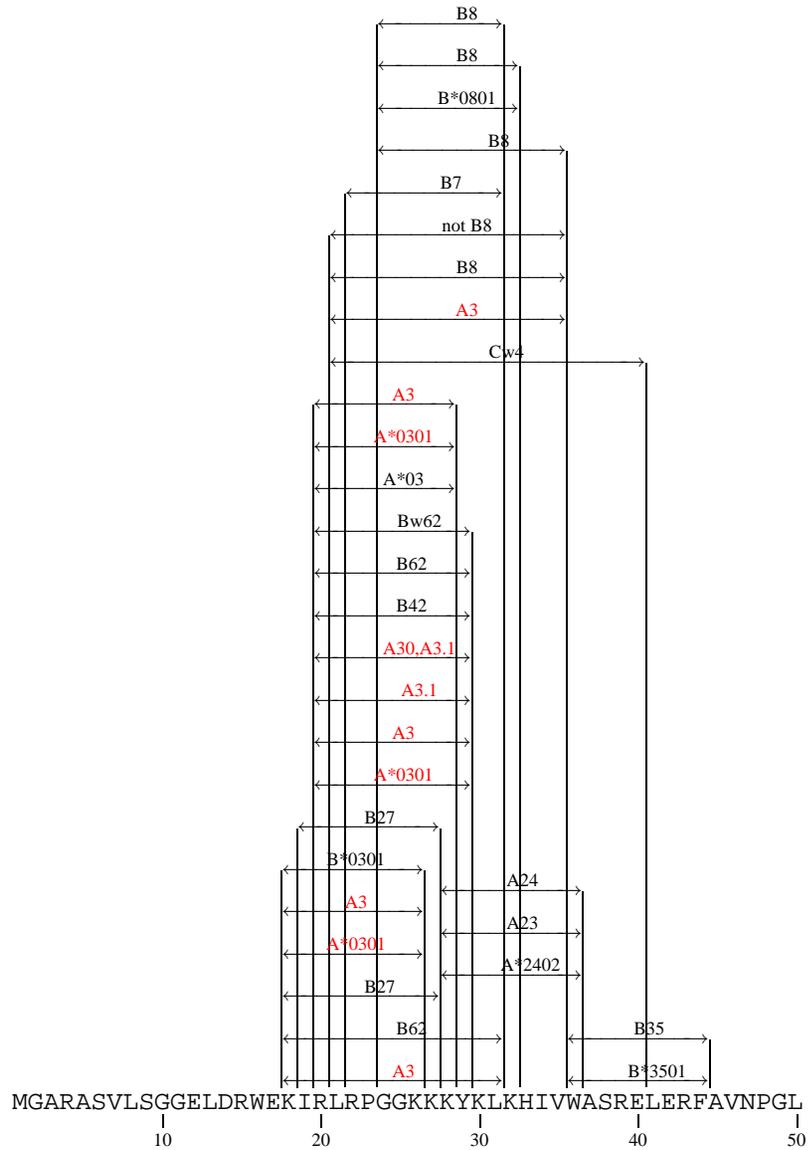
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV-1 infection	human(A3)	[Durali (1998)]
					<ul style="list-style-type: none"> • Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • One of the patients was shown to react to this epitope: QVPLRPMTYK
Nef(73–82)	Nef(73–82 LAI)	QVPLRPMTYK	HIV-1 infection	human(A3)	[Goulder (1997b), Goulder (1997a)]
					<ul style="list-style-type: none"> • Identical twin hemophiliac brothers were both infected with the same batch of factor VIII • Both had a response to this epitope • [Goulder (1997a)] is a review of immune escape that summarizes this study
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV-1 infection	human(A3)	[Lubaki (1997)]
					<ul style="list-style-type: none"> • Eighty two HIV-1-specific CTL clones from 5 long-term non-progressors were isolated and analyzed for breadth of response • A sustained Gag, Env and Nef response was observed, and clones were restricted by multiple HLA epitopes, indicating a polyclonal response • An A3+ subject had a strong response to this epitope, with 10/11 CTL clones being specific for this epitope, isolated at two time points, 1 year apart
Nef(73–82)	Nef(73–82 BRU)	QVPLRPMTYK	HIV-1 infection	human(A3, A11, B35)	[Culmann (1991)]
					<ul style="list-style-type: none"> • Nef CTL clones from HIV+ donors
Nef(73–82)	Nef(73–82 LAI)	QVPLRPMTYK	HIV-1 infection	human(A3.1)	[Koenig (1995)]
					<ul style="list-style-type: none"> • Alanine substitutions L76A, R77A, M79A, T80A significantly decreased immunogenicity of peptide • Nef CTL clones (4N225) were infused into an HIV-1 infected volunteer to evaluate effects of infusion on viral load/patient health • Infusion led to outburst of escape variants which resulted in higher viral load/accelerated disease progression
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV-1 infection	human(A3.1)	[Betts (2000)]
					<ul style="list-style-type: none"> • Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninety five optimally defined peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was A3, and responded to QVPLRPMTYK as well as two other A3.1 epitopes

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(73–82)	Nef(73–82)	QVPLRPMTYK	HIV-1 infection	human(B*0301)	[Wilson (2000)]
					<ul style="list-style-type: none"> • Three individuals with highly focused HIV-specific CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-specific CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-specific T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been defined earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWILGGLNK • The subject with A*0201 had a moderately strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL
Nef(73–82)	Nef(73–82 LAI)	QVPLRPMTYK		human(B27)	[Culmann(1998)]
					<ul style="list-style-type: none"> • Optimal epitope mapped by peptide titration
Nef(73–82)	Nef(73–82 LAI)	SVPLRPMTYK	HIV-1 infection	human(B35 or C4)	[Buseyne (1993)]
					<ul style="list-style-type: none"> • Vertical transmission of HIV ranges from 13% to 39% • Primary assays showed cytotoxic activity against at least one HIV protein was detected in 70% of infected children • Epitopes recognized in five children were mapped using synthetic peptides and secondary cultures • Patient EM13, who had a CTL response to three epitopes in Nef, was infected via blood transfusion after birth and went from CDC stage P2A to P2E during the study
Nef(74–81)	Nef(74–82)	VPLRPMTY		human(A3)	[Carreno (1992)]
					<ul style="list-style-type: none"> • Included in HLA-A3 binding peptide competition study
Nef(74–81)	Nef(73–82 LAI)	VPLRPMTY	HIV-1 or HIV-2 infection	human(B*3501)	[Brander & Goulder(2001)]
					<ul style="list-style-type: none"> • C. Brander notes this is a B*3501 epitope
Nef(74–81)	Nef(75–82)	VPLRPMTY	no CTL shown	human(B*3501)	[Smith (1996)]
					<ul style="list-style-type: none"> • Crystal structure of VPLRPMTY-class I B allele HLA-B*3501 complex
Nef(74–81)	Nef(73–82 LAI)	VPLRPMTY	HIV-1 or HIV-2 infection	human(B35)	[McMichael & Walker(1994), Culmann (1991)]
					<ul style="list-style-type: none"> • Review of HIV CTL epitopes – defined by B35 motif found within a larger peptide

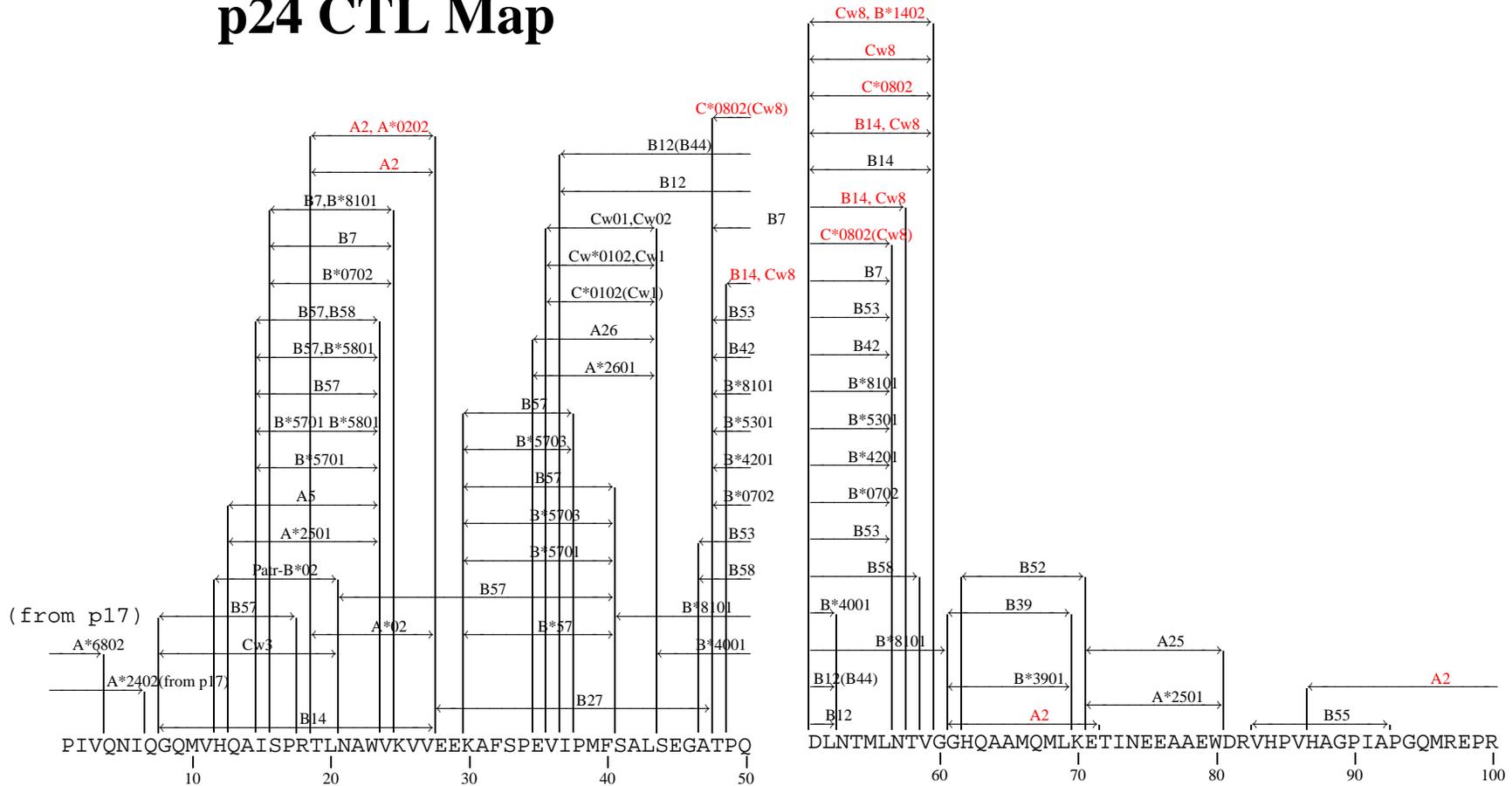
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(74–81)	Nef(73–82 LAI)	VPLRPMTY	HIV-1 or -2 infection	human(B35)	[Rowland-Jones (1995)]
					<ul style="list-style-type: none"> • VPLRPMTY also recognized by CTL from HIV-2 seropositives; epitope is conserved
Nef(74–81)	Nef()	VPLRPMTY	HIV-1 exposure	human(B35)	[Rowland-Jones (1998a)]
					<ul style="list-style-type: none"> • A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-defined B clade epitopes that tended to be conserved in A and D clades – such cross-reactivity could protect against both A and D and confer protection in Nairobi where both subtypes are circulating • The A and D subtype consensus are identical to the B clade epitope
Nef(74–81)	Nef(75–82)	VPLRPMTY	none	human(B35)	[Lalvani (1997)]
					<ul style="list-style-type: none"> • A peptide-based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-specific CTLp counts could be obtained via staining with peptide-Class I tetramers • This peptide was one of the B35 presented test peptides used in control experiments showing that the assay gave no activity using lymphocytes from 21 healthy B35 seronegative donors
Nef(74–81)	Nef()	VPLRPMTY	HIV-1 exposure	human(B35)	[Rowland-Jones (1998b)]
					<ul style="list-style-type: none"> • HIV-specific CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A, B, and D clade viruses
Nef(74–81)	Nef()	VPLRPMTY		human(B35)	[Rowland-Jones (1999)]
					<ul style="list-style-type: none"> • CTL responses in seronegative highly HIV-exposed African female sex workers in Gambia and Nairobi were studied – these women had no delta 32 deletion in CCR5 • In Gambia there is exposure to both HIV-1 and HIV-2, CTL responses to B35 epitopes in exposed, uninfected women are cross-reactive, • HIV-2 version of this epitope is conserved: VPLRPMTY, and CTLs are cross-reactive – one of five B35 CTL epitopes that are cross-reactive, see also [Rowland-Jones (1995)]
Nef(74–82)	Nef(73–82)	VPLRPMTYK	no CTL shown	human(A11)	[Zhang (1993)]
					<ul style="list-style-type: none"> • Exploration of A11 binding motif

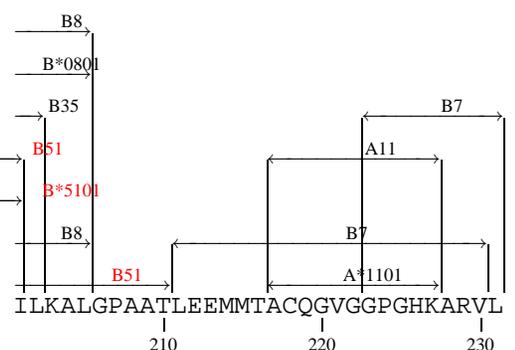
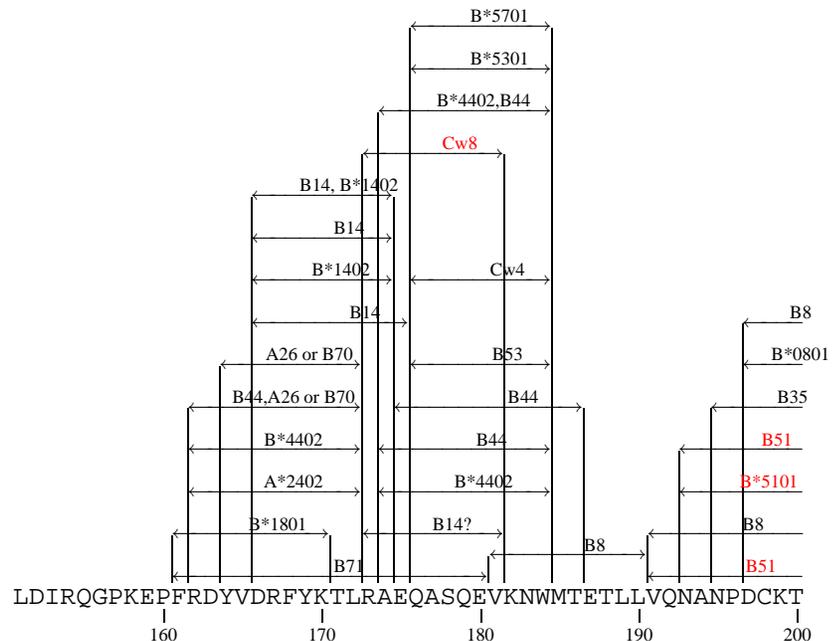
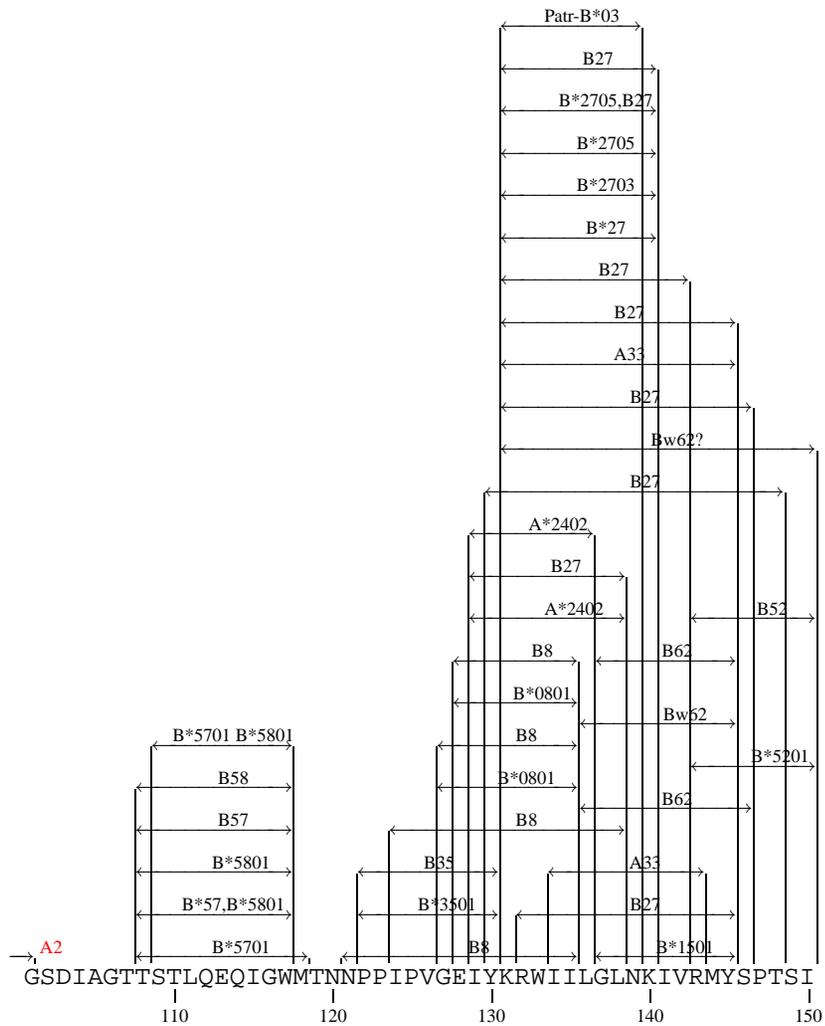
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(75–82)	Nef(75–82 LAI)	PLRPMTYK	HIV-1 infection	human(A*1101)	[McMichael & Walker(1994)] <ul style="list-style-type: none"> • Review of HIV CTL epitopes • C. Brander notes that this is an A*1101 epitope in the 1999 database
Nef(75–82)	Nef(75–82 LAI)	PLRPMTYK	HIV-1 infection	human(A*1101)	[Brander & Goulder(2001)] <ul style="list-style-type: none"> • C. Brander notes this is an A*1101 epitope
Nef(77–85)	Nef(77–85 LAI)	RPMTYKAAL	HIV-1 infection	human(B*0702)	[Bauer (1997)] <ul style="list-style-type: none"> • Structural constraints on the Nef protein may prevent escape • Noted in Brander 1999, this database, to be B*0702
Nef(77–85)	Nef(77–85 LAI)	RPMTYKAAL	HIV-1 infection	human(B*0702)	[Brander & Goulder(2001)] <ul style="list-style-type: none"> • C. Brander notes this is a B*0702 epitope
Nef(82–91)	Nef(82–91 LAI)	KAAVDLSHFL	HIV-1 infection	human(C*0802)	[Nixon (1999)] <ul style="list-style-type: none"> • A patient who made a mono-specific CTL response to this Nef specific epitope was given effective anti-retroviral therapy within 90 days of infection, reducing the antigenic stimulus • Within 7 days of therapy, his CTLp frequency dropped from 60 to 4 per million PBMC, as his viremia dropped • The patient went from having an activated effector population (detected by CTLp and clone specific RNA) to a non-activated quiescent population (detected by the CTL-clone specific DNA)
Nef(82–91)	Nef(82–91 LAI)	KAAVDLSHFL	HIV-1 infection	human(C*0802(Cw8))	[Brander & Goulder(2001)] <ul style="list-style-type: none"> • C. Brander notes this is a C*0802(Cw8) epitope
Nef(84–91)	Nef(84–91 LAI)	AVDLSHFL	HIV-1 infection	human(Bw62)	[Culmann-Penciolelli (1994)]
Nef(84–91)	Nef(84–91)	AVDLSHFL	HIV-1 infection	human(Bw62)	[Betts (2000)] <ul style="list-style-type: none"> • Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninety five optimally defined peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals that didn't respond to SLYNTVATL reacted with seven other epitopes including this epitope

p17 CTL Map

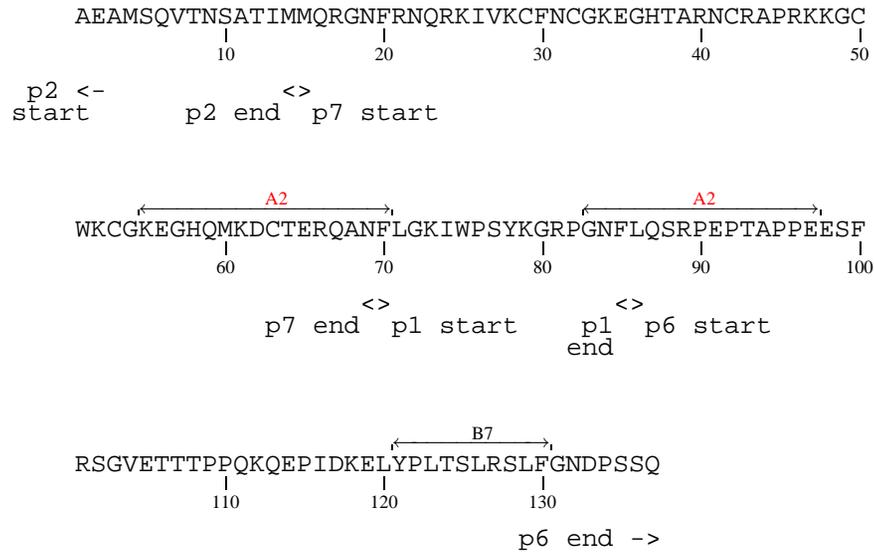


p24 CTL Map

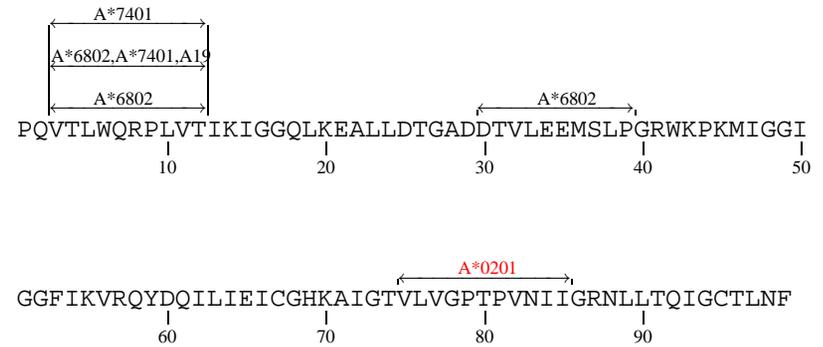




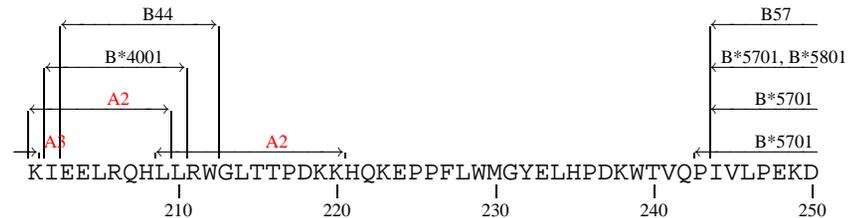
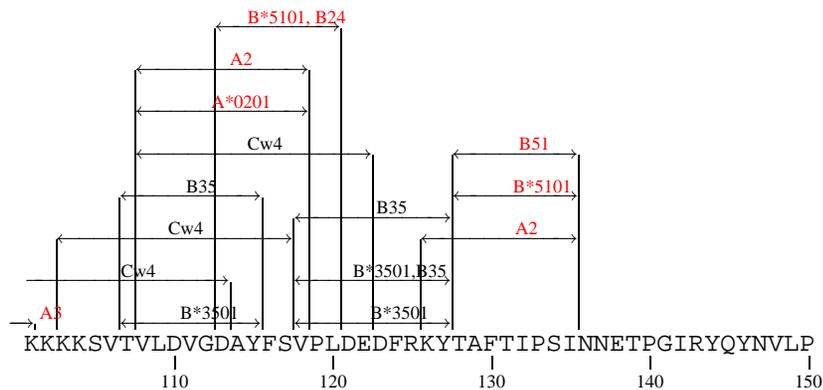
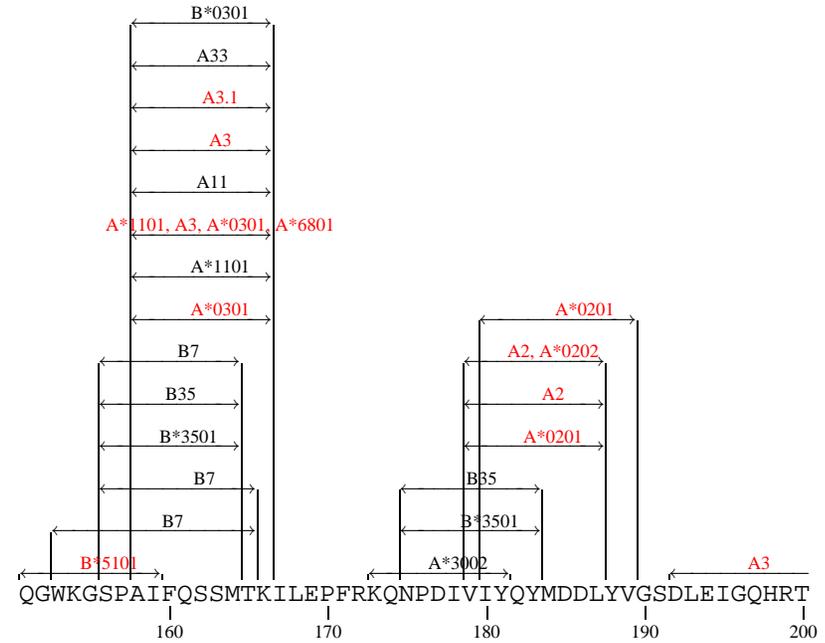
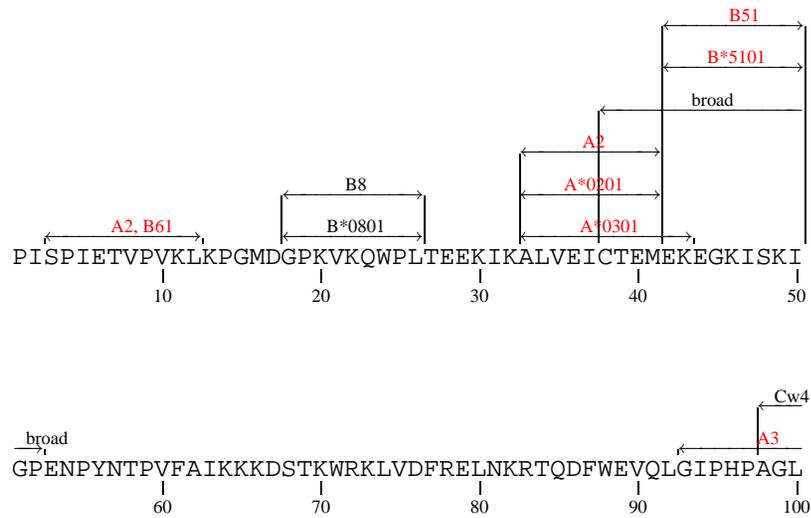
p2p7p1p6 CTL Map

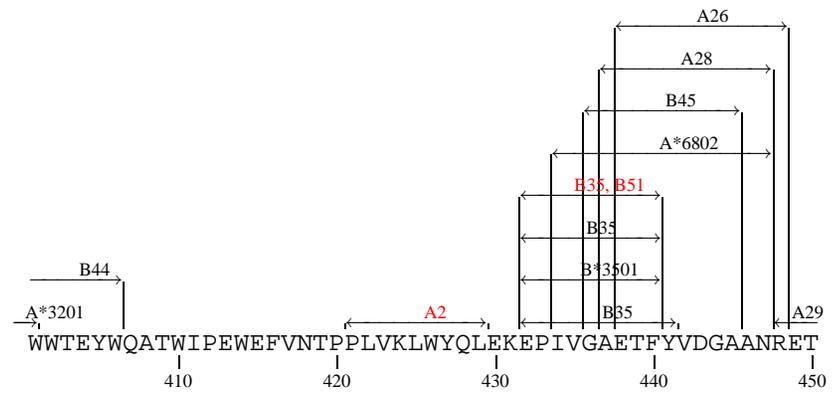
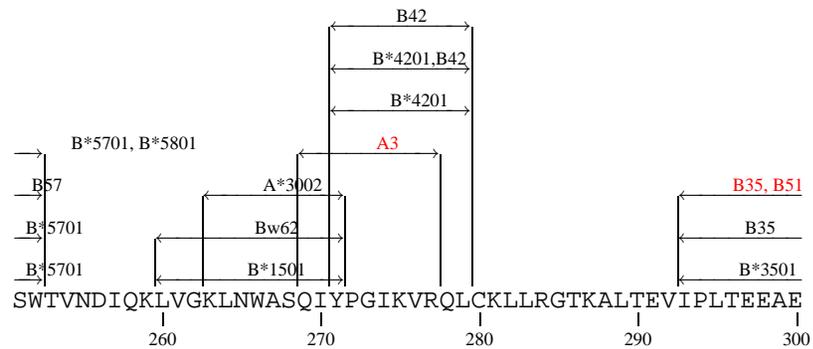


Protease CTL Map

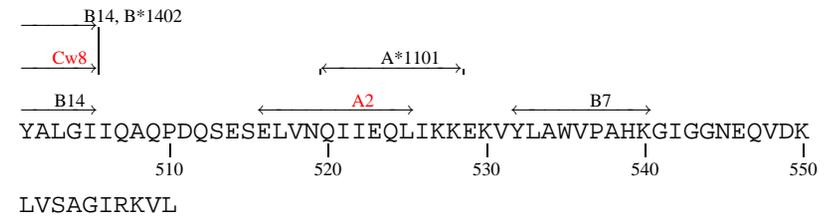
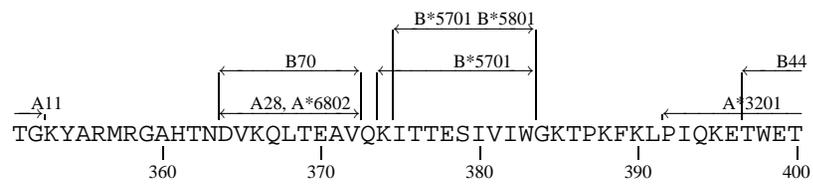
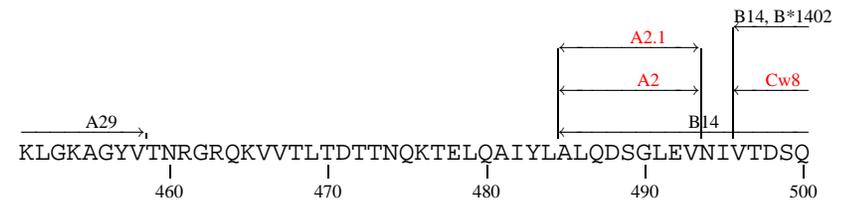


RT CTL Map



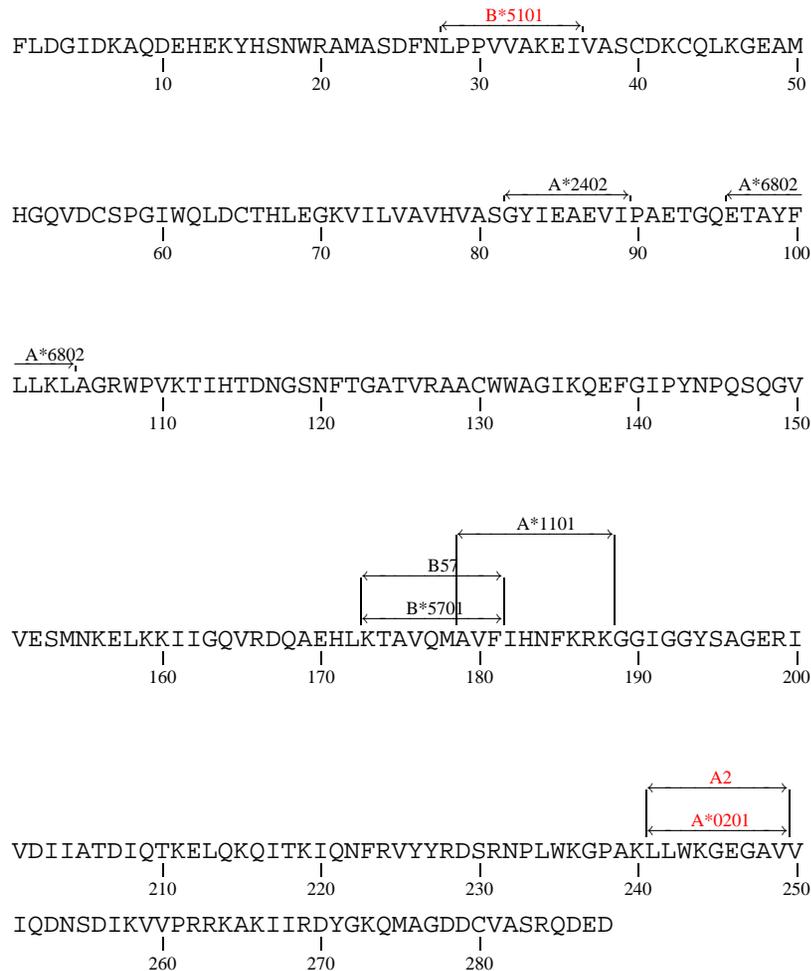


p15 RNase start <-

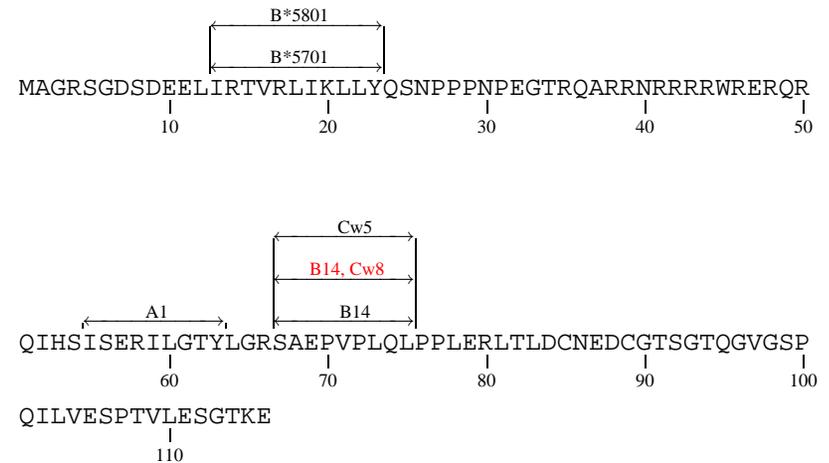


-> p15 RNase end

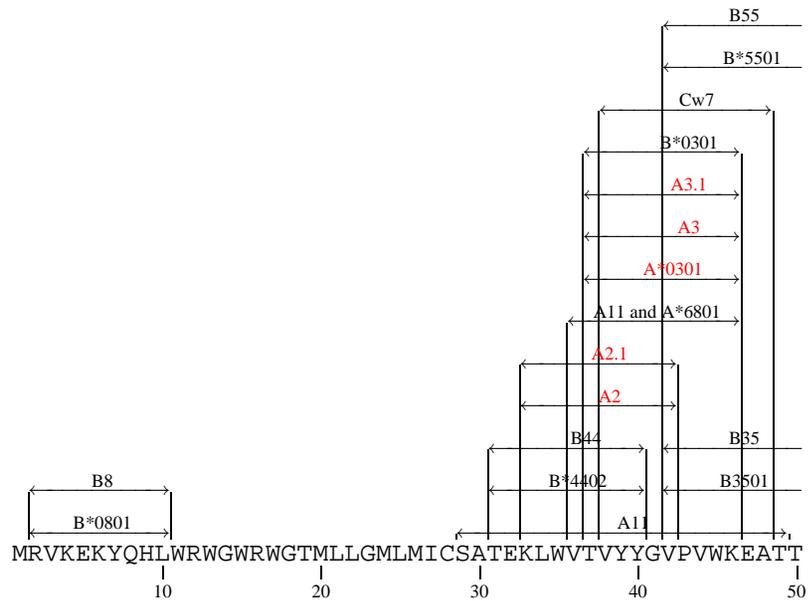
Integrase CTL Map



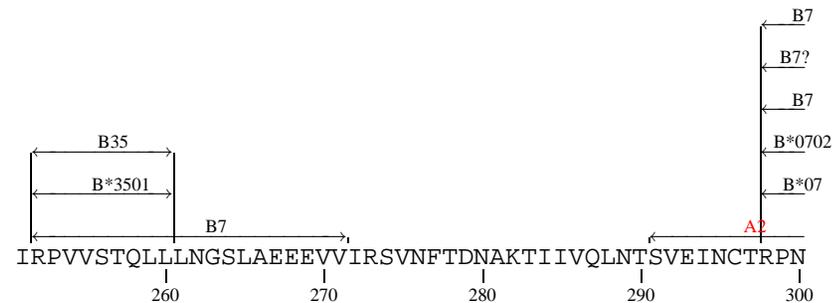
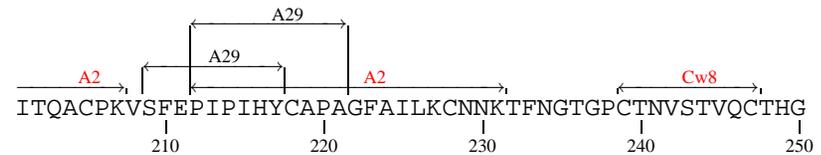
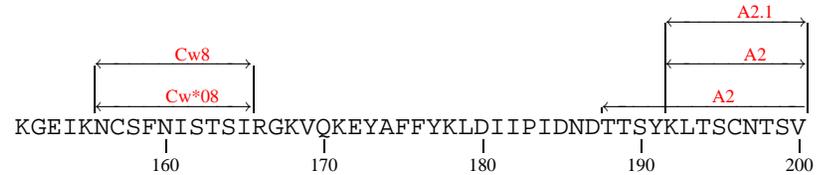
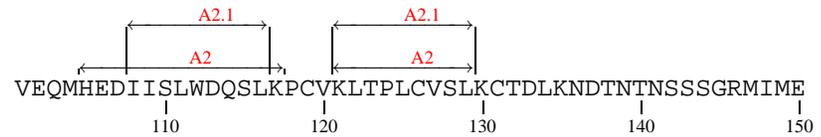
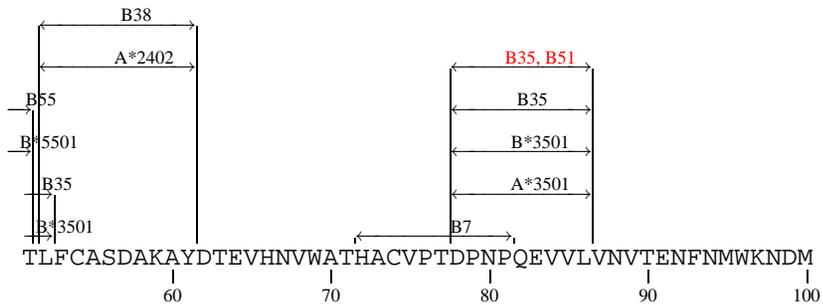
Rev CTL Map

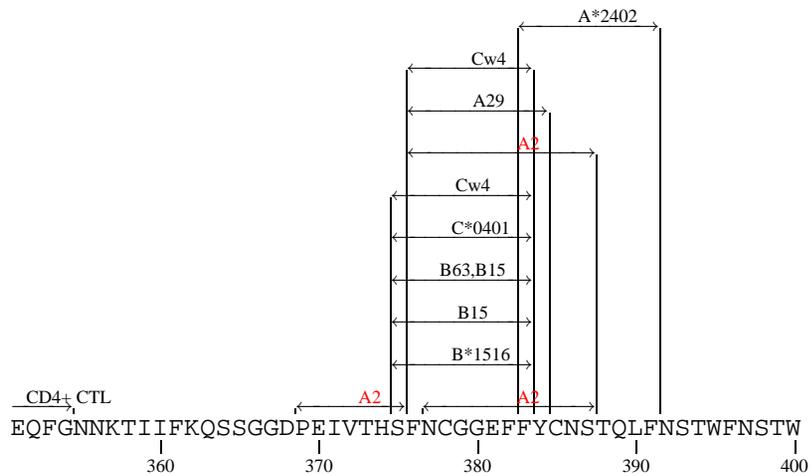
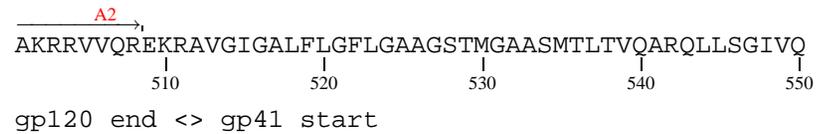
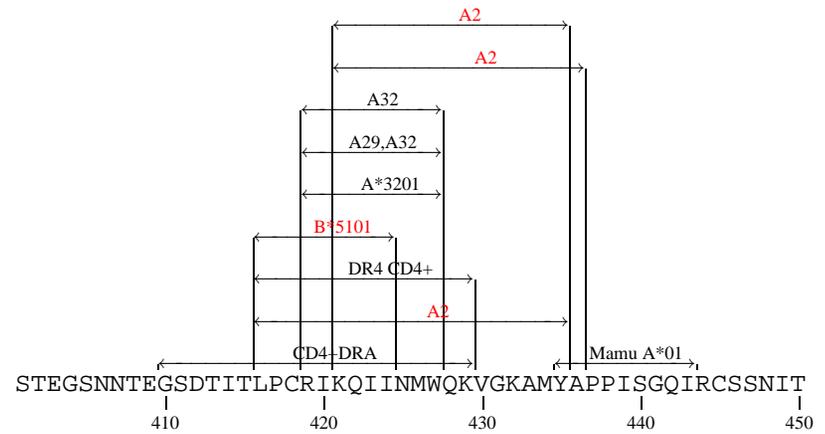
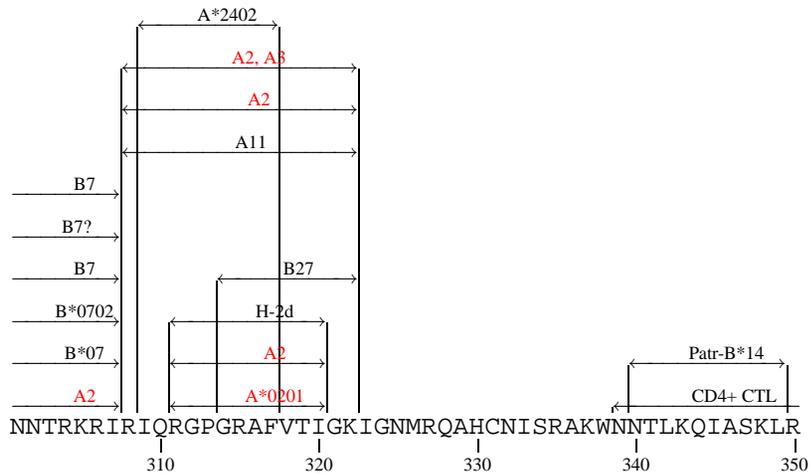


gp160 CTL Map

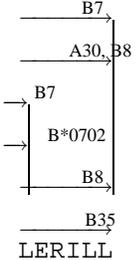


<- gp120 start

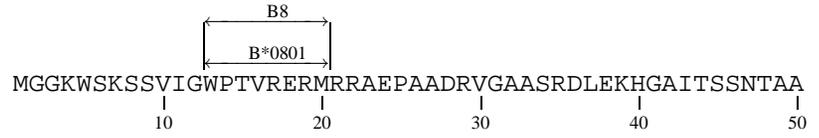


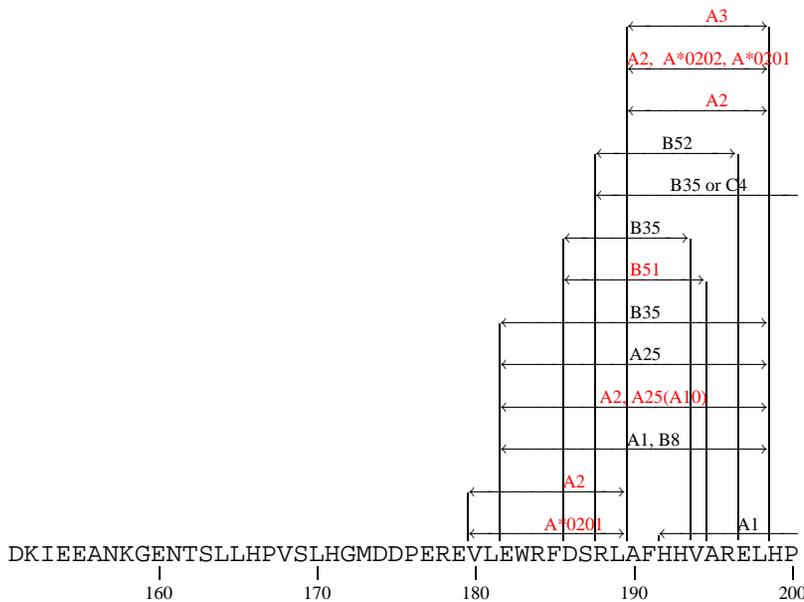


Nef CTL Map



-> gp41 end





$\xrightarrow{\text{A1}}$
 EYFKNC

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- cytotoxic T cells in HIV-exposed but uninfected Gambian women. *Nature Medicine* **1**:59–64, 1995. (Medline: 96071373) Notes: Four HIV-1 and -2 cross-reactive epitopes that are presented to CTL from HIV-infected Gambians by HLA-B35 were identified. These peptides could elicit HIV-specific CTLs from 3 of 6 repeatedly exposed but seronegative sex workers who carry the HLA-B35 allele. Most CTL derived from HIV-2 positive donors also recognized the HIV-2 peptide and the analogous HIV-1 peptide.
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