GVRYPLTFGWCYKLVPVEPD

QUERY	GVRYPLTFGWCYKLVPVEPD
CONSENSUS_A	-?---------f-----d--
A.FR.HIV232956	-T---------F-----D-V
A.FR.HIV232957	-T-F-------F-----D-S
A.FR.HIV232959	-T-F-------F-----D-A
A.KE.Q23-CXC-CG	-T-F-------F-----D--
A.SE.SE6594	-I-F-------F-----D--
A.SE.SE7253	----------F-----D--
A.SE.SE7535	-I---------F-----D--
A.SE.SE8131	-I---------F-----D--
A.SE.SE8538	----------F-------E
A.SE.SE8891	-T-F-------F-----D--
A.UG.92UG037	-I---------F-----DE-
A.UG.U455	-I---------------D-A
CONSENSUS_B	-i---------f-------e
B.-.E90NEF	-T---------F-------E
B.-.HIV232997	-I-F---xx--F-----PAE
B.-.HIV233002	-T-----L---F-------E
B.-.HIV233009	-T---------FR------E
B.-.HIV233016	-I---------F-------E
B.-.HIV233020	-I----C----F-----S-E
B.-.HIV233023	-T---VC----F-------E
B.-.HIV233029	-I---------F-------E
B.-.HIV233030	-T-F-------F-------E
B.-.HIV233032	-T-C--CL---F-------E
B.-.HIV233037	------------R------E
B.-.HIV233038	-T-F-------F-------E
B.-.HIV233043	-I---------F-------E
B.-.HIV233045	-I---------F------AE
B.-.HIV233046	---F--CL---F-----S-E
B.AU.1062-1-NEF	-T---------F-------G
B.AU.93JW-3	-P-F-------F-----D-E
B.AU.93LW-3	-P-F-------F-----D-E
B.AU.AF064660	-I---------F-------E
B.AU.AF064667	-P-F-------F--------
B.AU.AF064676	-I----C----F-------E
B.AU.MBC200	-T-W-------F-------E
B.AU.MBC925	-I---I-----F-------E
B.CN.AF033570	-T-F-------F-----D-K
B.CN.AF033572	-T-F-------F-----D-K
B.CN.PRC8	-T-F-------F-----D-E
B.CN.RL42	-T-F-------F-----D-E
B.DE.D31	-T-F-------F-----K-E
B.DE.HAN	----------F--------
B.DE.HEI28CS	---W-------F------xE
B.DE.HEI3BL	----------F-------x
B.DE.HEI4BL	---F-------F-----x-E
B.DE.HIVU52491	-T-F-------F-----D-E
B.DE.NEFCC	-T-----L---F--------
B.DE.NEFCG	-I---------F-----D-E
B.DE.NH53	-I---------F-----G--
B.ES.89SP061	-I---------F-------E
B.ES.AF082355	-I---------F------RE
B.ES.AF082357	-I---------F-----D-E
B.ES.AF082358	-T-F-------F-----D--
B.ES.AF082359	-I-------------L-QE
B.ES.AF082363	------C----F-------G
B.ES.AF082364	-I-----L---F-----D-E
B.ES.AF082366	-I---------F-----D-G
B.ES.AF082368	-T-F-------F--------
B.ES.AF082370	-T-F-------F-------E
B.ES.AF082375	-I---------F-----D--
B.ES.AF082376	-T---------F-----D--
B.ES.AF082377	-I---------F-----D-E
B.ES.AF082378	-I---------F-----D-E
B.ES.AF082380	-T-F-------F-----K-E
B.ES.AF082383	----------F-----D-E
B.ES.AF082386	---F-------F--------
B.FR.HIV232961	---F-------F-----K-N
B.FR.HIV232962	-I---------F------QE
B.FR.HIV232963	-T-F-------F-----D--
B.FR.HIV232964	-T-F-------F-------E
B.FR.HIV232965	-T----C----F-----D--
B.FR.HXB2	--------------------
B.FR.NE100	-I-W---L---F-------E
B.FR.SWB884	-I-W---P---F-------E
B.GA.OYI	-I----C----F----MD--
B.GB.001GH-93(1)	-I---------F-------E
B.GB.002EM-93(1)	---H-------F-----DQE
B.GB.003PW-93(1)	----------F------TE
B.GB.005PF1-93(1)	-I-F---S---F-------E
B.GB.006DC-93(1)	----------F-----D-A
B.GB.010JW-93(1)	-T-F-------F-------E
B.GB.011JR-93(4)	---F-------F-------E
B.GB.012WM-93(1)	-I---------F----M---
B.GB.013PP-94(2)	-T-F---Y---F--------
B.GB.016GB-93(1)	-TT--------F-------E
B.GB.023PA-93(1)	----------F------SE
B.GB.025JN-93(1)	----------F-------E
B.GB.027SL-93(1)	-I---------F-------E
B.GB.028JH-94(1)	-I---------F--------
B.GB.030JG-93(1)	-I---------F-------E
B.GB.031DA-93(1)	-I---------F-------E
B.GB.032AN-93(1)	---W-------F-------E
B.GB.037BS-94(2)	----------F-----D-E
B.GB.039NM-94(1)	-T---------F-------E
B.GB.044C1-94(2)	-I---------F--------
B.GB.046JM-94(1)	-I---------F--------
B.GB.048AD-94(1)	-T---------F-------E
B.GB.056RP-94B(1)	-I---------F-----D-E
B.GB.057DR-94(1)	-I---------F------SE
B.GB.065RK-94(1)	-T-F-------F-----D--
B.GB.067MM-94(2)	----------F-------E
B.GB.068JB-94(1)	----------F-------E
B.GB.098MS-94(1)	-I-----Y---F-------E
B.GB.103CD-94(1)	-I---------F--------
B.GB.104RT-94(1)	-I---------F-------E
B.GB.105AS-94(1)	-T---------F--------
B.GB.112CR-94(2)	-I---------F-----D--
B.GB.117CH-94(2)	-I---------F--------
B.GB.122PS-95(1)	---F-------F-----D-E
B.GB.124PD-95(1)	-T-F-I-----F-------E
B.GB.127RG-96(1)	-------L----------E
B.GB.130WDC-95(1)	-T-F--C----F-------E
B.GB.131MVS-95(1)	-T---------F----LD--
B.GB.143PL-95(1)	-I----------------E
B.GB.151DH-95(1)	-----I-----F-------E
B.GB.157GT-95(1)	-T---------F-----D-E
B.GB.160KO-95(1)	-I-----L---F-----D--
B.GB.161KC-95(1)	-I---------F-------G
B.GB.162BB-95(1)	-TKF-------F-------E
B.GB.163NG-95(1)	-T-F-------F-----D-E
B.GB.164SZ-95(1)	----------F-----D--
B.GB.165DH-95(1)	----------F-------E
B.GB.166PW-95(1)	-I---------F----M---
B.GB.167RW-95(1)	--------R--F-------E
B.GB.168MB-95(1)	----------F-------E
B.GB.CAM1	-I---------F-------E
B.GB.GLNEF1	--------------------
B.GB.MANC	-I----A----F-----D-E
B.GB.NEF2	----------F-------G
B.GB.NEF3	---F-------F--------
B.GB.NEF5	----------F-------E
B.IN.HIVP35A	-I---------F-----D-E
B.IT.AF011471	-I----------------E
B.IT.AF011474	----------F-------E
B.IT.AF011477	-I---------F-------E
B.IT.AF011478	-I---------F-------E
B.IT.AF011480	--------------------
B.IT.AF011482	-I---------F-----N-E
B.IT.AF011483	-T-F-------F--------
B.IT.AF011486	-I-----Y---F--------
B.IT.AF011488	-T-----YE--F-------E
B.IT.AF011492	------C----F------xx
B.IT.AF047080	-I---------F-------E
B.IT.AF047081	-I---------F-------E
B.IT.B.IT-L1	-x-x-------F-----D--
B.IT.B.IT-L2	-T---------F-----D-E
B.IT.B.IT-L3	-I---x-L---F-------E
B.IT.B.IT-L4	-T-----Y---F------TE
B.IT.B.IT-L5	-T---------F-------E
B.IT.B.IT-R1	-P-F------WC----L-KE
B.IT.B.IT-R2	-T-F-------F------QE
B.IT.B.IT-R3	-I--------SF-----D--
B.IT.B.IT-R4	-I---------F-------E
B.IT.B.IT-R5	-T---------F--E----E
B.KR.AF063915	-I---------F-------E
B.KR.AF063916	---F-------F-------E
B.KR.AF063919	-T---------F-------E
B.KR.AF063921	--------#--F-------E
B.KR.AF063926	-I---------F-------E
B.KR.AF063927	-T-F-------F-------E
B.KR.AF063931	---L-------FL------E
B.KR.HIVZ98019	-T-F-------F-------E
B.KR.HIVZ98022	-I---------F-----D-E
B.KR.HIVZ98024	---F-------F-------E
B.KR.HIVZ98025	-I---------F------KE
B.KR.HIVZ98027	-I---------F--------
B.KR.HIVZ98029	-T-F-------F-----D--
B.KR.HIVZ98030	-T-F-------F-------E

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B.KR.HIVZ98032        -I---------L-------E
B.KR.HIVZ98034        -T-F-------F-------E
B.NL.3202A21          -I---------F------QE
B.NL.NEFA             -P-F-------F-------E
B.NL.NEFD             -T-F-------F--------
B.NL.NEFE             -T---------L-------E
B.SE.AF047082         -T-F-------F-----D-V
B.SE.AF047083         -T---------F------KE
B.SE.AF047085         -T-F-------F-------E
B.TH.28-19            -----------F-------E
B.TH.AF082838         -T---------F-------E
B.TH.AF082839         -I---------F-------E
B.TH.AF082841         -I---------F-I--ID--
B.TW.LM49             -----------F-------E
B.US.HIV1U03375       -I---------F-----DQ-
B.US.005PF-96(1)      -I-F-------F------QE
B.US.AD-93(1)         -I---------F-------E
B.US.AD8              -----------F-------E
B.US.BC               -I---------F-----D-E
B.US.BIB              -----------F-------E
B.US.BJ-93(1)         -T-F-------F-------E
B.US.BO1              -----------F-------E
B.US.BRVA             -----------F-------E
B.US.BT-94(1)         -T-F-------F----M--E
B.US.CD1              -I---------F-------E
B.US.D8511            -T---------F-------E
B.US.DH1              RTGF--C-R--F-----D--
B.US.DH123            -I---------F-----D-E
B.US.DJ-93(1)         -I----C----F--------
B.US.E1               -I---------F-------E
B.US.E81NEF           -I---------F-------E
B.US.E88NEF           -T---------F-------E
B.US.EP-94(1)         -T----C----F-----D--
B.US.FA-93(1)         -----------F-----D-E
B.US.HIV1U16893       -T-F--C----F-----D--
B.US.HIV1U24455       -I---------F-------E
B.US.HIV1U26074       -T-W-------F-------E
B.US.HIV1U26098       -I---------F------KE
B.US.HIV1U26112       -I---------F-------E
B.US.HIV1U26119       -I---------F-------E
B.US.HIV1U26141       -I---------F-------Q
B.US.HIVU44444        -T---------F-------E
B.US.HIVU44450        -I---------F------QE
B.US.HIVU44456        -I---------F-------.
B.US.HIVU44465        ------C----F-----D-E
B.US.HIVU44468        -A-F-------F-----D--
B.US.HP87B1           -------L---F-----D-E
B.US.HS-93(1)         -T--------RF-------E
B.US.JRCSF            ---F-------F-----D-E
B.US.JRFL             -I-F-------F-------E
B.US.LM1              -T---------F----I--E
B.US.LT-87-1(1)       -----------F--A--D--
B.US.MB-94(1)         ------C----F-------E
B.US.MNCG             -I---------F-------E
B.US.NC7              -I---------F--------
B.US.NEF              --------------------
B.US.NEF164B          -I---------------DQE
B.US.NEF166E          -I---------F-------E

B.US.NEF179C          -----------F-----D--
B.US.NEF226B          -I---------F-------E
B.US.P102A13          -I---------F-------E
B.US.P233A17          -I---------F-------E
B.US.P248A01          -I---------F--------
B.US.P357A01          -I--------RF-----D--
B.US.P896             -I--------------------
B.US.PC-93(1)         -T-----L---F------TE
B.US.PRISO(1)         -----------F----A--G
B.US.RF               -T---------F--------
B.US.RP12             -T-F-------F------QE
B.US.RR1              -I---------F-------E
B.US.SC               -I----C----F-----K-E
B.US.SF2              -I---------F-------E
B.US.U16917           -I---------F-----D-E
B.US.WEAU160          -T----C----F-------E
B.US.WR27             -T---------F----L---
B.US.YU2              -T-W-------F-------E

CONSENSUS_C           -----------f-----D-r
C.BR.92BR025          ---F-------F-----D-R
C.BW.96BW01B21        -----------F-----D-R
C.BW.96BW0402         ----------------D-K
C.BW.96BW0502         --K--------F-----D-G
C.BW.96BW1104         -----------F-----D-G
C.BW.96BW1210         -----------F-----D-G
C.BW.96BW15B03        ----------------D-R
C.BW.96BW16B01        -----V-----F-----D-R
C.BW.96BW17A09        ----------------D-R
C.ET.ETH2220          -----------F-----D-S
C.FR.HIV232966        -----------F-----D-K
C.FR.HIV232967        -T---------F-----D-G
C.FR.HIV232968        -----------F-----D-G
C.FR.HIV232969        -----------F-----D-S
C.FR.HIV232970        ---F-------F-----D-R
C.FR.HIV232971        ----------PF-----D-R
C.FR.HIV232972        ----------PF-----D-R
C.FR.HIV232973        ---L-------F-----D-R
C.FR.HIV232976        -----------F-----D-E
C.FR.HIV232977        ---F-------F-----D-G
C.FR.HIV232978        -----------V--L--D-R
C.FR.HIV232979        ----------LF-----D-S
C.FR.HIV232980        ----------------D-R
C.FR.HIV232996        ---H-I----LF-----D-K
C.IN.21068            ---F-------F-----D-R
C.IN.301904           -----------F-----D-R
C.IN.301999           ---F-------F-----D-R
C.IN.94IN11246        -T-F-------F-----D-R
C.IN.HIVY15117        ---F-------F-----D-K
C.IN.HIVY17884        -T-F-------F-----D-R
C.IN.HIVY17891        -----------F-----D-R
C.IN.HIVY17892        -T-F-------F-----D-R

CONSENSUS_D           -I---------fe----d-q
D.CD.84ZR085          -I---------FE----D-E
D.CD.ELI              -I----------E----D-Q
D.CD.NDK              -I---------FQ----D-Q
D.UG.94UG1141         -I---------FE---M--K

CONSENSUS_F           -i-f-------F-----D-e
F.CM.HIV232985        -I-----L---F-----D-E
F.CM.HIV232986        -P-F-------F-----D-E
F.FR.HIV232987        -I-F-------F-----D--

CONSENSUS_F1          -i---------F-----d-e
F1.BE.VI850           -I-----L---F-----D-E
F1.BR.93BR020.1       -I-----M---F-----D-E
F1.FI.FIN9363         -----------F-------E
F1.FR.MP411           -I-F-------F-----D--

CONSENSUS_F2          -?-?---?---F-----D-E
F2.CM.MP255           -I-----L---F-----D-E
F2.CM.MP257           -P-F-------F-----D-E

CONSENSUS_G           -t-f-------F----md-a
G.BE.DRCBL            -T-V-------F----M--S
G.FI.HH8793           -T-F-------F--E--D-A
G.ML.HIV232990        -T-F-------F----MD-A
G.NG.92NG083          -T-L-------F----MD-A
G.NG.HIV232991        -I-F-------F----LD-T
G.NG.HIV232992        -I-F-------F----MD-A
G.SE.SE6165           -T-F-------F----MD-A

CONSENSUS_H           -e---------F-----d-q
H.BE.VI991            -E---------F-----D-Q
H.BE.VI997            -EG--------F--I--D-Q
H.CD.HIV232994        -----------F-----D-Q
H.CD.HIV232995        -----------F-----D-L
H.CF.90CF056          -E-F-------F-----N-Q

CONSENSUS_J           -??--------?-----D-S
J.SE.SE9173           -Tx--------F-----D-S
J.SE.SE9280           -I---------------D-S

CONSENSUS_K           -I---------------D-?
K.CD.EQTB11C          -I---------------D-R
K.CM.MP535            -I---------------D-A
N.CM.YBF30            -I---V-----F----LSAE

CONSENSUS_O           -?-F------LF-----S?E
O.CM.ANT70C           -T-F------LF-----SEE
O.CM.MVP5180          -P-F------LF-----SAE
CRF01_AE.CF.90CF402   ------C----F-----D-R
CRF01_AE.FR.232982    -I----C----F-----DTG
CRF01_AE.FR.232983    -I-F--C----F-----D-R
CRF01_AE.FR.232984    -I----C----F-----D-G
CRF01_AE.TH.1-2       -I----C----F-----D-R
CRF01_AE.TH.1-3       -I----C----F-----D-R
CRF01_AE.TH.11-25     -I-F--C----F-----D-K
CRF01_AE.TH.11-31     -I-F--C----F-----D-G
CRF01_AE.TH.122-21    -I----C----F-----D-R
CRF01_AE.TH.18-47     -I----C----F-----D-R
CRF01_AE.TH.235-3     -I----C----F-----D-R
CRF01_AE.TH.235-32    -I----C----F-----D-R
CRF01_AE.TH.24-54     -I----C----F-----D-R
CRF01_AE.TH.240-12    -I-F--C----F-----DQR
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CRF01_AE.TH.26-3      -I----C----F-----D-R
CRF01_AE.TH.35-6      -I----C----F-----DSR
CRF01_AE.TH.6-9       -I-F--C----F-----D-R
CRF01_AE.TH.73-44     ------C----F-----D-R
CRF01_AE.TH.74-26     -I----C----F-----DSG
CRF01_AE.TH.89-30     -I----C----F-----D-R
CRF01_AE.TH.9-3       -I----C----F-----D-R
CRF01_AE.TH.93TH253   -I----C----F-----D-R
CRF01_AE.TH.98-4      -I----C----F-----D-G
CRF01_AE.TH.CM240     -I-F--C----F-----DQR
CRF01_AE.TH.TH022     ---L--C----F-----D-R
CRF01_AE.TH.TH047     -I-F--C----F-----D-R
CRF02_AG.FR.DJ263     -T---------FN-E-ID-A
CRF02_AG.FR.DJ264     -T---------F--E-MD-A
CRF02_AG.NG.IBNG      -T-F-------F----MD-A
CRF03_AB.RU.KAL1532   -I-F-------------D-A
CRF04_cpx.CY.94CY03   -E-F--C----F-----D-Q
CRF04_cpx.GR.97PVCH   -E-F--C----F-----D-Q
CRF04_cpx.GR.97PVMY   -T-F--C----F-----D-Q
AC.IN.21301           -----------F-----D-R
AC.RW.92RW009         ----------------D-R
AC.SE.SE9488          -I---------F-----N--
AC.ZM.ZAM184          -T-F-------F-----D-S
ACD.SE.SE8603         -T---------F-----D-R
AD.SE.SE6954          -I----I----FE----D-K
AD.SE.SE7108          -T-F-------F-----D--
ADHU.NO.NOGIL3        -E-F-------F-----D-Q
ADU.CD.MAL            -I-F-------F----MS-E
AF.GA.HIV232981       -I-----S---------D-V
AG.NG.G3              -T-F----R--F----MD-A
AG.SE.SE7812          -T-F-------F----MD-A
AGHU.GA.VI354         -I----C----------D-K
AGJ.AU.BFP90          -T-F-------------D-E
AGJ.ML.95ML84         -I---------------D-R
AGU.CD.Z321           -T----C----F-----D-R
BF.BR.93BR029.4       -T-----L---F-----D-E
DF.BE.VI961           -T---------F-----D-E
GH.GA.HIV232993       ----------------LD-T
GU.FR.HIV232974       -T-F-------F----MD-S
U.CD.VI1126           -I----C----F----MD-Q
U.CM.HIV232988        -I---------------D-A
U.FR.HIV232958        -I----C----------D-R
U.FR.HIV232960        -I----C----------D-R

CONSENSUS_CPZ         -?-?--??---F----LTEE
CPZ.GA.CPZGAB         -T-F--C----F----LTEE
CPZ.US.CPZUS          -------Y---F----LTEE
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Study Subject ID:00RCH96
Study Subject Clone:
Study Subject HLA:A23,A30,B27,B35,Cw2,Cw4
Sequence: Known reactive 20Mer0: GVRYPLTFGWCYKLVPVEPD Nef(132–151)

Possible HLA

A23	A*2301
A30	A*3001,A*3002,A*3003,A*3004
B27	B*27,B*2701,B*2702,B*2703,B*2704,B*2705,B*2706,B*2707,B*2709,B*2710,B*2711,B*2713
B35	B*35,B*1522,B*3501,B*3502,B*3503,B*3504,B*3505,B*3506,B*3507,B*3508,B*3509,B*3511,B*3512,B*3513,B*3514,B*3515,B*3517,B*3518,B*3519,B*3520
Cw2	Cw*0202
Cw4	C4,Cw*0401,C*0401,Cw*0402

Possible Epitopes based on anchor residues

(2-10)	VRYPLTFGW	B27
(2-9)	VRYPLTFG	B27
(2-11)	VRYPLTFGWC	B27
(2-10)	VRYPLTFGW	B*2702
(4-12)	YPLTFGWCY	B*35
(4-12)	YPLTFGWCY	B*3501
(7-14)	TFGWCYKL	Cw*0401

Anchor Residues Searched

B27	X[R]XXXXXXX
B27	X[R]XXXXXX
B27	X[R]XXXXXXXX
B*2702	X[R]XXXXXX[FYILW]
B*2702	X[R]XXXXX[FYILW]
B*2702	X[R]XXXXXXX[FYILW]
B*2705	X[R]XXXXXX[LF]
B*2705	X[R]XXXXX[LF]
B*2705	X[R]XXXXXXX[LF]
B*35	X[P]XXXXXX[YFMLI]
B*35	X[P]XXXXX[YFMLI]
B*35	X[P]XXXXXXX[YFMLI]
B*3501	X[P]XXXXXX[YFMLI]
B*3501	X[P]XXXXX[YFMLI]
B*3501	X[P]XXXXXXX[YFMLI]
B*3503	X[P]XXXXXX[M]
B*3503	X[P]XXXXX[M]
B*3503	X[P]XXXXXXX[M]

Cw*0401 X[YPF]XXXXXX[LF]
Cw*0401 X[YPF]XXXXX[LF]
Cw*0401 X[YPF]XXXXXXX[LF]

This table lists epitopes that are experimentally observed to be presented by a HLA type carried by the patient, but the de£ned 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(124–132)	NSSKVSQNY	HSNQVSQNY	NSSQVSQNY	B*3501	
p17(124–132)	NSSKVSQNY	HSNQVSQNY	NSSQVSQNY	B35	
p24(122–130)	PPIPVGDIY	PPIPVGEIY	PPIPVGEIY	B*3501	
p24(122–130)	NPVPVGNIY	PPIPVGEIY	PPIPVGEIY	B*3501	
p24(122–130)	PPIPVGDIY	PPIPVGEIY	PPIPVGEIY	B35	
p24(122–130)	PPIPVGDIY	PPIPVGEIY	PPIPVGEIY	B35	
p24(131–139)	KRWIILG-NK	KRWIILGLNK	KRWIILGLNK	B27	
p24(131–140)	KRWIILLGLNK	KRWIIL-GLNK	KRWIIL-GLNK	B*27	
p24(131–140)	RRWIQLGLQK	KRWIILGLNK	KRWIILGLNK	B*2703	
p24(131–140)	KRWIILGGLNK	KRWIILG-LNK	KRWIILG-LNK	B*2705	
p24(131–140)	RRWIQLGLQK	KRWIILGLNK	KRWIILGLNK	B27	
p24(131–140)	KRWIIMGLNK	KRWIILGLNK	KRWIILGLNK	B27	
p24(131–140)	KRWIIMG-NK	KRWIILGLNK	KRWIILGLNK	B27	
p24(131–140)	KRWIIMGLNK	KRWIILGLNK	KRWIILGLNK	B27	
RT(118–127)	VPLDKDFRKY	VPLDEDFRKY	VPLDKDFRKY	B*3501	
RT(118–127)	VPLDKDFRKY	VPLDEDFRKY	VPLDKDFRKY	B35	
RT(175–183)	HPDIVIYQY	NPDIVIYQY	NPDIVIYQY	B*3501	
RT(175–183)	HPDIVIYQY	NPDIVIYQY	NPDIVIYQY	B35	
RT(175–183)	HPDIVIYQY	NPDIVIYQY	NPDIVIYQY	B35	
RT(175–183)	HPDIVIYQY	NPDIVIYQY	NPDIVIYQY	B35	
gp160(78–86)	DPNPQEVVL	DPNPQEVEL	DPNPQEVVL	B*3501	
gp160(78–86)	DPNPQEVVL	DPNPQEVEL	DPNPQEVVL	B35	
gp160(78–86)	DPNPQEVVL	DPNPQEVEL	DPNPQEVVL	B35, B51	
gp160(252–260)	RPIVSTQLL	RPVVSTQLL	RPVVSTQLL	B*3501	
gp160(252–260)	RPIVSTQLL	RPVVSTQLL	RPVVSTQLL	B35	
gp160(314–322)	GRAFVTIGK	GRAFYTTKN	GRAFYTTGE	B27	
gp160(606–614)	TAVPWNASW	TTVPWNASW	TAVPWNASW	B*3501	
gp160(606–614)	TAVPWNASW	TTVPWNASW	TAVPWNASW	B35	
gp160(704–712)	IVNRNRQGY	IVNRVRQGY	IVNRVRQGY	A*3002	
gp160(786–794)	GRRGWEALK	GRRGWEVLK	GRRGWEALK	B27	
gp160(786–795)	GRRGWEALKY	GRRGWEVLKY	GRRGWEALKY	B*2705	
gp160(786–795)	GRRGWEALKY	GRRGWEVLKY	GRRGWEALKY	B27	
gp160(794–802)	KYCWNLLQY	KYWWNLLQY	KYWWNLLQY	A*3002	

Nef(68–76)	FPVRPQVPL	FPVTPQVPL	FPVRPQVPL	B*3501
Nef(68–76)	FPVRPQVPL	FPVTPQVPL	FPVRPQVPL	B35
Nef(69–79)	RPQVPLRPMTY	TPQVPLRPMTY	RPQVPLRPMTY	B35
Nef(71–81)	RPQVPLRPMTY	TPQVPLRPMTY	RPQVPLRPMTY	B*3501
Nef(71–81)	RPQVPLRPMTY	TPQVPLRPMTY	RPQVPLRPMTY	B35
Nef(73–82)	SVPLRPMTYK	QVPLRPMTYK	QVPLRPMTYK	B35 or C4
Nef(135–143)	YPLTFGWCF	YPLTFGWCY	YPLTFGWCF	B35

Table 1: **p17**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(124–132)	p17(124–132 LAI)	NSSKVSQNY	HIV-1 or -2 infection	human(B*3501)	[Brander & Goulder(2001)]
	• Noted by Brander to be B*3501 epitope				
p17(124–132)	p17(124–132 LAI)	NSSKVSQNY	HIV-1 infection	human(B35)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes				

Table 2: **p24**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(122–130)	p24(260–268 LAI)	PPIPVGDIY	HIV-1 or -2 infection	human(B*3501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*3501 epitope				
p24(122–130)	p24(245–253 HIV-2)	NPVPVGNIY	HIV-1 infection	human(B*3501)	[Rowland-Jones (1995)]
p24(122–130)	p24(260–268 LAI)	PPIPVGDIY	HIV-1 or -2 infection	human(B35)	[Rowland-Jones (1995)]
	• De£ned as minimal peptide by titration curve, PPIPVGEIY and HIV-2 form NPVPVGNIY are also recognized				
p24(122–130)	p24()	PPIPVGDIY		human(B35)	[Rowland-Jones (1999)]
	• 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, and the B35 allele seems to be protective • HIV-2 version of this epitope is not conserved: NPVPVGNIY, but the CTLs are cross-reactive – one of £ve B35 CTL epitopes that are cross-reactive, see also [Rowland-Jones (1995)]				
p24(131–139)	p24(263–272)	KRWIILGNK	HIV-1 infection	human(B27)	[Durali (1998)]
	• 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: KRWIILGNK				
p24(131–140)	p24(263–272)	KRWIILLGLNK	HIV-1 infection	human(B*27)	[Huang (2000)]
	• The single cell ELISPOT assay was optimized and highly speci£c, and found to work well even after the primary cells had been frozen and thawed • Increases in gamma interferon producing cells were observed in response to anti-retroviral therapy using single cell IFN-gamma-production ELISPOT • In 3/3 HLA A*02, B*27 individuals, the dominant response in gag measured by both gamma IFN production and T cell lysis was to the B27 epitope, KRWIILLGLNK, not the A2 SLYNTVATL epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(131–140)	p24(260–269 HIV-2)	RRWIQLGLQK		human(B*2703)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*2703 epitope				
p24(131–140)	p24()	KRWIILGGLNK	HIV-1 infection	human(B*2705)	[Wilson (2000)]
	• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c 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 • Tetramers with peptide variants KRWIILGGLNK and KRWIIMGGLNK were used – CTL from most B27 donors recognize both variants, although one of the three subjects recognized only KRWIILGGLNK • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong 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			
p24(131–140)	p24(260–269 HIV-2)	RRWIQLGLQK		human(B27)	[Brander & Walker(1996)]
	• HIV-2, HLA-B*2703, S. Rowland-Jones, Pers. Comm.				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Klenerman (1994)]
	• Naturally occurring variant KRWIILGLNK may act as antagonist				
p24(131–140)	p24(263–272)	KRWIIMGNK	HIV-1 infection	human(B27)	[Nowak (1995)]
	• Longitudinal study of CTL response and immune escape – the form KRWIILGNK was also found, and both forms stimulate CTL				
p24(131–140)	p24(263–272)	KRWIIMGLNK	HIV-1 infection	human(B27)	[Goulder (1997b), Goulder (1997a)]
	• Six HLA-B27 donors studied make a strong response to this epitope • In 4/6 cases, this was the immunodominant or only CTL response • Two of the cases had an epitope switch to the form KKWIIMGLNK during a period of rapid decline to AIDS, following their asymptomatic period • The arginine to lysine switch is in an anchor residue, and results in immune escape due to severely diminished binding to the B27 molecule • [Goulder (1997a)] is a review of immune escape that summarizes this study in the context of CTL escape to £xation				

Table 3: **RT**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(118–127)	RT(273–282 SF2)	VPLDKDFRKY	HIV-1 infection	human(B*3501)	[Tomiyama (1997), Menendez-Arias (1998)]
	• A CTL clone responsive to this epitope was obtained • 4/7 B35-positive individuals had a CTL response to this epitope • A K to E substitution at position 5 abrogates speci£c lysis, and reduces binding to B*3501 • [Menendez-Arias (1998)], in a review, notes that a Glu to Lys (E to K) change abrogates CTL activity, but that both VPLDEDFRKY and VPLDKDFRKY can serve as HLA-B35 epitopes, so the change must alter T cell receptor binding – residues in this epitope may be important for polymerase activity				
RT(118–127)	()	VPLDKDFRKY	HIV-1 infection	human(B35)	[Kawana (1999)]
	• HLA B35 is associated with rapid disease progression • The sequences of 9 previously described HIV-1 B35 CTL epitopes were obtained in 10 HLA B35+ and 19 HLA B35- individuals • 3/9 CTL epitopes had substitutions that were more common in B35+ individuals than in B35- individuals – only one of these reduced the binding of the peptide to B35 and was shown to be an escape mutation • ––E—- was found in 8/10 of the B35+ individuals, and three of the B35- individuals – the D –> E substituted peptide had similar binding af£nity to B35 and was equally susceptible to a CTL clone				
RT(175–183)	RT(342–350 LAI)	HPDIVIYQY	HIV-1 infection	human(B*3501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*3501 epitope				
RT(175–183)	RT(342–350 LAI)	HPDIVIYQY	HIV-1 infection	human(B35)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes				
RT(175–183)	RT(329–337)	HPDIVIYQY	none	human(B35)	[Lalvani (1997)]
	• 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-speci£c 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				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(175–183)	Pol()	HPDIVIYQY		human(B35)	[Rowland-Jones (1999)]

- 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, and the B35 allele seems to be protective
- HIV-2 version of this epitope is not conserved: NPDVILIQY, but the CTLs are cross-reactive – one of £ve B35 CTL epitopes that are cross-reactive, see also [Rowland-Jones (1995)]

Table 4: **gp160**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(78–86)	gp120(77–85)	DPNPQEVVL	HIV-1 infection	human(B*3501)	[Ogg (1998)]
	• This epitope was included to illustrate the speci£city of HIV-tetrameric staining, in a cross-sectional study correlating HLA A*0201 CTL effector cells and low viral load				
gp160(78–86)	Env(77–85)	DPNPQEVVL	HIV-1 infection	human(B35)	[Dyer (1999)]
	• CTL speci£c responses were measured over a 1.3 to 1.5 year period in members of the Sydney Blood Bank Cohort (SBBC) who had been infected with a natural attenuated strain of HIV-1 which was Nef-defective • Some of these patients had prolonged high levels of CTL effector and memory cells despite low viral load				
gp160(78–86)	gp120(77–85 SF2)	DPNPQEVVL	HIV-1 infection	human(B35, B51)	[Shiga (1996)]
	• Binds HLA-B*3501 and B*5101 – binds and kills gp120-vaccinia virus infected cells carrying B35 or B51				
gp160(252–260)	gp120(255–263 SF2)	RPIVSTQLL	HIV-1 infection	human(B*3501)	[Tomiyama (1997)]
	• A CTL clone responsive to this epitope was obtained • Only 1/7 B35-positive individuals had a CTL response to this epitope • An I to V substitution at position 3 reduces speci£c lysis, but not binding to B*3501 • A Q to H substitution at position 7 abrogates speci£c lysis, but not binding to B*3501				
gp160(252–260)	gp120(255–263 SF2)	RPIVSTQLL	HIV-1 infection	human(B35)	[Shiga (1996)]
	• Binds HLA-B*3501				
gp160(314–322)	gp120(314–322)	GRAFVTIGK	no CTL shown	human(B27)	[Jardetzky (1991)]
	• Study of peptide binding to HLA-B27				
gp160(606–614)	gp41(605–615 LAI)	TAVPWNASW	gp160 vaccinia	human(B*3501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*3501 epitope				
gp160(606–614)	gp41(605–615 LAI)	TAVPWNASW	gp160 vaccinia	human(B35)	[Johnson (1994)]
	• Epitope for vaccine induced CD8+ clone				
gp160(704–712)	gp160(704–712 LAI)	IVNRNRQGY		human(A*3002)	[Brander & Goulder(2001), Goulder (2001)]
	• C. Brander notes this is an A*3002 epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(786–794)	gp41(791–799 LAI)	GRRGWEALK	HIV-1 infection	human(B27)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes • Also: J. Liebermann 1992 and pers. comm. J. Liebermann				
gp160(786–795)	gp41(791–800 LAI)	GRRGWEALKY	HIV infection	human(B*2705)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*2705 epitope				
gp160(786–795)	gp41(791–800 LAI)	GRRGWEALKY	HIV infection	human(B27)	[Lieberman(1998)]
	• Optimal peptide mapped by titration J. Lieberman, Pers. Comm.				
gp160(794–802)	gp160(794–802 LAI)	KYCWNLLQY		human(A*3002)	[Brander & Goulder(2001), Goulder (2001)]
	• C. Brander notes this is an A*3002 epitope				

Table 5: **Nef**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(68–76)	Nef(72–80 SF2)	FPVRPQVPL	HIV-1 infection	human(B*3501)	[Tomiyama (1997)]
	• A CTL clone responsive to this epitope was obtained • 3/7 B35-positive individuals had a CTL response to this epitope • An R to T substitution at position 4 abrogates speci£c lysis, but not binding to B*3501				
Nef(68–76)	Nef(72–80 SF2)	FPVRPQVPL	HIV-1 infection	human(B35)	[Shiga (1996)]
	• Binds HLA-B*3501				
Nef(69–79)	()	RPQVPLRPMTY	HIV-1 infection	human(B35)	[Kawana (1999)]
	• HLA B35 is associated with rapid disease progression • The sequences of 9 previously described HIV-1 B35 CTL epitopes were obtained in 10 HLA B35+ and 19 HLA B35- individuals • 3/9 CTL epitopes had substitutions that were more common in B35+ individuals than in B35- individuals – only one of these reduced the binding of the peptide to B35 and was shown to be an escape mutation • ————-F was found in 9/10 of the B35+ individuals, none of the B35- individuals – the Y –> F substituted peptide had a similar binding af£nity with B35 and was recognized by a CTL clone equally with wildtype				
Nef(71–81)	Nef(75–85 SF2)	RPQVPLRPMTY	HIV-1 infection	human(B*3501)	[Tomiyama (1997)]
	• A CTL clone responsive to this epitope was obtained • 4/7 B35-positive individuals had a strong CTL response to this epitope • An R to T substitution at position 1 abrogates speci£c lysis, but not binding to B*3501 • An R to H substitution at position 7 did not alter reactivity				
Nef(71–81)	Nef(75–85 SF2)	RPQVPLRPMTY	HIV-1 infection	human(B35)	[Shiga (1996)]
	• Binds HLA-B*3501				
Nef(73–82)	Nef(73–82 LAI)	SVPLRPMTYK	HIV-1 infection	human(B35 or C4)	[Buseyne (1993)]
	• 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 £ve 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(135–143)	Nef(139–147 SF2)	YPLTFGWCF	HIV-1 infection	human(B35)	[Shiga (1996)]
	• Binds HLA-B*3501				

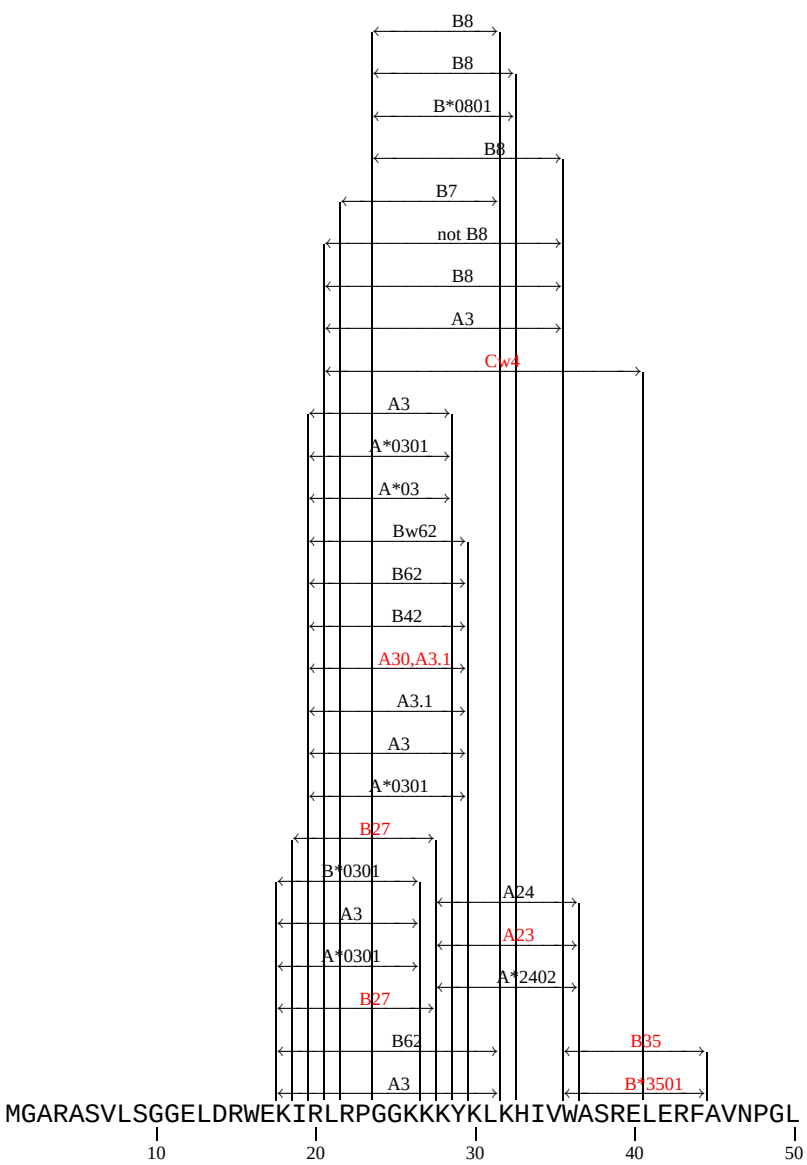
Table 6: **All De£ned Epitopes within the 20mer, regardless of HLA type**

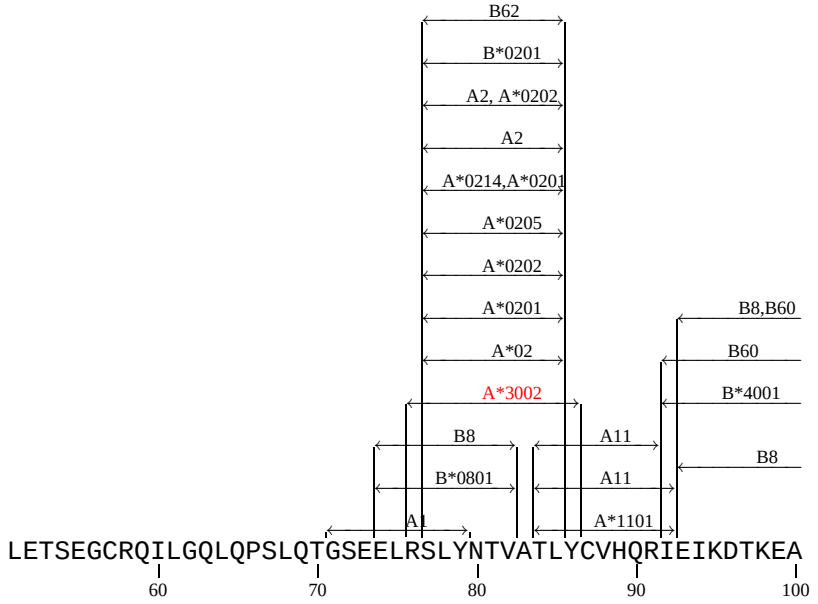
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(132–147)	Nef(132–147 BRU)	GVRYPLTFGWCYKLVP	HIV-1 infection	human(A1, B8)	[Hadida (1992)]
	• HIV-1 speci£c CTLs detected in lymphoid organs				
Nef(132–147)	Nef(132–147 BRU)	GVRYPLTFGWCYKLVP	HIV-1 infection	human(B18)	[Culmann (1991)]
	• Nef CTL clones from HIV+ donors				
Nef(133–148)	Nef(133–148 LAI)	VRYPLTFGWCYKLVPV		human(B57)	[Brander & Walker(1996)]
	• P. Goulder, pers. comm.				
Nef(134–141)	Nef(138–147 LAI)	RYPLTFGW	HIV-1 infection	human(A*2402)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*2402 epitope				
Nef(134–141)	Nef(134–141 LAI)	RYPLTFGW		human(B27)	[Culmann(1998)]
	• Optimal peptide de£ned by titration				
Nef(134–143)	Nef(138–147 SF2)	RYPLTFGWCF	HIV-1 infection	human(A*2402)	[Ikeda-Moore (1997)]
	• De£ned using reverse immunogenetics – 59 HLA-A*2402 binding peptides were predicted by searching for A*2402 anchors in HIV proteins (Tyr at 2, and Phe, Leu or Ile at the C term) – 53 of the 59 peptides bound A*2402 • This peptide induced CTL in 3/4 HIV-1+ people tested • RYPLTFGWCF bound to A*2402 strongly, the epitope can be processed in a vaccinia construct and presented – two speci£c CTL clones were obtained				
Nef(134–144)	Nef(134–144 LAI)	RYPLTFGWCYK	HIV-1 infection	human(B18)	[Couillin (1994), Goulder (1997a)]
	• 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				
Nef(135–143)	Nef(135–143 LAI)	YPLTFGWCY	HIV-1 exposure	human(B*1801)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*1801 epitope				

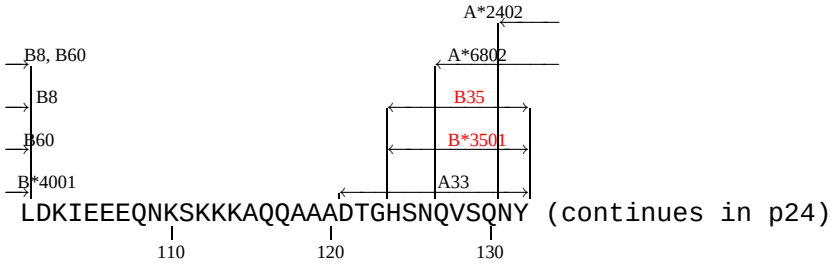
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(135–143)	Nef()	YPLTFGWCF	HIV-1 exposed seronegative	human(B18)	[Kaul (2000)]
	• 11/16 heavily HIV exposed but persistently seronegative sex-workers in Nairobi had HIV-speci£c CD8 gamma-IFN responses in the cervix – systemic 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(135–143)	Nef(135–143 LAI)	YPLTFGWCY	HIV-1 exposure	human(B18)	[Culmann (1991), Culmann-Penciolelli (1994)]
	• Nef CTL clones from HIV+ donors				
Nef(135–143)	Nef(139–147 SF2)	YPLTFGWCF	HIV-1 infection	human(B35)	[Shiga (1996)]
	• Binds HLA-B*3501				
Nef(135–143)	Nef()	YPLTFGWCY	HIV-1 exposure	human(B49)	[Rowland-Jones (1998a)]
	• A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-de£ned 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 subtype consensus is identical to the B clade epitope • The D subtype consensus is YPLTFGWCf				
Nef(135–143)	Nef()	YLPTFGWCY	HIV-1 exposure	human(B49)	[Rowland-Jones (1998b)]
	• HIV-speci£c 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 and B clade viruses • The Clade D version of the epitope, YPLTFGWCF, was preferentially recognized by CTL				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(136–145)	Nef(136–145)	PLTFGWCYKL	*in vitro* stimulation	human(A*0201)	[Wilson (1999)]
	• Dendritic cells are the most potent for priming T cell responses – DCs can stimulate autologous CTL responses from T cells cultured from HIV negative donors • Th1-biasing cytokines IL-12 or IFN alpha enhance CTL responses *in vitro* whether the epitope is delivered by pulsing from peptide, or expressed from within • B7 and A2 Nef epitopes were studied and the relative binding af£nity of A2 epitopes for A2 was: PLTFGWCYKL greater than VLEWRFDSRL which was much greater than AFHHVAREL • Noted in Brander *et al*., 1999 this database, to be A*0201				
Nef(136–145)	Nef(136–145 LAI)	PLTFGWCYKL	Nef(180-189)	human(A*0201)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*0201 epitope				
Nef(136–145)	Nef(136–145)	PLTFGWCFKL	HIV-1 infection	human(A2)	[Durali (1998)]
	• 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(136–145)	Nef(157–166)	PLTFGWCFKL	HIV-1 infection (human) or HIV A2-polyepitope (polytope) DNA vaccine with vaccinia boost (rVV.HIV.pt) (mouse)	human(A2)	[Woodberry (1999)]
	• A polyepitope vaccine was generated in a vaccinia construct that contiguously encoded seven epitopes, all presented by HLA A-2 • HHD mice have a transgene of HLA A2 linked to the transmembrane and cytotoxic domains of H-2D^{d} – this transgene is the only MHC molecule expressed in the mice • CTL responses to Gag (77-85) SLYNTVATL, Pol (476-484) ILKEPVHGV, gp120 (120-128) KLTPLCVTL, and Nef (190-198) AFHHVAREL were observed in HIV polytope HHD-vaccinated mice, and these responses were enhanced with vaccinia boost • No CTL immune responses were generated against HLA A2-restricted HIV epitopes Nef 157-166 (PLTFGWCYKL), Pol 346-354 (VIYQYMDDL), and Nef 180-189 (VLEWRFDSRL) • Sixteen HLA A2+ patients were tested for their ability to make CTL responses by peptide restimulation in culture with the epitopes selected for inclusion in the polytope – one individual recognized all seven of these epitopes; 7 patients had CTL cultures able to recognize at least one of the epitopes, and 6 of those 7 recognized more than one epitope, but they were not able to test all peptides for all patients; many patients only had three peptides tested • PLTFGWCFKL was recognized by 1 of the HLA-A2 patients				

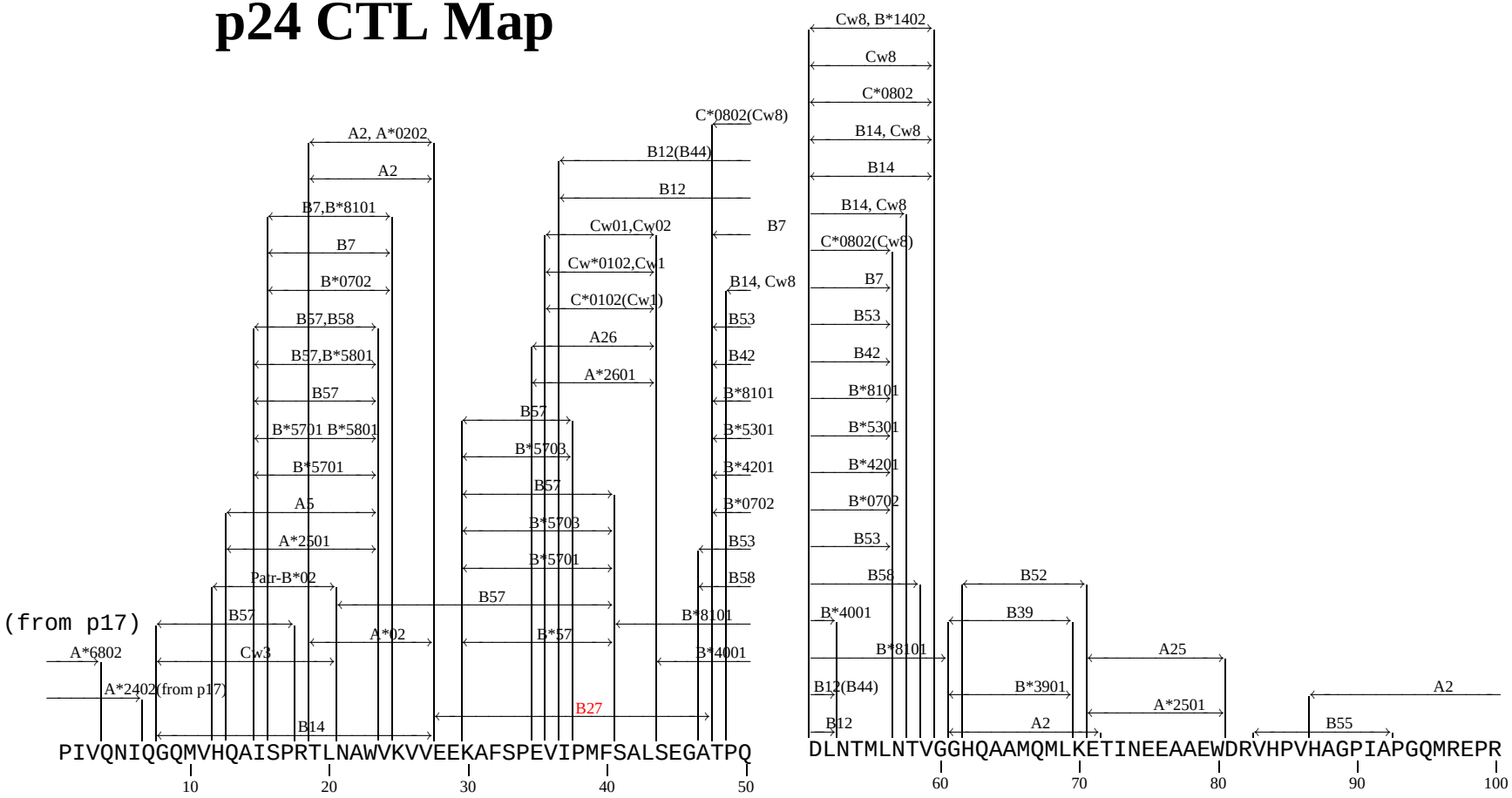
p17 CTL Map

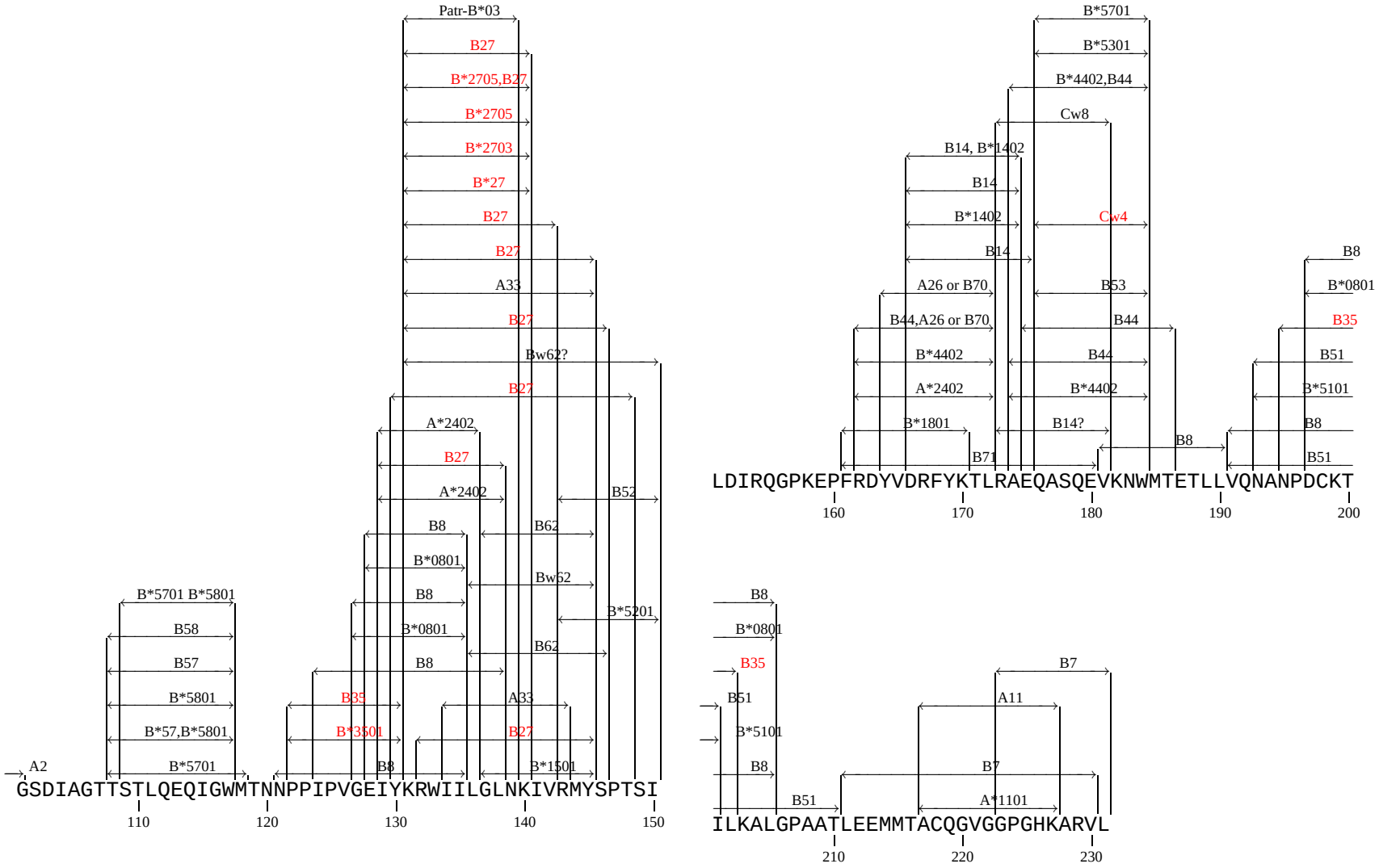

B8
B8
B*0801
B8
B7
not B8
B8
A3
Cw4
A3
A*0301
A*03
Bw62
B62
B42
A30,A3.1
A3.1
A3
A*0301
B27
B*0301
A3
A*0301
B27
B62
A3
A24
A23
A*2402
B35
B*3501
MGARASVLSGGELDRWEKIRLRPGGKKKYKLKHIVWASRELERFAVNPGL
10
20
30
40
50


B62
B*0201
A2, A*0202
A2
A*0214,A*0201
A*0205
A*0202
A*0201
A*02
A*3002
B8
B*0801
A1
A11
A11
A*1101
B8,B60
B60
B*4001
B8
LETSEGCRQILGQLQPSLQTGSEELRSLYNTVATLYCVHQRIEIKDTKEA
60
70
80
90
100

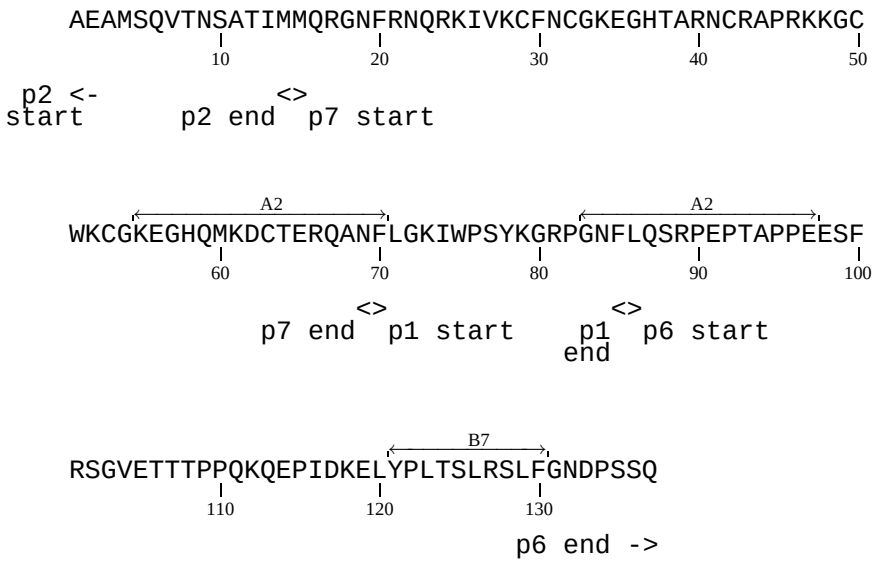

A*2402
A*6802
B35
B*3501
A33
B8, B60
B8
B60
B*4001
LDKIEEEQNKSKKKAQQAAADTGHSNQVSQNY (continues in p24)
110
120
130

p24 CTL Map

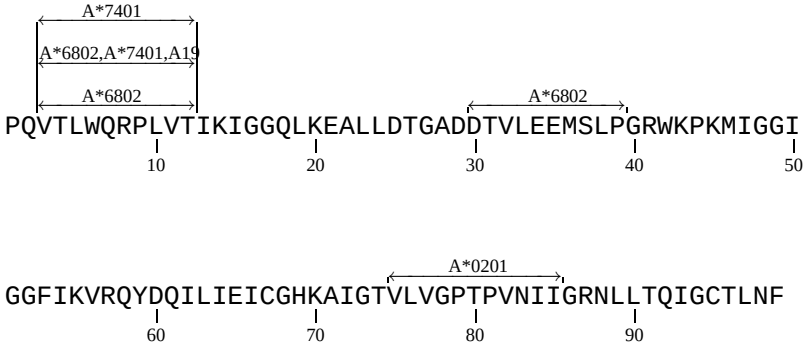

(from p17)
A*6802
A*2402(from p17)
A2, A*0202
A2
B7,B*8101
B7
B*0702
B57,B58
B57,B*5801
B57
B*5701 B*5801
B*5701
A5
A*2501
Pair-B*02
B57
A*02
Cw3
B14
C*0802(Cw8)
B12(B44)
B12
Cw01,Cw02
Cw*0102,Cw1
C*0102(Cw1)
A26
A*2601
B57
B*5703
B57
B*5703
B*5701
B57
B*57
B7
B14, Cw8
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*8101
B*4001
B27
PIVQNIQGQMVHQAISPRTLNAWVKVVEEKAFSPEVIPMFSALSEGATPQ
10
20
30
40
50
Cw8, B*1402
Cw8
C*0802
B14, Cw8
B14
B14, Cw8
C*0802(Cw8)
B7
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*4001
B*8101
B12(B44)
B12
B52
B39
A25
B*3901
A*2501
A2
A2
B55
DLNTMLNTVGGHQAAMQMLKETINEEAAEWDRVHPVHAGPIAPGQMREPR
60
70
80
90
100


Patr-B*03
B27
B*2705,B27
B*2705
B*2703
B*27
B27
B27
A33
B27
Bw62?
B27
A*2402
B27
A*2402
B52
B8
B62
B*0801
Bw62
B*5701 B*5801
B8
B*5201
B58
B*0801
B62
B57
B8
B*5801
B35
A33
B*57,B*5801
B*3501
B27
A2
B*5701
B8
B*1501
GSDIAGTTSTLQEQIGWMTNNPPIPVGEIYKRWIILGLNKIVRMYSPTSI
110
120
130
140
150
B*5701
B*5301
B*4402,B44
Cw8
B14, B*1402
B14
B*1402
Cw4
B14
A26 or B70
B53
B44,A26 or B70
B44
B*4402
B44
A*2402
B*4402
B*1801
B14?
B71
B8
B8
B*0801
B35
B51
B*5101
B8
B51
LDIRQGPKEPFRDYVDRFYKTLRAEQASQEVKNWMTETLLVQNANPDCKT
160
170
180
190
200
B8
B*0801
B35
B51
B*5101
B8
B51
B7
A11
B7
A*1101
ILKALGPAATLEEMMTACQGVGGPGHKARVL
210
220
230

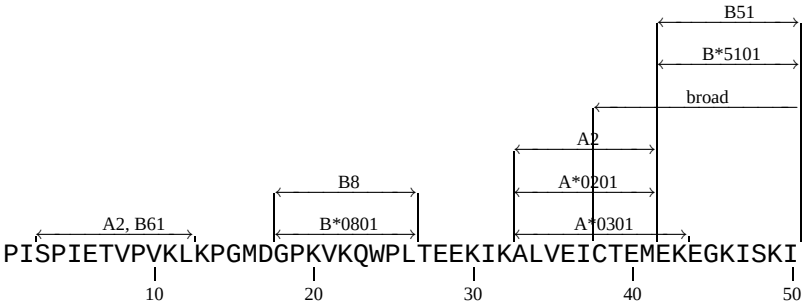
p2p7p1p6 CTL Map



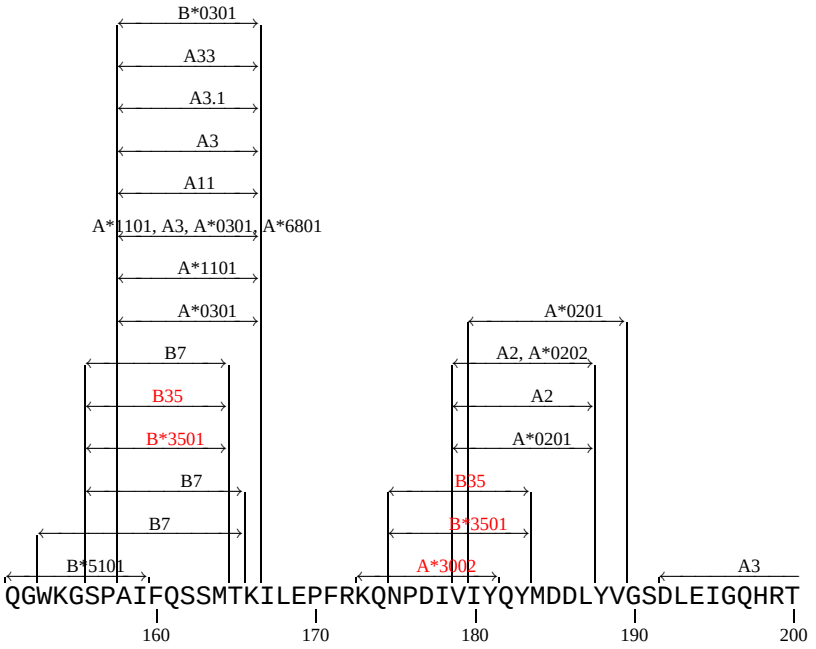
Protease CTL Map

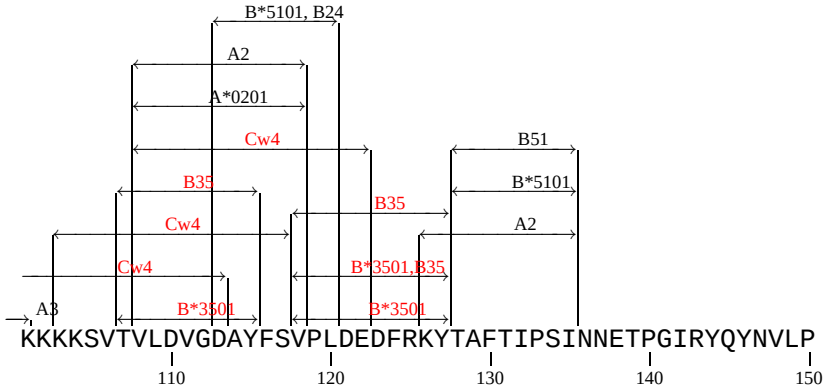


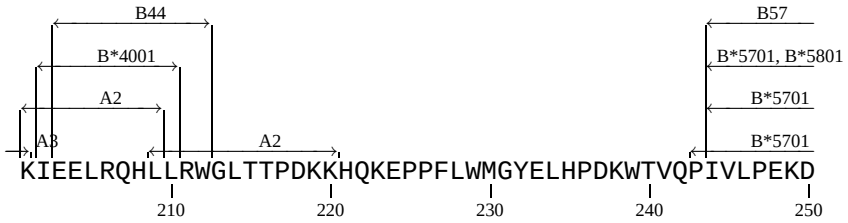
RT CTL Map

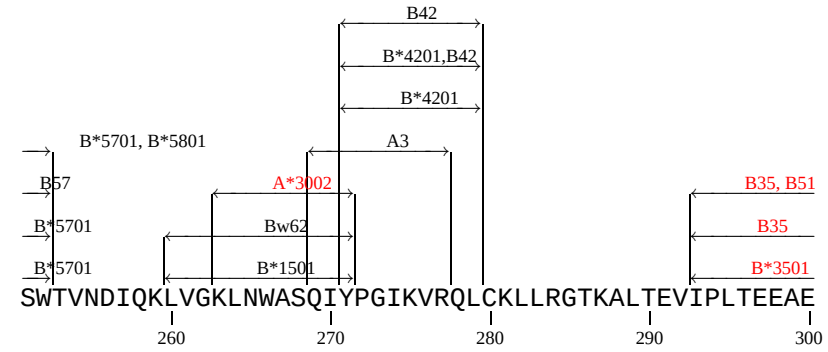

B51
B*5101
broad
A2
B8
A*0201
A2, B61
B*0801
A*0301
PISPIETVPVKLKPGMDGPKVKQWPLTEEKIKALVEICTEMEKEGKISKI
10
20
30
40
50

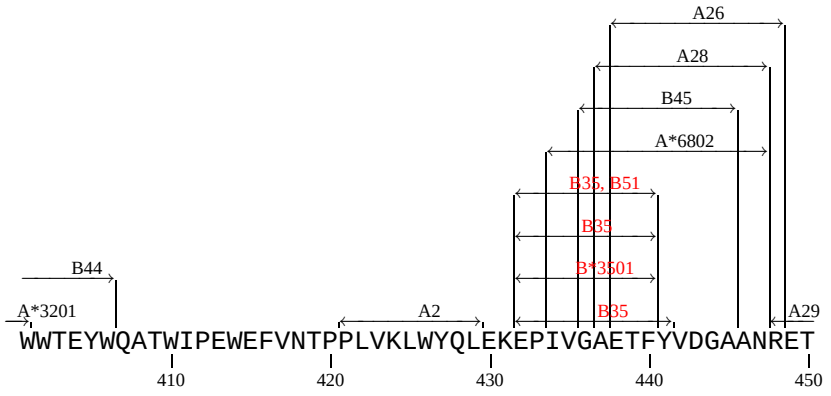

Cw4
broad
A3
GPENPYNTPVFAIKKKDSTKWRKLVDFRELNKRTQDFWEVQLGIPHPAGL
60
70
80
90
100


B*0301
A33
A3.1
A3
A11
A*1101, A3, A*0301, A*6801
A*1101
A*0301
B7
B35
B*3501
B7
B7
B*5101
A*0201
A2, A*0202
A2
A*0201
B35
B*3501
A*3002
A3
QGWKGSPAIFQSSMTKILEPFRKQNPDIVIYQYMDDLYVGSDLEIGQHRT
160
170
180
190
200

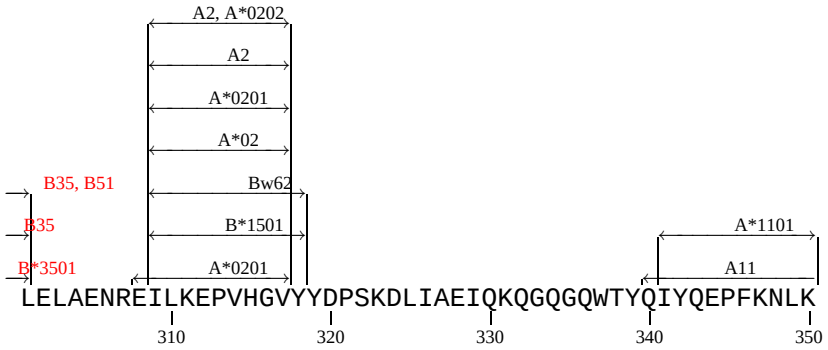

B*5101, B24
A2
A*0201
Cw4
B51
B35
B*5101
B35
Cw4
A2
Cw4
B*3501,B35
A3
B*3501
B*3501
KKKKSVTVLDVGDAYFSVPLDEDFRKYTAFTIPSINNETPGIRYQYNVLP
110
120
130
140
150

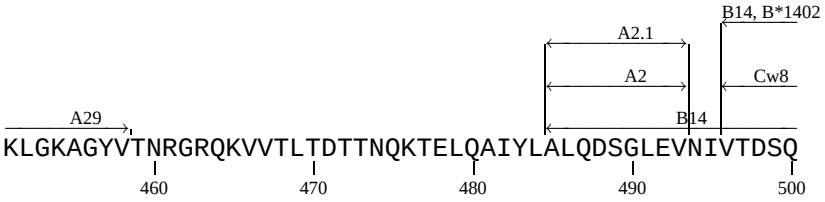

B44
B57
B*4001
B*5701, B*5801
A2
B*5701
A3
A2
B*5701
KIEELRQHLLRWGLTTPDKKHQKEPPFLWMGYELHPDKWTVQPIVLPEKD
210
220
230
240
250

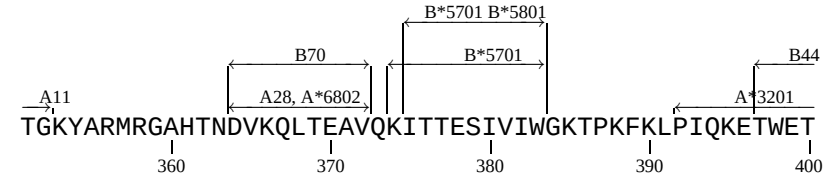


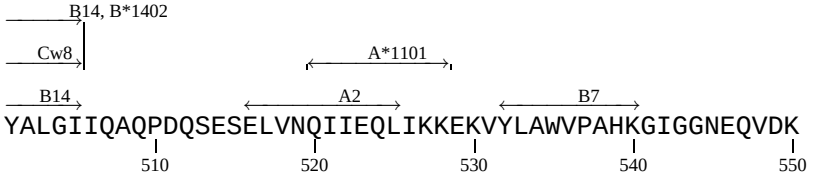


p15 RNase start <-





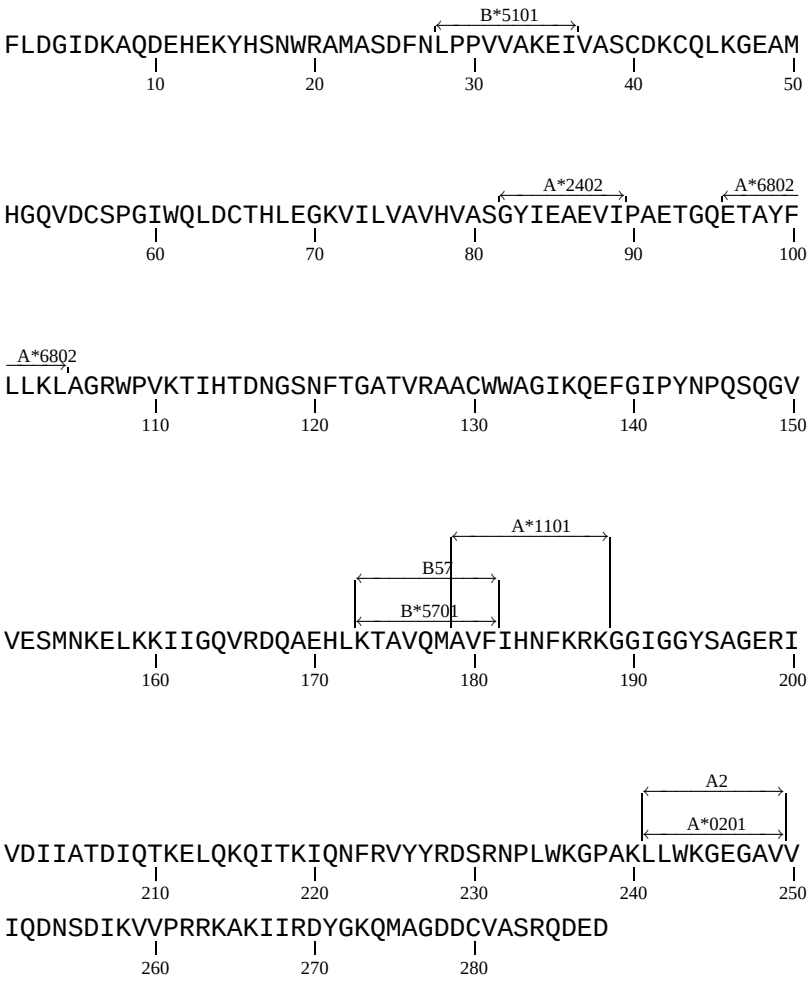




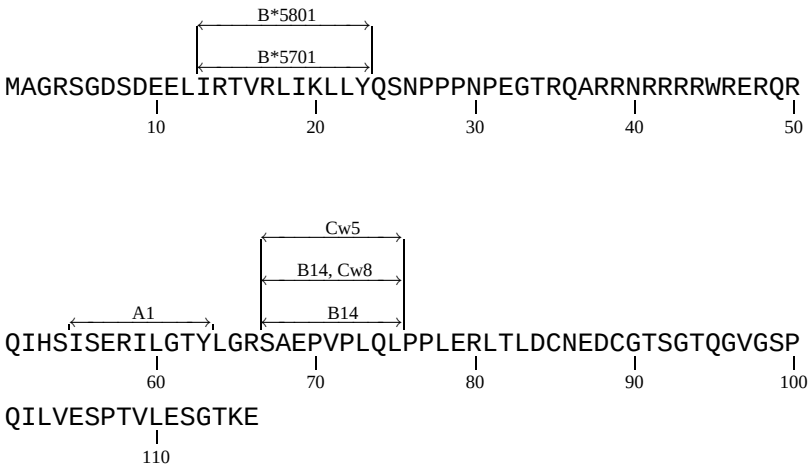
LVSAGIRKVL

-> p15 RNase end

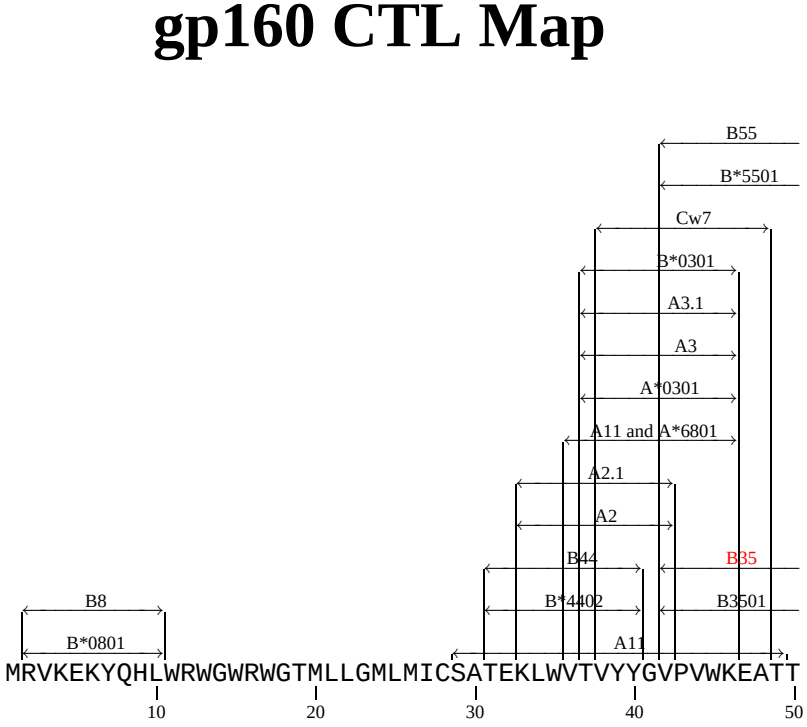
Integrase CTL Map



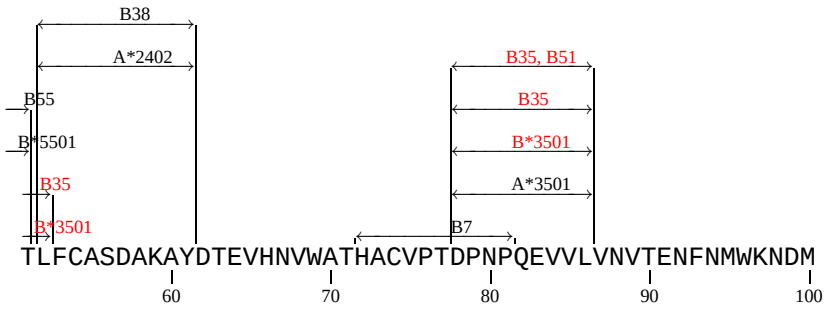
Rev CTL Map

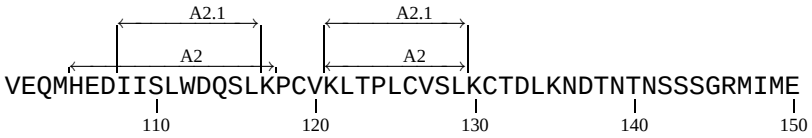


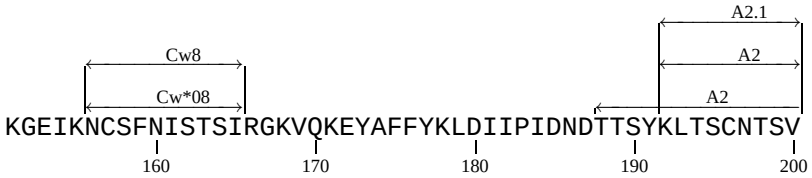
gp160 CTL Map

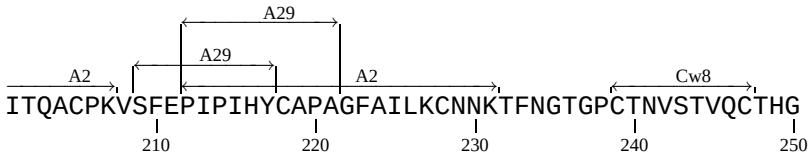


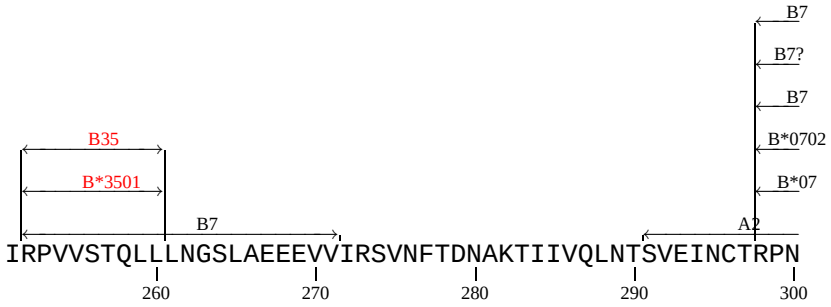
<- gp120 start

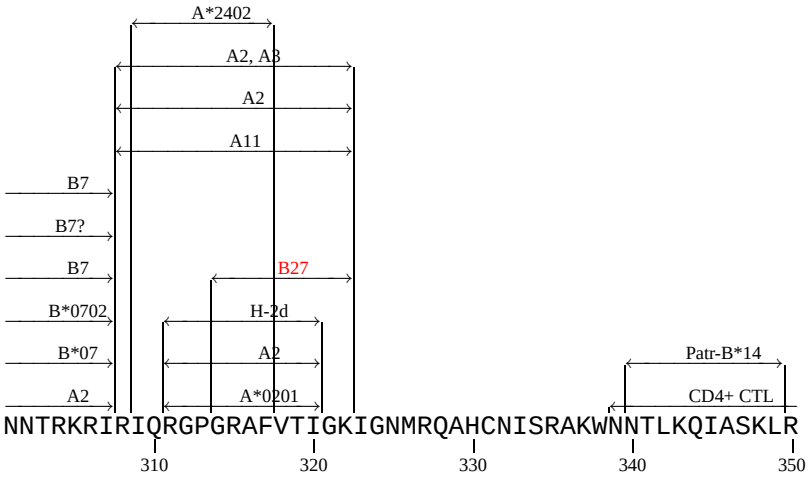


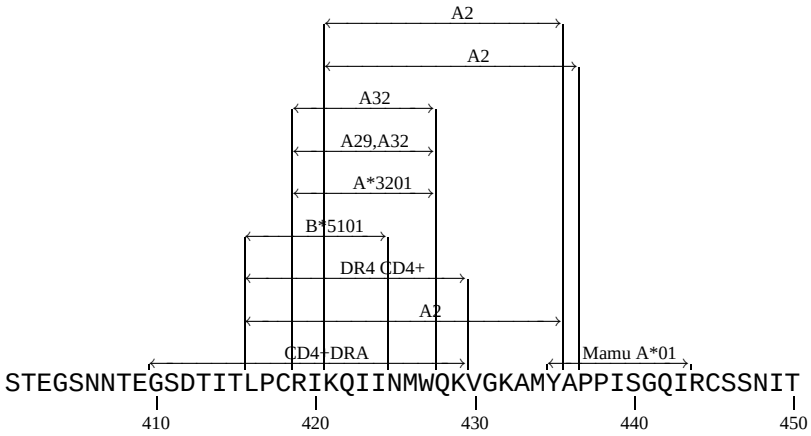


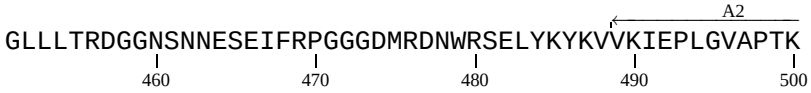




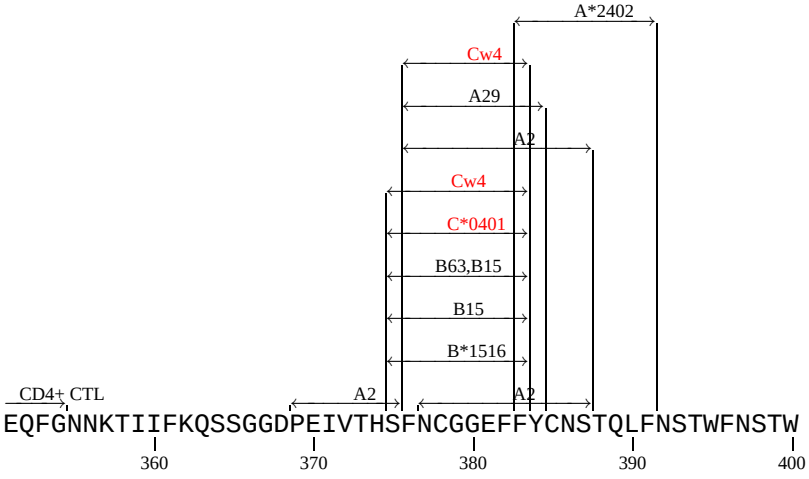


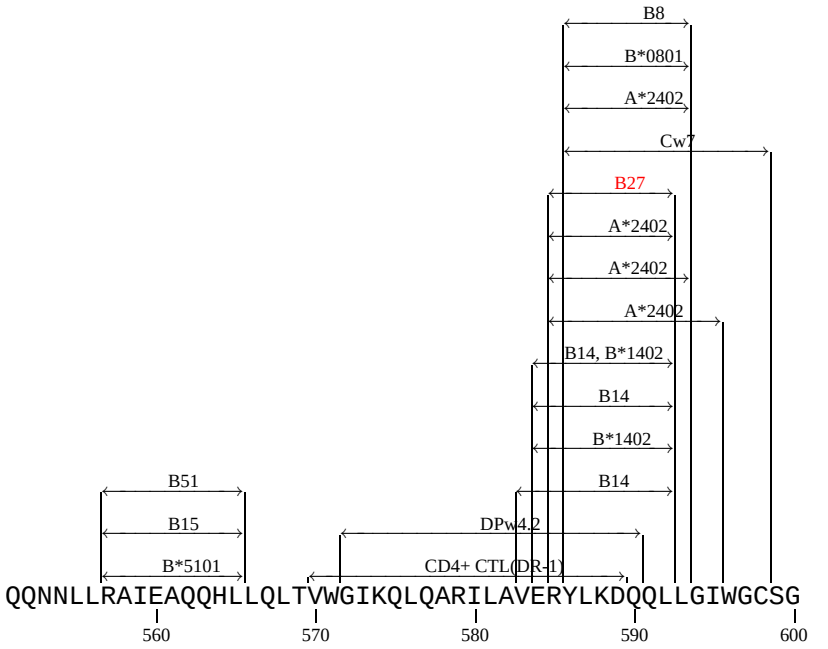


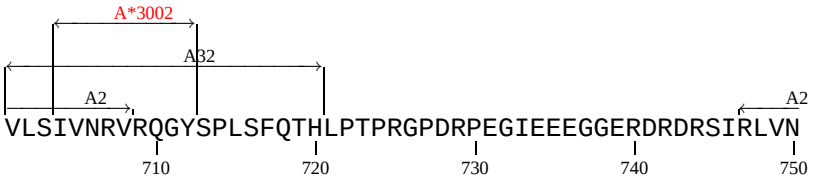


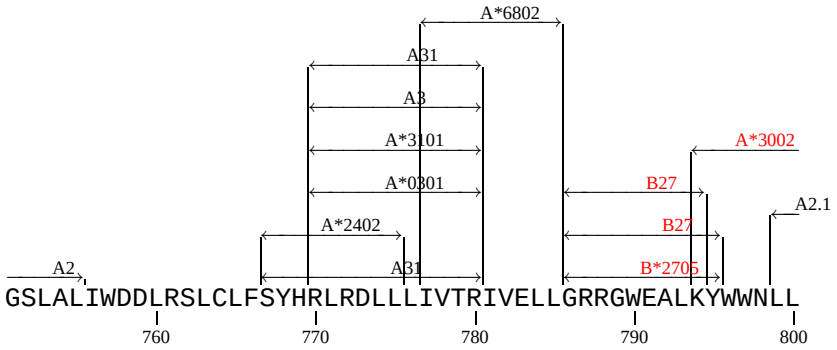


gp120 end <> gp41 start

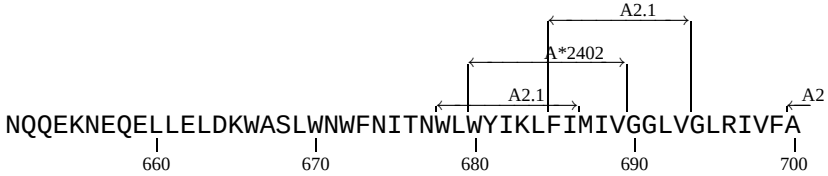


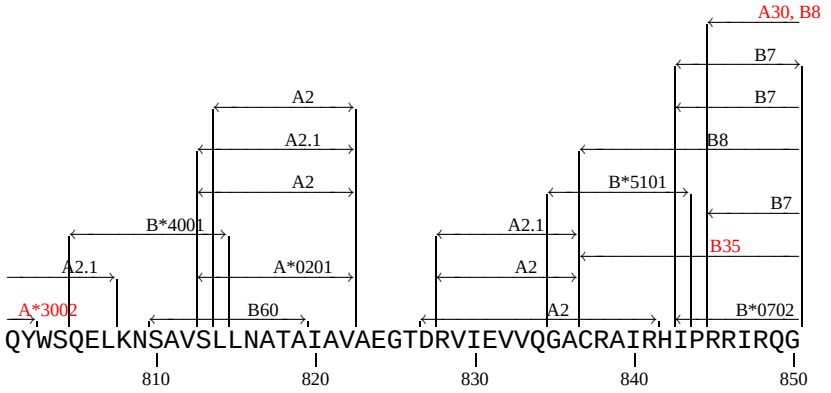

B8
B*0801
A*2402
Cw7
B27
A*2402
A*2402
A*2402
B14, B*1402
B14
B*1402
B14
B51
B15
B*5101
DPw4.2
CD4+ CTL(DR-1)
QQNNLLRAIEAQQHLLQLTVWGIKQLQARILAVERYLKDQQLLGIWGCSG
560
570
580
590
600

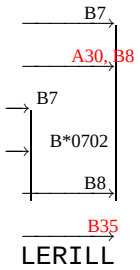

A*3002
A32
A2
A2
VLSIVNRVRQGYSPLSFQTHLPTPRGPDRPEGIEEEGGERDRDRSIRLVN
710
720
730
740
750


A*6802
A31
A3
A*3101
A*0301
A*2402
A2
A31
A*3002
B27
B27
B*2705
A2.1
GSLALIWDDLRSLCLFSYHRLRDLLLIVTRIVELLGRRGWEALKYWWNLL
760
770
780
790
800


B35
B*3501
KLICTTAVPWNASWSNKSLEQIWNHTTWMEWDREINNYTSLIHSLIEESQ
610
620
630
640
650


A2.1
A*2402
A2.1
A2
NQQEKNEQELLELDKWASLWNWFNITNWLWYIKLFIMIVGGLVGLRIVFA
660
670
680
690
700

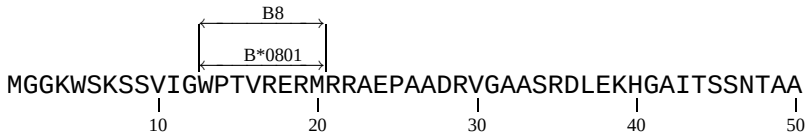

A30, B8
B7
B7
A2
A2.1
B8
A2
B*5101
B*4001
A2.1
B7
A2.1
B35
A*0201
A2
A*3002
B60
A2
B*0702
QYWSQELKNSAVSLLNATAIAVAEGTDRVIEVVQGACRAIRHIPRRIRQG
810
820
830
840
850

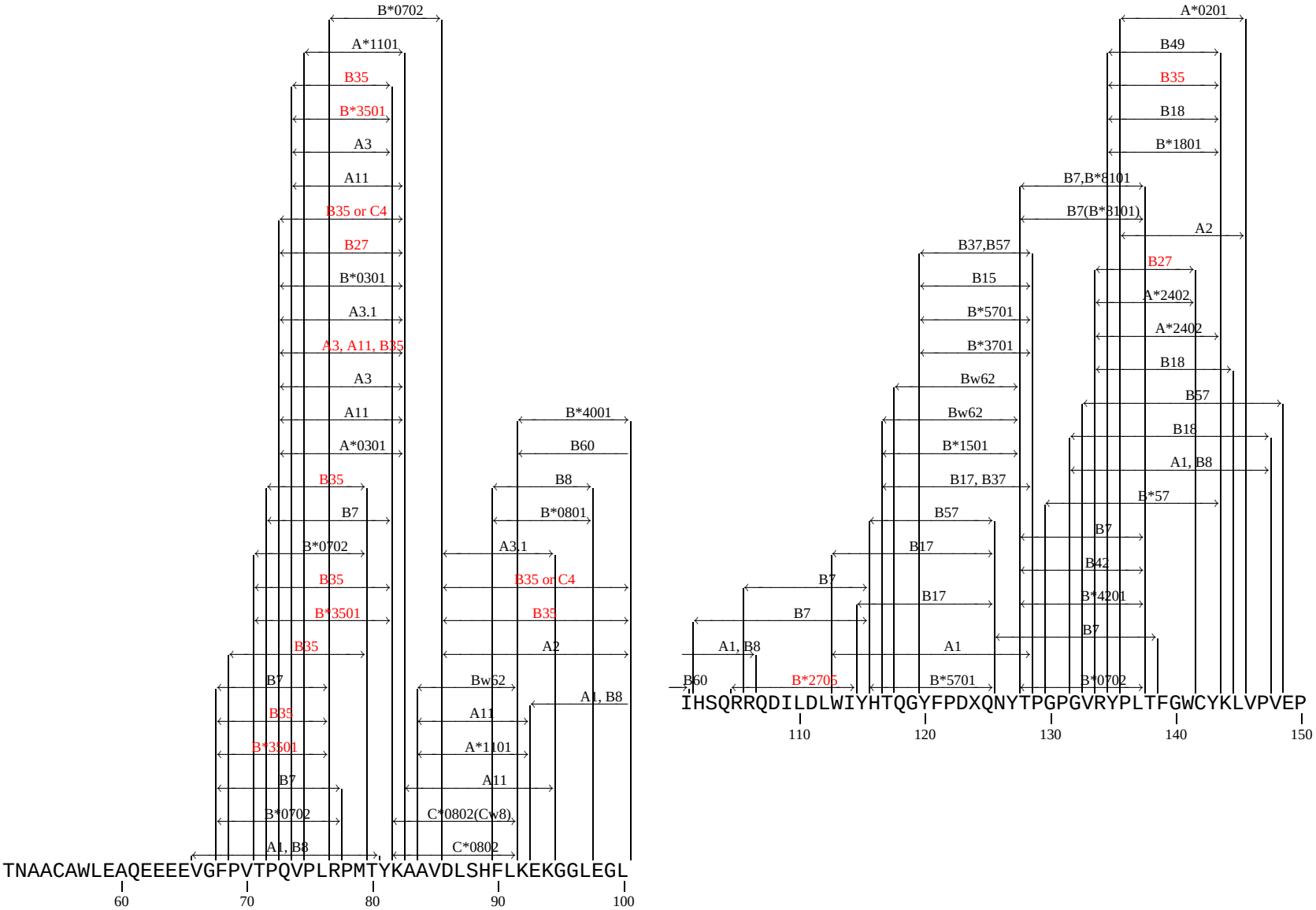


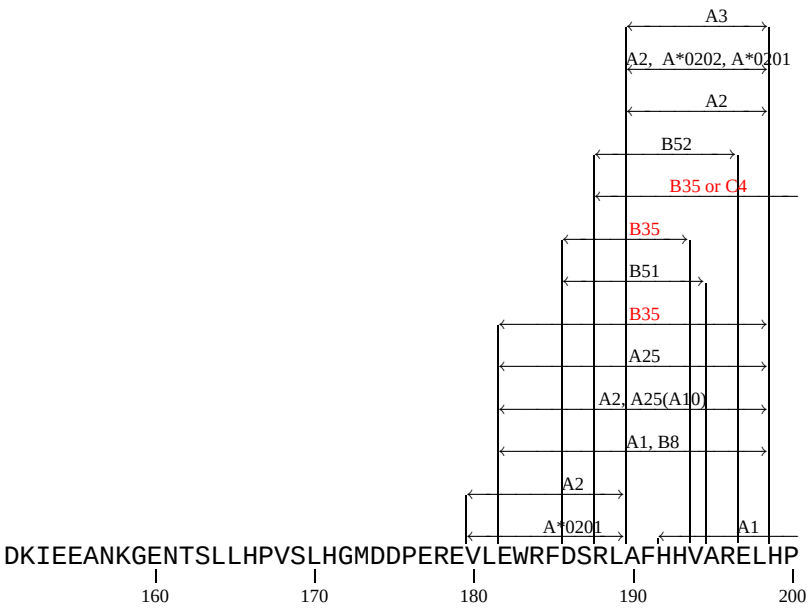
LERILL

-> gp41 end

Nef CTL Map




B*0702
A*1101
B35
B*3501
A3
A11
B35 or C4
B27
B*0301
A3.1
A3, A11, B35
A3
A11
A*0301
B35
B7
B*0702
B35
B*3501
B35
B7
B*0702
A1, B8
B*4001
B60
B8
B*0801
A3.1
B35 or C4
B35
A2
Bw62
A1, B8
A11
A*1101
A11
C*0802(Cw8)
C*0802
TNAACAWLEAQEEEEVGFPVTPQVPLRPMTYKAAVDLSHFLKEKGGLEGL
60
70
80
90
100
A*0201
B49
B35
B18
B*1801
B7,B*8101
B7(B*8101)
A2
B37,B57
B27
B15
A*2402
B*5701
A*2402
B*3701
B18
Bw62
B57
Bw62
B18
B*1501
A1, B8
B17, B37
B*57
B57
B7
B17
B42
B7
B17
B*4201
B7
B7
A1, B8
A1
B60
B*2705
B*5701
B*0702
IHSQRRQDILDLWIYHTQGYFPDXQNYTPGPGVRYPLTFGWCYKLVPVEP
110
120
130
140
150



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variants studied could serve as antagonists when they were processed from recombinant vaccinia, replicated HIV, or when they were synthetic peptides. Both agonist and antagonist sequences were found in the study subjects from whom the CTL clones were derived.

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[McMichael & Walker(1994)] A. J. McMichael & B. D. Walker. Cytotoxic T lymphocytes epitopes: implications for HIV vaccine. *AIDS* **8S**:S155–S173, 1994. Notes: Comprehensive review summarizing CTL epitopes that have known HLA type and are £ne mapped to indicate epitope boundaries. Anchor residues are indicated when known for different HLA restricted epitopes. Includes a summary of the published literature, as well as much work that was in press or submitted for publication.

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