EKASFPEVIPMFSALSEGAT

QUERY	EKASFPEVIPMFSALSEGAT
CONSENSUS_A	---fs----------
A.KE.Q23-CXC-CG	---FS----------
A.SE.SE6594	--GFN----------
A.SE.SE7253	---FS-----V---------
A.SE.SE7535	---FS----------
A.SE.SE8131	-R-FS----------
A.SE.SE8538	--GFN----------
A.SE.SE8891	--GFS----------
A.UG.92UG037	---LS----------
A.UG.U455	D--FS----------
CONSENSUS_B	---FS----------
B.AU.AF128998	---FS----------
B.-.NL43E9	---FS----------
B.AU.MBC18	---FS----------
B.AU.MBC200	---FS----------
B.AU.MBC925	---FS----------
B.AU.MBCC54	---FS----------
B.AU.MBCC98	---FS----------
B.AU.MBCD36	---FS-------T-------
B.CN.RL42	---FS----------
B.DE.D31	---FS----------
B.DE.HAN	---FS----------
B.ES.89SP061	---FS----------
B.FR.HXB2	---FS----------
B.GA.OYI	---FS----------A----
B.GB.CAM1	---FS----------
B.GB.MANC	---FS----------I
B.JP.JH31	---FS----------
B.NL.3202A21	---FS----------
B.TW.LM49	---FS----------
B.US.85WCIPR54	---FS----------
B.US.AD8	---FS----------
B.US.BC	---FS----------
B.US.DH123	---FS----------
B.US.JRCSF	---FS----------
B.US.JRFL	---FS----------
B.US.MNCG	---FS----------
B.US.NC7	---FS----------
B.US.NY5CG	---FS----------
B.US.P896	---FS----------
B.US.RF	---FS----------
B.US.SF2	---FS----------
B.US.WC001	---FS----------
B.US.WEAU160	---FS----------
B.US.WR27	---FS----------
B.US.YU2	---FS----------
CONSENSUS_C	---FS-------T-------
C.BR.92BR025	---FS-------T-------
C.BW.96BW01B22	---FS-------T-------
C.BW.96BW0402	---FS-------T-------
C.BW.96BW0502	---FS-------T-------
C.BW.96BW1104	---FS-------T-------
C.BW.96BW1210	---FS--I----T-------
C.BW.96BW15B03	---FS-------T-------
C.BW.96BW1626	---FS-------T-------
C.BW.96BW17A09	---FS-------T-------
C.ET.ETH2220	---FS-------T-------
C.IN.93IN904	---FS-------T-------
C.IN.93IN905	---FS-------T-------
C.IN.93IN999	---FS-------T-------
C.IN.94IN11246	---FS-------T-------
C.IN.95IN21068	---FS-------T-------
CONSENSUS_D	---Fs----------
D.CD.84ZR085	---FN----------
D.CD.ELI	---FS----------
D.CD.NDK	---FS----------
D.CD.Z2Z6	---FS----------
D.UG.94UG1141	---FN----------
CONSENSUS_F	---FS----------
F.BR.BZ162	---FS----------
F.CD.VI174	---FS----------
F.RW.VI69	---FS----------
CONSENSUS_F1	---FS----------
F1.BE.VI850	---FS----------
F1.BR.93BR020.1	---FS----------
F1.FI.FIN9363	---FS----------
F1.FR.MP411	---FS----------
CONSENSUS_F2	---FS----------
F2.CM.MP255	---FS----------
F2.CM.MP257	---FS----------
CONSENSUS_G	---FS----------
G.BE.DRCBL	---FS-------T-------
G.FI.HH8793	---FS----------
G.NG.92NG083	---FS----------
G.SE.SE6165	---FS----------
CONSENSUS_H	---FS----------
H.BE.VI991	---FS----------
H.BE.VI997	---FS----------
H.CF.90CF056	---FS----------
CONSENSUS_J	---FS----------
J.SE.SE9173	---FS----------
J.SE.SE9280	---FS----------
CONSENSUS_K	---FS----------
K.BE.VI325	---FS----------AD---
K.CD.EQTB11C	---FS----------
K.CM.MP535	---FS-------T-------
N.CM.YBF30	---FS-------M-------
CONSENSUS_O	---FN--I----M------?
O.CM.ANT70C	---FN--I----M------I
O.CM.MVP5180	---FN--I----M------V
CRF01-AE.CF.90CF40	--GFN----------
CRF01-AE.TH.93TH25	--GFN----------
CRF01-AE.TH.CM240	--GFN----------
CRF01-AE.TH.TH022	--GFN----------
CRF01-AE.TH.TH047	--GFS----------
CRF02_AG.FR.DJ263	---FS-------T-------
CRF02_AG.FR.DJ264	---FS-------T-------
CRF02_AG.NG.IBNG	--GFS----------
CRF03_AB.RU.KAL15	---FS----------
CRF04_cpx.CY.94CY0	---FS----------
CRF04_cpx.GR.97PVC	---FS----------
CRF04_cpx.GR.97PVM	--GFS----------
AC.ET.E3099G	---FS----------
AC.IN.21301	---FS--I----T-------
AC.RW.92RW009	---FSQ------T-------
AC.SE.SE9488	D--FS-------T-------
AC.ZM.ZAM174-21	---FS-------T-------
AC.ZM.ZAM184	---FS----------
AC.ZM.ZAM716-17	---FS-------T-------
ACD.SE.SE8603	---FS----------
AD.SE.SE6954	---FS----------A
AD.SE.SE7108	---FS----------
ADHU.NO.NOGIL3	---FS-----------D---
ADU.CD.MAL	---FS----------
AG.NG.G3	--NFS-------T-------
AG.SE.SE7812	---FS----------
AGHU.GA.VI354	--GFS----------
AGJ.AU.BFP90	D--FS-------T-------
AGJ.ML.95ML8	---FS----------
AGU.CD.Z321	--NFS----------
BF.BR.93BR029.4	---FS----------
DF.CD.VI961	---FS-------T-------
U.CD.VI1126	---FS-------T-------
CONSENSUS_CPZ	---Fn----------
CPZ.CD.CPZANT	--NFN----------
CPZ.GA.CPZGAB	---FS----------L
CPZ.US.CPZUS	---FN-------M-------

MFSALSEGATPQDLNTMLNT

QUERY	MFSALSEGATPQDLNTMLNT
CONSENSUS_A	----------m---i
A.KE.Q23-CXC-CG	----------M---I
A.SE.SE6594	----------M---I
A.SE.SE7253	V---------M---I
A.SE.SE7535	----------M---I
A.SE.SE8131	----------H---M---I
A.SE.SE8538	----------I
A.SE.SE8891	----------G---M---I
A.UG.92UG037	----------M---I
A.UG.U455	----------M---V
CONSENSUS_B	----------
B.AU.AF128998	----------
B.-.NL43E9	----------
B.AU.MBC18	----------
B.AU.MBC200	----------
B.AU.MBC925	----------
B.AU.MBCC54	----------
B.AU.MBCC98	----------
B.AU.MBCD36	--T---------
B.CN.RL42	----------
B.DE.D31	----------
B.DE.HAN	----------
B.ES.89SP061	----------
B.FR.HXB2	----------
B.GA.OYI	-----A----------
B.GB.CAM1	----------
B.GB.MANC	---------I----------
B.JP.JH31	----------
B.NL.3202A21	----------
B.TW.LM49	----------
B.US.85WCIPR54	----------
B.US.AD8	----------
B.US.BC	----------
B.US.DH123	----------
B.US.JRCSF	----------
B.US.JRFL	----------
B.US.MNCG	----------
B.US.NC7	----------
B.US.NY5CG	----------
B.US.P896	----------
B.US.RF	----------
B.US.SF2	----------
B.US.WC001	----------
B.US.WEAU160	----------
B.US.WR27	----------Y-----
B.US.YU2	----------
CONSENSUS_C	--T---------
C.BR.92BR025	--T---------
C.BW.96BW01B22	--T---------
C.BW.96BW0402	--T---------
C.BW.96BW0502	--T---------
C.BW.96BW1104	--T----------T-
C.BW.96BW1210	--T---------
C.BW.96BW15B03	--T---------
C.BW.96BW1626	--T---------
C.BW.96BW17A09	--T---------
C.ET.ETH2220	--T---------
C.IN.93IN904	--T---------
C.IN.93IN905	--T---------
C.IN.93IN999	--T---------
C.IN.94IN11246	--T---------
C.IN.95IN21068	--T---------
CONSENSUS_D	----------
D.CD.84ZR085	----------
D.CD.ELI	----------
D.CD.NDK	----------
D.CD.Z2Z6	----------
D.UG.94UG1141	----------
CONSENSUS_F	----------
F.BR.BZ162	----------
F.CD.VI174	----------
F.RW.VI69	----------
CONSENSUS_F1	----------
F1.BE.VI850	----------T--------
F1.BR.93BR020.1	----------
F1.FI.FIN9363	----------
F1.FR.MP411	----------
CONSENSUS_F2	----------
F2.CM.MP255	----------
F2.CM.MP257	----------
CONSENSUS_G	----------xx-
G.BE.DRCBL	--T---------
G.FI.HH8793	----------
G.NG.92NG083	----------
G.SE.SE6165	----------L----
CONSENSUS_H	----------A----
H.BE.VI991	----------A----
H.BE.VI997	----------A----
H.CF.90CF056	----------A----
CONSENSUS_J	----------
J.SE.SE9173	----------
J.SE.SE9280	----------
CONSENSUS_K	----------
K.BE.VI325	-----AD----------
K.CD.EQTB11C	----------
K.CM.MP535	--T---------
N.CM.YBF30	--M--------S--------
CONSENSUS_O	--M------??Y-I-----A
O.CM.ANT70C	--M------ISY-I-----A
O.CM.MVP5180	--M------V-Y-I-----A
CRF01-AE.CF.90CF40	----------M---I
CRF01-AE.TH.93TH25	----------M---I
CRF01-AE.TH.CM240	----------M---I
CRF01-AE.TH.TH022	----------M---I
CRF01-AE.TH.TH047	----------M---I
CRF02_AG.FR.DJ263	--T----------M---I
CRF02_AG.FR.DJ264	--T----------M---I
CRF02_AG.NG.IBNG	----------M---I
CRF03_AB.RU.KAL15	----------M---I
CRF04_cpx.CY.94CY0	----------M---I
CRF04_cpx.GR.97PVC	----------M---I
CRF04_cpx.GR.97PVM	----------M---I
AC.ET.E3099G	----------
AC.IN.21301	--T---------
AC.RW.92RW009	--T---------
AC.SE.SE9488	--T---------
AC.ZM.ZAM174-21	--T---------
AC.ZM.ZAM184	----------
AC.ZM.ZAM716-17	--T---------
ACD.SE.SE8603	----------M---I
AD.SE.SE6954	---------A--------S-
AD.SE.SE7108	----------M---I
ADHU.NO.NOGIL3	------D--------M---I
ADU.CD.MAL	----------M---I
AG.NG.G3	--T---------
AG.SE.SE7812	----------M---I
AGHU.GA.VI354	----------M---I
AGJ.AU.BFP90	--T----------M---I
AGJ.ML.95ML8	----------M---I
AGU.CD.Z321	----------
BF.BR.93BR029.4	----------
DF.CD.VI961	--T---------
U.CD.VI1126	--T---------
CONSENSUS_CPZ	----------v-----A
CPZ.CD.CPZANT	----------H-------A
CPZ.GA.CPZGAB	---------L---V-----A
CPZ.US.CPZUS	--M----------V-----A

Study Subject ID:00RCH71
Study Subject Clone:
Study Subject HLA:A1,A2,B57,B81,Cw7,Cw18
Sequence: Known reactive 20Mer0: EKASFPEVIPMFSALSEGAT p24(29–48)

Possible HLA

A1	A*0101,A*0102
A2	A2.1,A*0201,A*0202,A*0203,A*0204,A*0205,A*0206,A*0207,A*0208,A*0209,A*0210,A*0211,A*0212,A*0213,A*0214,A*0216,A*0217,A*0218,A*0220,A*02
B57	Bw57,B*57,B*5701,B*5702,B*5703,B*5704
B81	B*8101
Cw7	Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(7-15)	EVIPMFSAL	A*0205
(8-15)	VIPMFSAL	A*0205
(7-15)	EVIPMFSAL	A*0214
(4-12)	SFPEVIPMF	Cw*0702
(7-15)	EVIPMFSAL	Cw*0702
(5-12)	FPEVIPMF	Cw*0702
(8-15)	VIPMFSAL	Cw*0702
(3-12)	ASFPEVIPMF	Cw*0702
(6-15)	PEVIPMFSAL	Cw*0702

Anchor Residues Searched

A1	XX[DE]XXXXX[Y]
A1	XX[DE]XXXX[Y]
A1	XX[DE]XXXXXX[Y]
A*0201	X[LM]XXXXXX[VL]
A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXXXX[VL]
A*0202	X[L]XXXXXX[LV]
A*0202	X[L]XXXXX[LV]
A*0202	X[L]XXXXXXX[LV]
A*0204	X[L]XXXXXX[L]
A*0204	X[L]XXXXX[L]
A*0204	X[L]XXXXXXX[L]
A*0205	X[VLIMQ]XXXXXX[L]
A*0205	X[VLIMQ]XXXXX[L]
A*0205	X[VLIMQ]XXXXXXX[L]
A*0206	X[V]XXXXXX[V]
A*0206	X[V]XXXXX[V]

A*0206	X[V]XXXXXXX[V]
A*0207	X[L][D]XXXXX[L]
A*0207	X[L][D]XXXX[L]
A*0207	X[L][D]XXXXXX[L]
A*0214	X[VQL]XXXXXX[LV]
A*0214	X[VQL]XXXXX[LV]
A*0214	X[VQL]XXXXXXX[LV]
Cw*0702	XXXXXXXX[YFL]
Cw*0702	XXXXXXX[YFL]
Cw*0702	XXXXXXXXX[YFL]

Study Subject ID:00RCH71
Study Subject Clone:
Study Subject HLA:A1,A2,B57,B81,Cw7,Cw18
Sequence: Known reactive 20Mer1: MFSALSEGATPQDLNTMLNT p24(39–58)

Possible HLA

A1	A*0101,A*0102
A2	A2.1,A*0201,A*0202,A*0203,A*0204,A*0205,A*0206,A*0207,A*0208,A*0209,A*0210,A*0211,A*0212,A*0213,A*0214,A*0216,A*0217,A*0218,A*0220,A*02
B57	Bw57,B*57,B*5701,B*5702,B*5703,B*5704
B81	B*8101
Cw7	Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(10-17)	PQDLNTML	A*0205
(10-17)	PQDLNTML	A*0214
(5-13)	SEGATPQDL	Cw*0702
(9-17)	TPQDLNTML	Cw*0702
(6-13)	EGATPQDL	Cw*0702
(10-17)	PQDLNTML	Cw*0702
(4-13)	LSEGATPQDL	Cw*0702
(8-17)	ATPQDLNTML	Cw*0702

Anchor Residues Searched

A1	XX[DE]XXXXX[Y]
A1	XX[DE]XXXX[Y]
A1	XX[DE]XXXXXX[Y]
A*0201	X[LM]XXXXXX[VL]
A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXXXX[VL]
A*0202	X[L]XXXXXX[LV]
A*0202	X[L]XXXXX[LV]
A*0202	X[L]XXXXXXX[LV]
A*0204	X[L]XXXXXX[L]
A*0204	X[L]XXXXX[L]
A*0204	X[L]XXXXXXX[L]
A*0205	X[VLIMQ]XXXXXX[L]
A*0205	X[VLIMQ]XXXXX[L]
A*0205	X[VLIMQ]XXXXXXX[L]
A*0206	X[V]XXXXXX[V]
A*0206	X[V]XXXXX[V]
A*0206	X[V]XXXXXXX[V]

A*0207	X[L][D]XXXXX[L]
A*0207	X[L][D]XXXX[L]
A*0207	X[L][D]XXXXXX[L]
A*0214	X[VQL]XXXXXX[LV]
A*0214	X[VQL]XXXXX[LV]
A*0214	X[VQL]XXXXXXX[LV]
Cw*0702	XXXXXXXX[YFL]
Cw*0702	XXXXXXX[YFL]
Cw*0702	XXXXXXXXX[YFL]

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(77–85)	SLFNTVATL	SLYNTVATL	SLYNTVATL	A*0201	
p24(15–23)	LSPRTLNAW	ISPRTLNAW	ISPRTLNAW	B57,B58	
p24(108–117)	TSTLQEQIGWF	TSTLQEQIGWM	TSTLQEQIGWM	B*57,B*5801	
p24(108–118)	TSTLQEQIGWF	TSTLQEQIGWM	TSTLQEQIGWM	B*5701	
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(121–129)	KLTPLCVSL	KLTPLCVTL	KLTPLCVTL	A2	
gp160(192–200)	KLTSCNTSV	RLISCNTSV	RLISCNTSV	A2	
gp160(192–200)	TLTSCNTSV	RLISCNTSV	RLISCNTSV	A2	
gp160(192–200)	TLTSCNTSV	RLISCNTSV	RLISCNTSV	A2.1	
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(700–708)	AVLSVVNRV	AVLSIVNRV	AVLSIVNRV	A2	
gp160(747–755)	RLVNGSLAL	RLVHGFLAI	RLVDGFLAL	A2	
gp160(770–778)	RLRDLLLIV	HHRDLLLIA	RLRDLLLIV	A*0201	
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	
Nef(136–145)	PLTFGWCFKL	PLTFGWCYKL	PLTFGWCFKL	A2	

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)]
	• 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: **p24**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(15–23)	p24()	LSPRTLNAW	HIV-1 exposed seronegative	human(B57,B58)	[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				
p24(108–117)	p24(240–249 LAI)	TSTLQEQIGWF	HIV-1 infection	human(B*57,B*5801)	[Goulder (1996b)]
	• Response to this epitope was found in 4 slow progressing HLA-B*57 individuals, in 2 it was dominant or very strong • For one donor (from Zimbabwe) this was de£ned as the optimal peptide • This epitope can be presented in the context of the closely related HLA molecules B*5801 and B*57				
p24(108–118)	p24(240–249 LAI)	TSTLQEQIGWF	HIV-1 infection	human(B*5701)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*5701 epitope				

Table 3: **RT**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(179–187)	RT()	VIYQYMMDL	HIV-1 exposure	human(A2)	[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 and D consensus sequences are both VIYQYMMDL				
RT(179–187)	Pol()	VIYQYMMDL	HIV-1 exposure	human(A2, A*0202)	[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, B and D clade viruses				
RT(308–317)	RT()	EILKEPVGHV	HIV-1 infection	human(A*0201)	[van der Burg (1997), Menendez-Arias (1998)]
	• 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 4: **gp160**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(121–129)	gp120(121–129)	KLTPLCVSL	*in vitro* stimulation	human(A2)	[Zarling (1999)]
	• 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-de£ned HIV epitopes: KIRLRPGGK, ILKEPVHGV, IRLRPGGK, GPKVKQWPL				
gp160(192–200)	gp120(192–199 HXB2R)	KLTSCNTSV	HIV-1 infection	human(A2)	[Brander (1995)]
	• Epitope predicted on HLA binding motif, and studied in the context of inclusion in a synthetic vaccine				
gp160(192–200)	gp120(197–205)	TLTSCNTSV	no CTL shown	human(A2)	[Garboczi (1992)]
	• Crystallization of HLA-A2 molecules complexed with antigenic peptides – refers to Dadaglio *et al* 1991				
gp160(192–200)	gp120(199–207)	TLTSCNTSV	peptide immunization and HIV-1 infection	human(A2.1)	[Brander (1996)]
	• 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				
gp160(311–320)	gp160(318–327 IIIB)	RGPGRAFVTI	CTL line from HIV-donor	human(A*0201)	[Alexander-Miller (1996)]
	• 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				
gp160(311–320)	gp160(318–327 IIIB)	RGPGRAFVTI	vaccinia IIIB gp160	human(A2)	[Achour (1996)]
	• Individual was immunized with rec vaccinia gp160 IIIB and boosted with puri£ed 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 speci£c CTL response				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(311–320)	gp160(318–327 SIMI)	MGPKRAFYAT	vaccinia SIMI gp160	human(A2)	[Achour (1996)]
	• Individual was immunized with rec vaccinia gp160 SIMI and boosted with puri£ed recombinant gp160 SIMI • P18 MN and RF peptides were able to stimulate the HIV-speci£c CTL that arose in response to the SIMI vaccination, thus the P18 MN peptide (IGPGRAFYTT) and the P18 RF peptide (KGPGRVIYAT) could cross-react • The P18 IIIB peptide does not cross-react (RGPGRAFVTI in the epitope region) • gp160 SIMI primed immune cells could generate a signi£cantly broader speci£city when stimulated with P18 MN or P18RF peptides, but not P18 IIIB				
gp160(369–375)	gp120(374–380 BRU)	PEIVTHS	HIV-1 infection	human(A2)	[Dadaglio (1991)]
	• De£ned through blocking CTL activity, and Env deletions				
gp160(377–387)	gp120(377–387)	NSGGEFFYSNS		human(A2)	[Hickling (1990)]
	• Peptides recognized by class I restricted CTL can bind to class II				
gp160(700–708)	gp41(705–714)	AVLSVVNRV	HIV-1 infection	human(A2)	[Ferris (1999)]
	• This epitope is processed by a TAP1/2 dependent mechanism				
gp160(747–755)	gp41(747–755)	RLVNGSLAL	HIV-1 infection	human(A2)	[Parker (1992)]
	• Studied in the context of HLA-A2 peptide binding				
gp160(770–778)	Env(679–777)	RLRDLLLIV	HIV-1 infection	human(A*0201)	[Kmieciak (1998)]
	• 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 *in vitro* • Peptides 5.3 and D2 bound to HLA A*0201 with low af£nity and were variable, particularly D2;				
gp160(813–822)	gp41(814–823 LAI)	SLLNATDIAV	MN rec gp160	human(A*0201)	[Dupuis (1995)]
	• 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 *et al*., 1999 database				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(813–822)	gp41(814–823)	SLLNATDIAV	HIV-1 infection	human(A2)	[Kundu (1998b)]
	• 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-speci£c 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 SLFNAIDIAV or SLLNTTDIVV and no detectable CTL response • CTL demonstrated against peptide-coated target, epitope is naturally processed and enhancible with vaccine				
gp160(813–822)	Env(814–823 Clade B)	SLLNATDIAV	HIV-1 MN rgp160	human(A2.1)	[Kundu (1998a)]
	• 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 £fty 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 identi£ed in gp160, of which 25 had a high or intermediate binding af£nity • Eleven peptides were studied that had high HLA-A2 binding af£nity – 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 NLFNTTAIAVA or SLLNATAITVA				
gp160(814–822)	gp41(815–823 LAI)	LLNATDIAV	MN rec gp160	human(A2)	[Dupuis (1995)]
	• Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823				

Table 5: **Nef**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
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				

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

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(30–37)	p24(162–170 LAI)	KAFSPEVI	HIV-1 infection	human(B*5703)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*5703 epitope				
p24(30–37)	p24(30–37)	KAFSPEVI	HIV-1 infection	human(B57)	[Goulder (2000b)]
	• Two strong clonal CTL responses were generated in donor 026-BMC (HLA A3/-, B42/B57, Cw7/17) against different optimal versions of this epitope, one 8 amino acids long, one 11 • Improved stabilization of the B57-peptide complex was demonstrated by the 11 mer which £ts the B57 binding motif, relative to the 8 mer, which does not • B57 tolerates marked difference in optimal peptide length – and B57 is associated with non-progressive infection				
p24(30–40)	p24()	KAFSPEVIPMF	HIV-1 infection	human(B*57)	[Spiegel (1999)]
	• Study examines the effect of highly active antiretroviral therapy (HAART) on HIV-1 plasma viral load, CTLp and CTLe frequencies in 8 infected children • CTLp (precursors) were measured by stimulating in culture and assaying using 51Cr release, against vaccina expressed IIIB Env, Gag, Pol, Nef, and CTLe were measured by ELISPOT • CTL against B*57-KAFSPEVIPMF was a de novo response observed in one of the children when viral load increased as a result of stopping therapy • HIV-1 speci£c CTL responses initially increased in children with complete viral suppression, but then decreased, suggesting viral replication is needed to maintain CTL responses				
p24(30–40)	p24(162–172 LAI)	KAFSPEVIPMF	HIV-1 infection	human(B*5701)	[Goulder (1996b)]
	• This peptide was recognized by CTL from £ve slow progressors • Peptide de£ned on the basis of B*5801 binding motif, yet not cross-restricted except at high concentrations • This epitope is highly conserved				
p24(30–40)	p24(162–172 LAI)	KAFSPEVIPMF	HIV-1 infection	human(B*5701)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*5701 epitope				
p24(30–40)	p24(162–172 LAI)	KAFSPEVIPMF	HIV-1 infection	human(B*5703)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*5703 epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(30–40)	p24(30–40)	KAFSPEVIPMF	HIV-1 infection	human(B57)	[Goulder (2000b)]
	• Two strong clonal CTL responses were generated in donor 026-BMC (HLA A3/-, B42/B57, Cw7/17) against different optimal versions of this epitope, one 8 amino acids long, one 11 • Improved stabilization of the B57-peptide complex was demonstrated by the 11mer which £ts the B57 binding motif, relative to the 8 mer, which does not • B57 tolerates marked difference in optimal peptide length – and B57 is associated with non-progressive infection				
p24(30–40)	p24(162–172)	KAFSPEVIPMF	HIV-1 infection	human(B57)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was HLA A*0201, A1, B57 and responded to four B57 epitopes and two others				
p24(30–40)	p24()	KAFSPEVIPMF	HIV-1 infection	human(B57)	[Goulder (2000a)]
	• The CTL-dominant response was focused on this epitope in a HIV+ Caucasian living in Boston – this epitope is not among the most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p24(35–43)	p24(167–175 LAI)	EVIPMFSAL		human(A*2601)	[Goulder (1996a)]
	• Identi£ed as optimal epitope within Gag sequence AFSPEVIPMFSALSEGATPQ • Relatively conserved epitope within B clade and in other clades • Suspected binding motif for HLA-A26 includes T or V anchor at position 2, negative charge at position 1 • C. Brander notes that this is an A*2601 epitope in the 1999 database				
p24(35–43)	p24(167–175 LAI)	EVIPMFSAL		human(A*2601)	[Brander & Goulder(2001)]
	• C. Brander notes that this is an A*2601				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(35–43)	p24(167–175)	EVIPMFSAL	HIV-1 infection	human(A26)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned 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				
p24(36–43)	p24(168–175 LAI)	VIPMFSAL	?	human(C*0102(Cw1))	[Brander & Goulder(2001)]
	• C. Brander notes this is a C*0102(Cw1) epitope				
p24(36–43)	p24(168–175 LAI)	VIPMFSAL		human(Cw*0102,Cw1)	[Goulder (1997)]
p24(36–43)	p24(168–175)	VIPMFSAL	HIV-1 infection	human(Cw01,Cw02)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned 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				

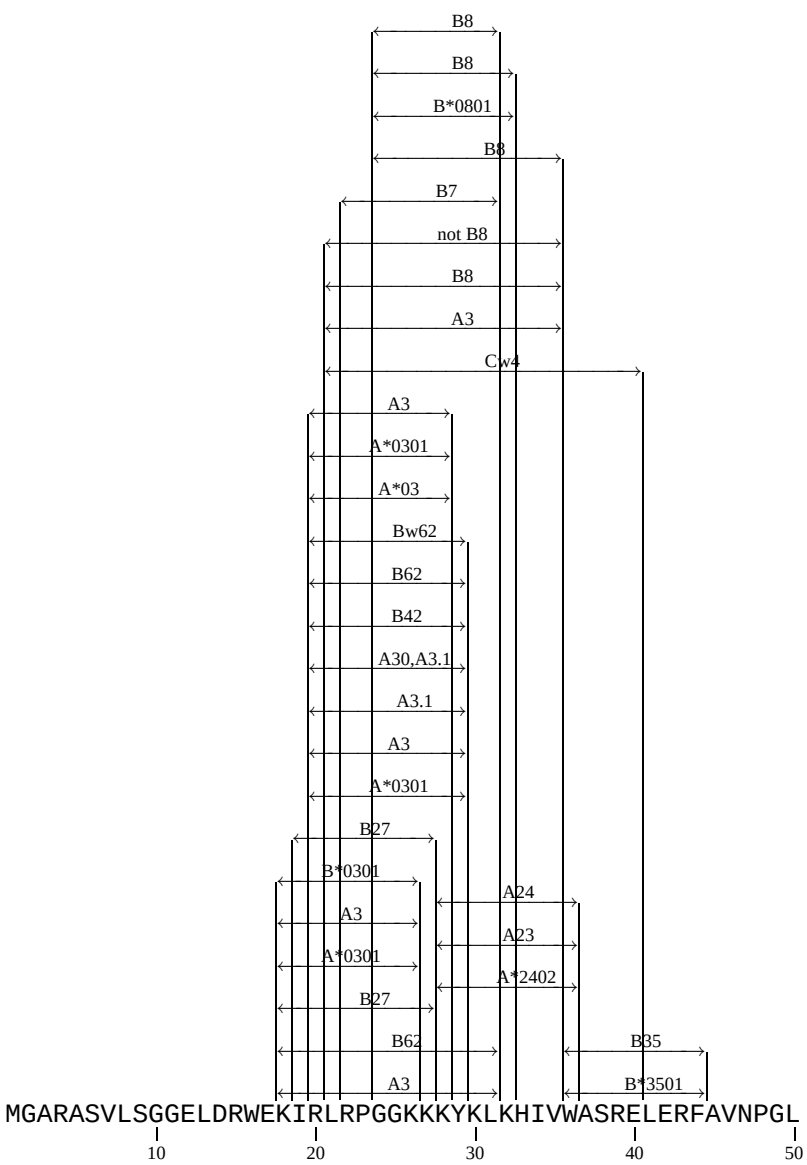
Table 7: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(30–37)	p24(162–170 LAI)	KAFSPEVI	HIV-1 infection	human(B*5703)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*5703 epitope				
p24(30–37)	p24(30–37)	KAFSPEVI	HIV-1 infection	human(B57)	[Goulder (2000b)]
	• Two strong clonal CTL responses were generated in donor 026-BMC (HLA A3/-, B42/B57, Cw7/17) against different optimal versions of this epitope, one 8 amino acids long, one 11 • Improved stabilization of the B57-peptide complex was demonstrated by the 11 mer which £ts the B57 binding motif, relative to the 8 mer, which does not • B57 tolerates marked difference in optimal peptide length – and B57 is associated with non-progressive infection				
p24(30–40)	p24()	KAFSPEVIPMF	HIV-1 infection	human(B*57)	[Spiegel (1999)]
	• Study examines the effect of highly active antiretroviral therapy (HAART) on HIV-1 plasma viral load, CTLp and CTLe frequencies in 8 infected children • CTLp (precursors) were measured by stimulating in culture and assaying using 51Cr release, against vaccina expressed IIIB Env, Gag, Pol, Nef, and CTLe were measured by ELISPOT • CTL against B*57-KAFSPEVIPMF was a de novo response observed in one of the children when viral load increased as a result of stopping therapy • HIV-1 speci£c CTL responses initially increased in children with complete viral suppression, but then decreased, suggesting viral replication is needed to maintain CTL responses				
p24(30–40)	p24(162–172 LAI)	KAFSPEVIPMF	HIV-1 infection	human(B*5701)	[Goulder (1996b)]
	• This peptide was recognized by CTL from £ve slow progressors • Peptide de£ned on the basis of B*5801 binding motif, yet not cross-restricted except at high concentrations • This epitope is highly conserved				
p24(30–40)	p24(162–172 LAI)	KAFSPEVIPMF	HIV-1 infection	human(B*5701)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*5701 epitope				
p24(30–40)	p24(162–172 LAI)	KAFSPEVIPMF	HIV-1 infection	human(B*5703)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*5703 epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(30–40)	p24(30–40)	KAFSPEVIPMF	HIV-1 infection	human(B57)	[Goulder (2000b)]
	• Two strong clonal CTL responses were generated in donor 026-BMC (HLA A3/-, B42/B57, Cw7/17) against different optimal versions of this epitope, one 8 amino acids long, one 11 • Improved stabilization of the B57-peptide complex was demonstrated by the 11mer which £ts the B57 binding motif, relative to the 8 mer, which does not • B57 tolerates marked difference in optimal peptide length – and B57 is associated with non-progressive infection				
p24(30–40)	p24(162–172)	KAFSPEVIPMF	HIV-1 infection	human(B57)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was HLA A*0201, A1, B57 and responded to four B57 epitopes and two others				
p24(30–40)	p24()	KAFSPEVIPMF	HIV-1 infection	human(B57)	[Goulder (2000a)]
	• The CTL-dominant response was focused on this epitope in a HIV+ Caucasian living in Boston – this epitope is not among the most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p24(35–43)	p24(167–175 LAI)	EVIPMFSAL		human(A*2601)	[Goulder (1996a)]
	• Identi£ed as optimal epitope within Gag sequence AFSPEVIPMFSALSEGATPQ • Relatively conserved epitope within B clade and in other clades • Suspected binding motif for HLA-A26 includes T or V anchor at position 2, negative charge at position 1 • C. Brander notes that this is an A*2601 epitope in the 1999 database				
p24(35–43)	p24(167–175 LAI)	EVIPMFSAL		human(A*2601)	[Brander & Goulder(2001)]
	• C. Brander notes that this is an A*2601				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(35–43)	p24(167–175)	EVIPMFSAL	HIV-1 infection	human(A26)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned 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				
p24(36–43)	p24(168–175 LAI)	VIPMFSAL	?	human(C*0102(Cw1))	[Brander & Goulder(2001)]
	• C. Brander notes this is a C*0102(Cw1) epitope				
p24(36–43)	p24(168–175 LAI)	VIPMFSAL		human(Cw*0102,Cw1)	[Goulder (1997)]
p24(36–43)	p24(168–175)	VIPMFSAL	HIV-1 infection	human(Cw01,Cw02)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned 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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B7
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B8
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Cw4
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B42
A30,A3.1
A3.1
A3
A*0301
B27
B*0301
A3
A*0301
B27
B62
A3
A24
A23
A*2402
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B*3501
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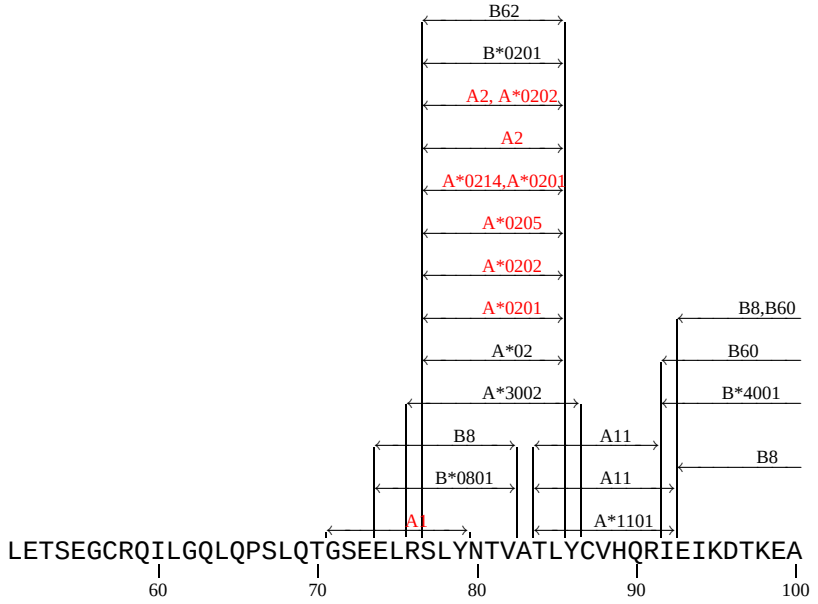
30
40
50


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
B8,B60
B60
B*4001
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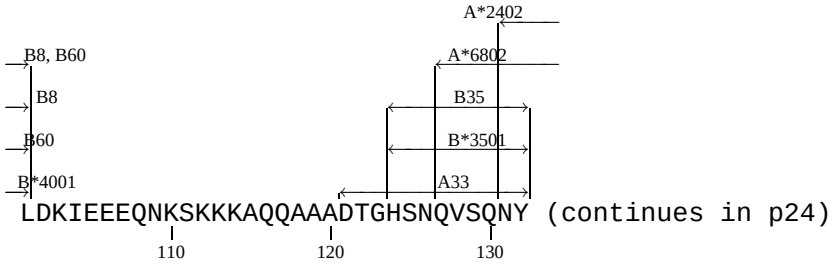
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A*6802
B35
B*3501
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B60
B*4001
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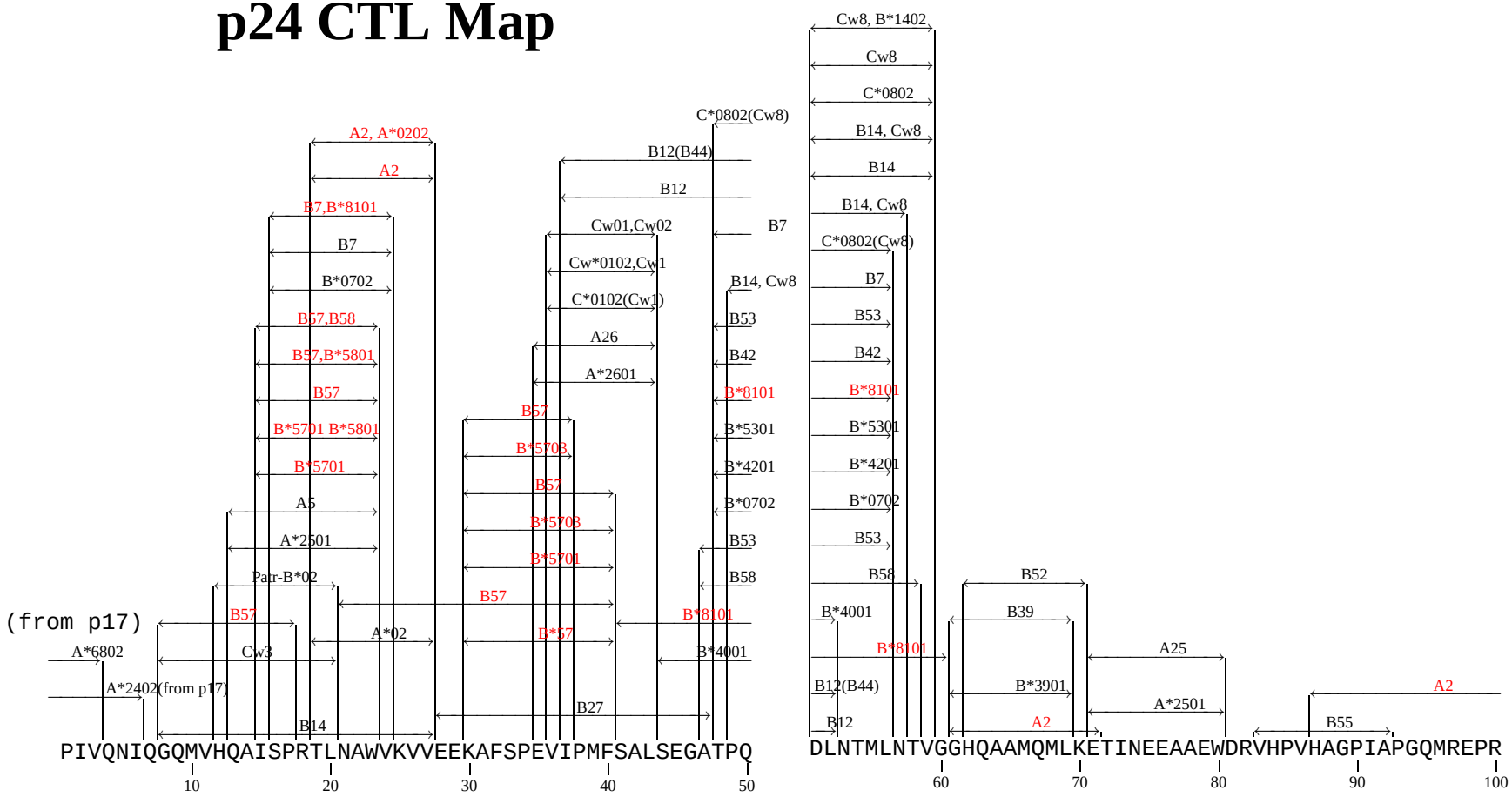
120
130

p24 CTL Map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A*2402(from p17)
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B7
B*0702
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B57,B*5801
B57
B*5701 B*5801
B*5701
A5
A*2501
Pair-B*02
B57
Cw3
B14
A*02
C*0802(Cw8)
B12(B44)
B12
Cw01,Cw02
Cw*0102,Cw1
C*0102(Cw1)
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A*2601
B57
B*5703
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B57
B*57
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B7
B14, Cw8
B53
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B53
B58
B*4001
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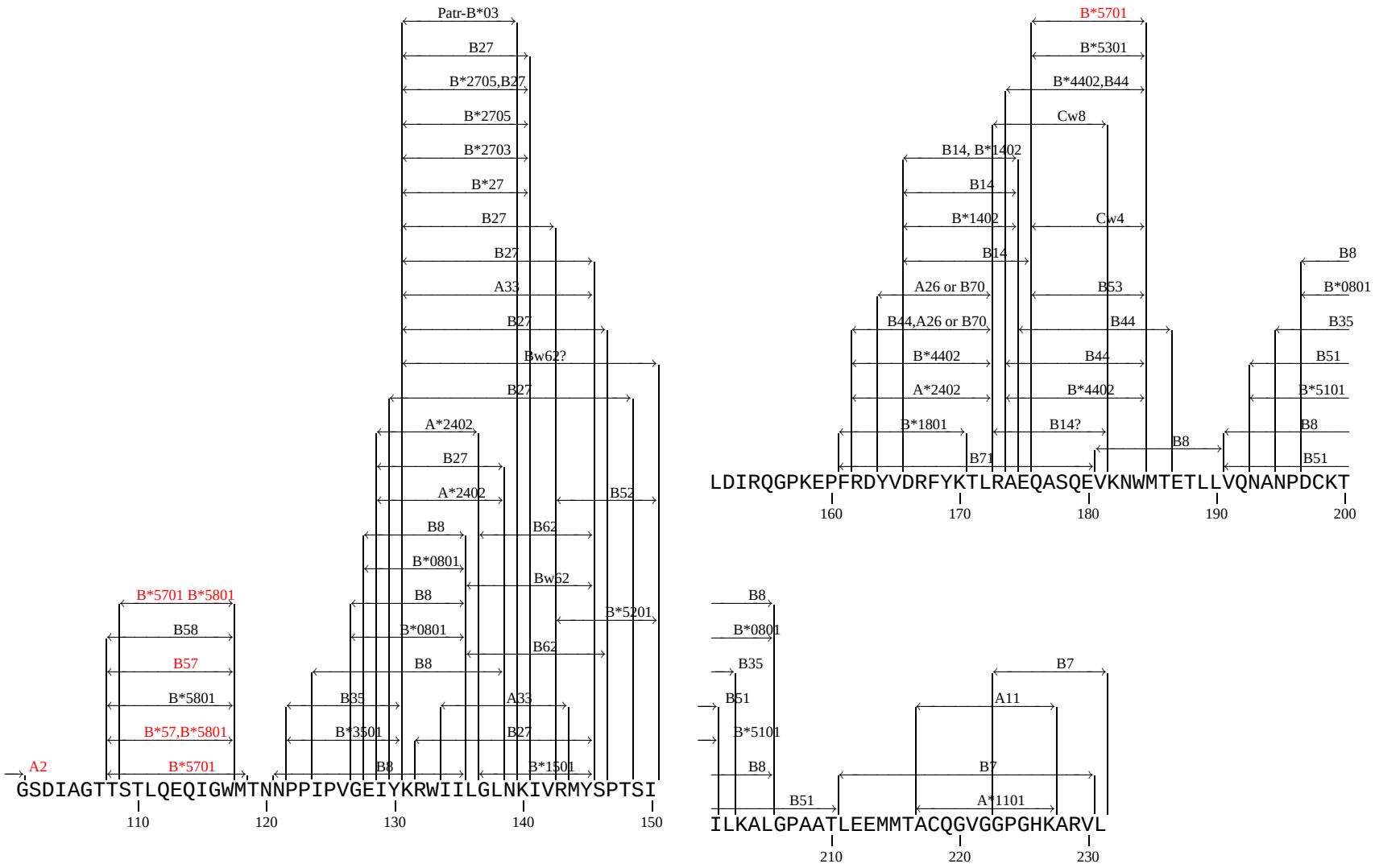
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A*2402
B27
A*2402
B52
B8
B62
B*0801
Bw62
B*5701 B*5801
B8
B*5201
B58
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140
150
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Cw8
B14, B*1402
B14
B*1402
Cw4
B14
B8
A26 or B70
B53
B*0801
B44,A26 or B70
B44
B35
B*4402
B44
B51
A*2402
B*4402
B*5101
B*1801
B14?
B8
B8
B71
B51
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160
170
180
190
200
B8
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B35
B51
B*5101
B8
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A11
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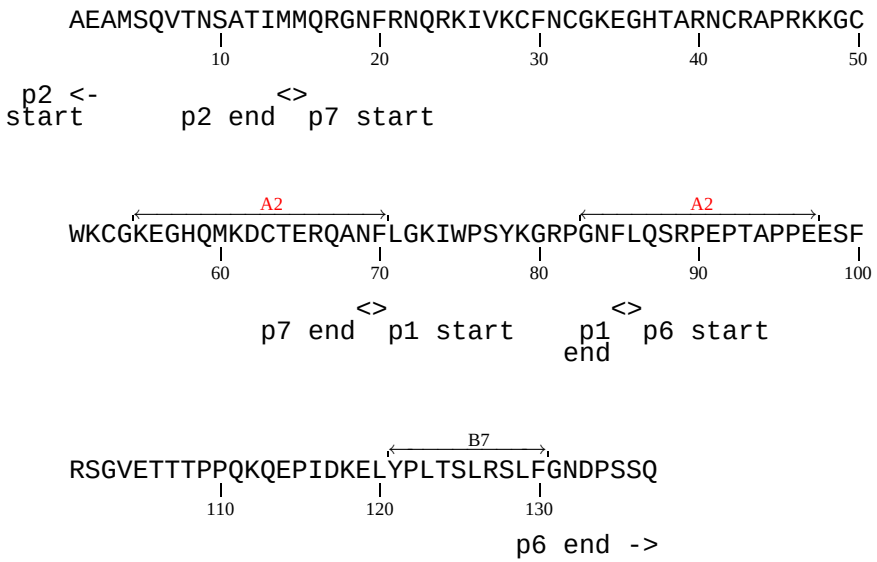
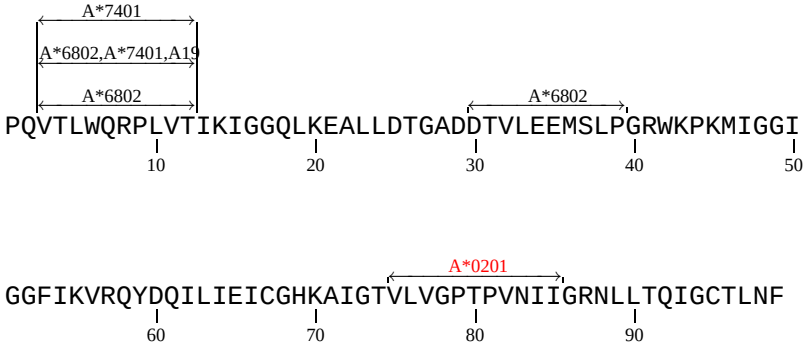
A*1101

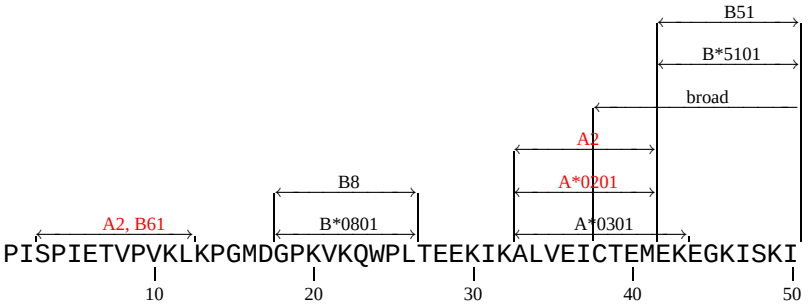
p2p7p1p6 CTL Map



Protease CTL Map



RT CTL Map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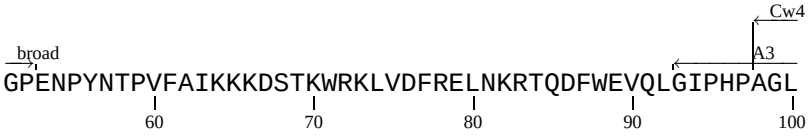
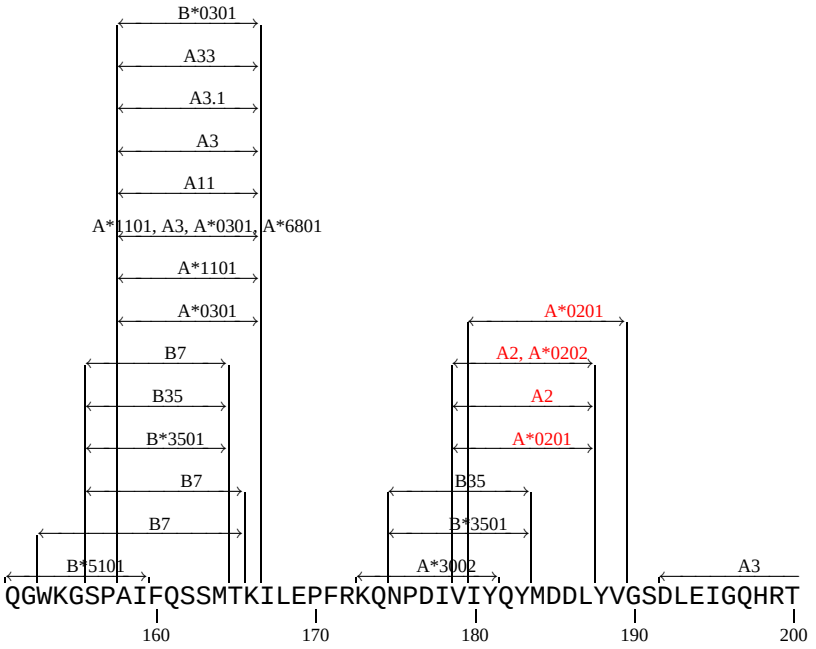
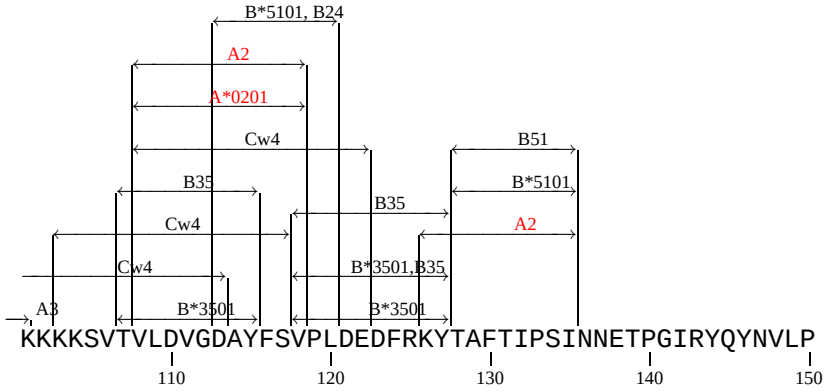
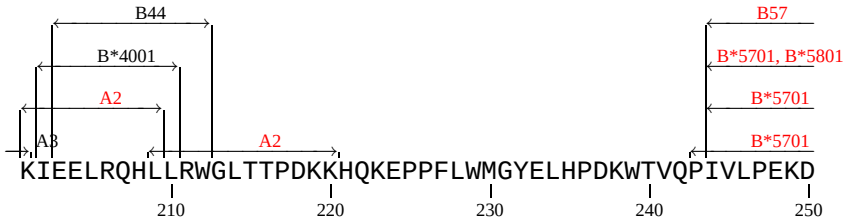
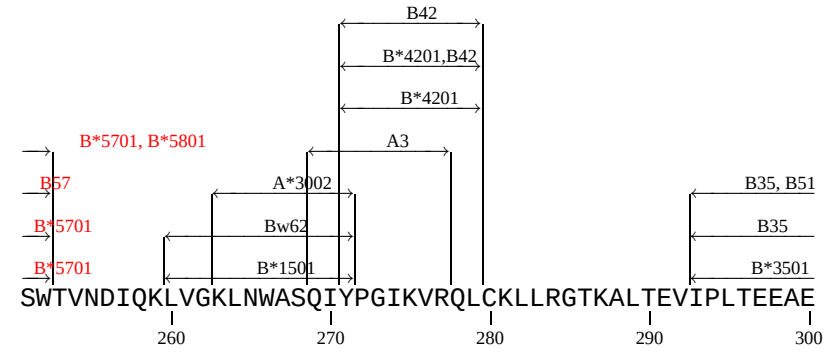
broad
A2
B8
A*0201
A2, B61
B*0801
A*0301
PISPIETVPVKLKPGMDGPKVKQWPLTEEKIKALVEICTEMEKEGKISKI
10
20
30
40
50

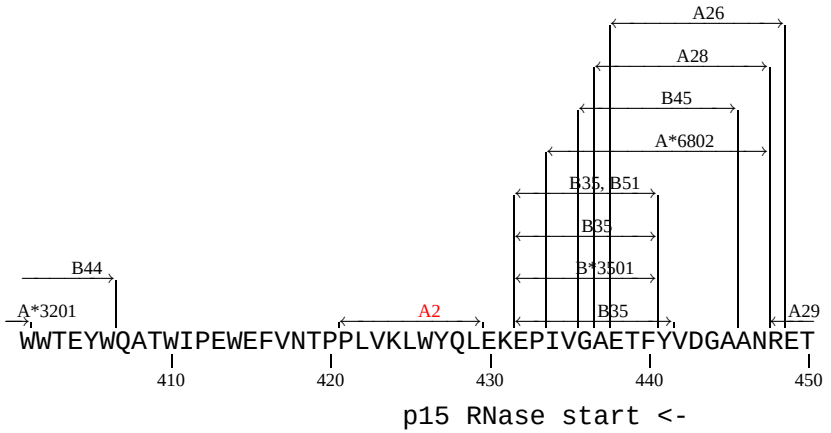

Cw4
broad
A3
GPENPYNTPVFAIKKKDSTKWRKLVDFRELNKRTQDFWEVQLGIPHPAGL
60
70
80
90
100


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

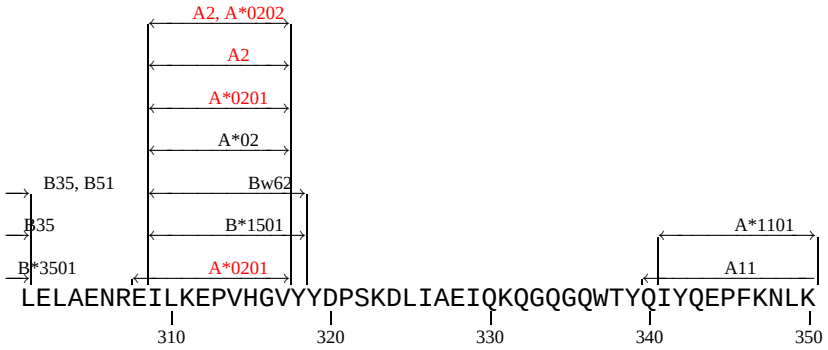

B*5101, B24
A2
A*0201
Cw4
B51
B35
B*5101
B35
Cw4
A2
Cw4
B*3501,B35
A3
B*3501
B*3501
KKKKSVTVLDVGDAYFSVPLDEDFRKYTAFTIPSINNETPGIRYQYNVLP
110
120
130
140
150

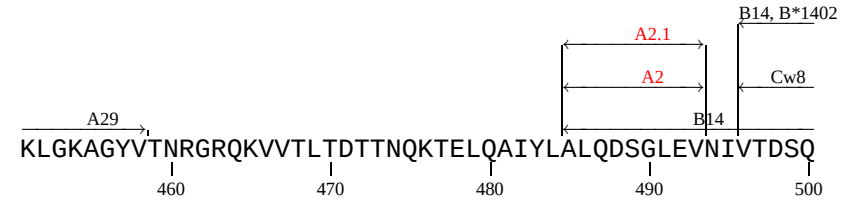

B44
B57
B*4001
B*5701, B*5801
A2
B*5701
A3
A2
B*5701
KIEELRQHLLRWGLTTPDKKHQKEPPFLWMGYELHPDKWTVQPIVLPEKD
210
220
230
240
250

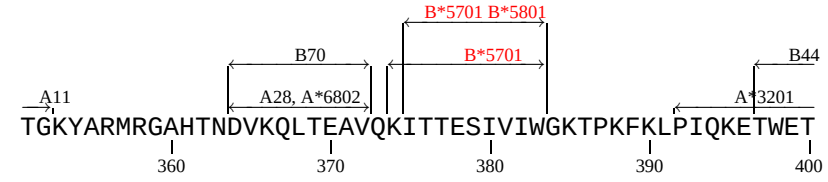


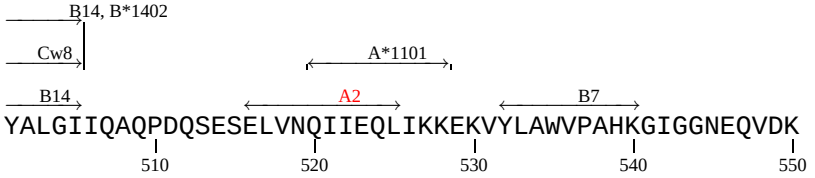


p15 RNase start <-





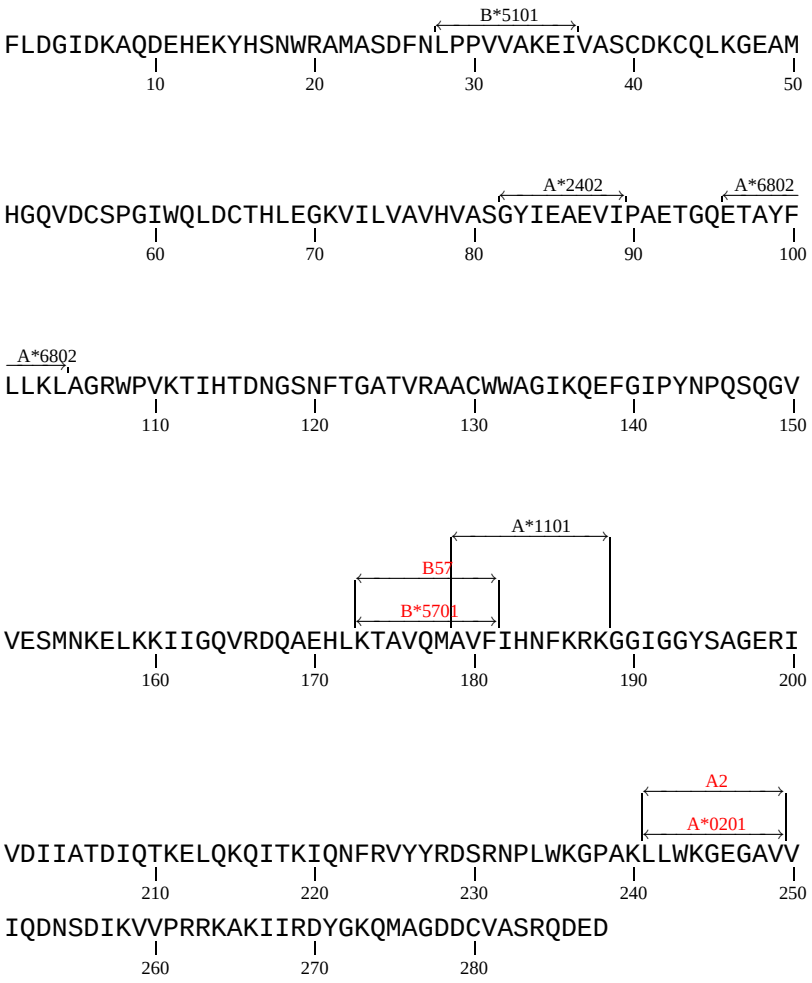




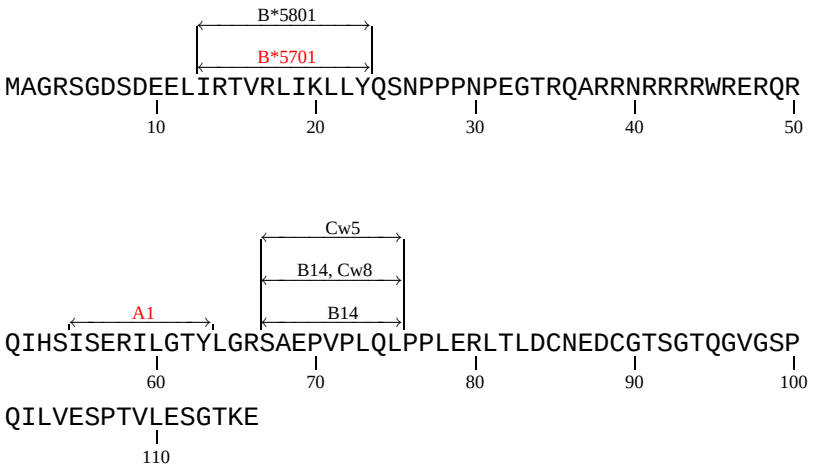
LVSAGIRKVL

-> p15 RNase end

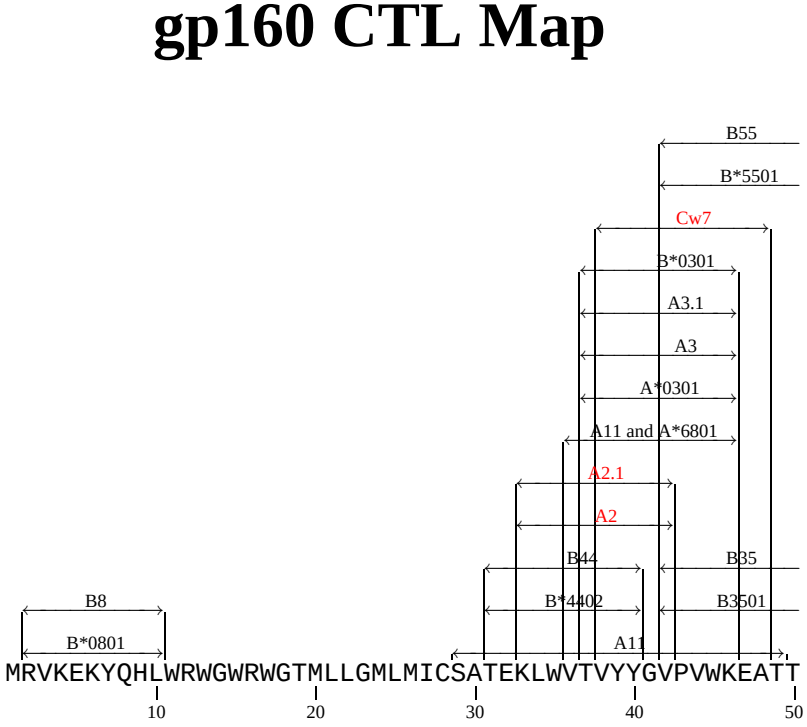
Integrase CTL Map



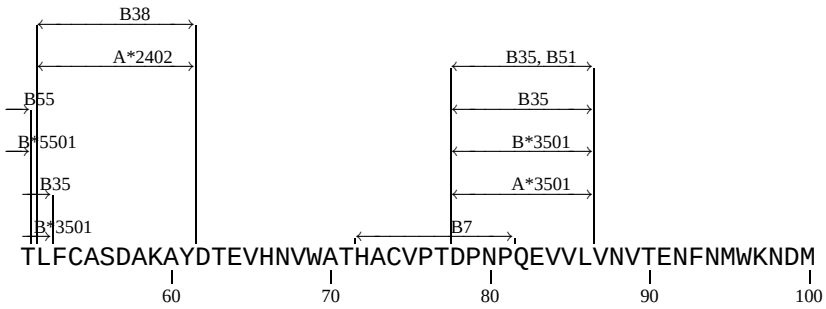
Rev CTL Map

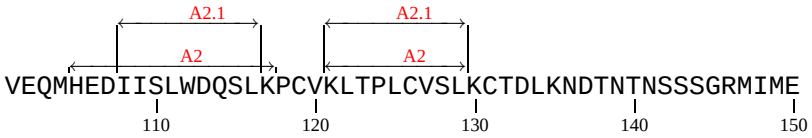


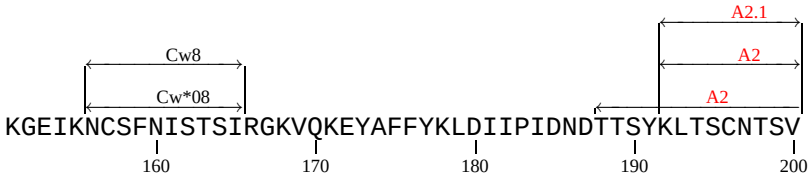
gp160 CTL Map

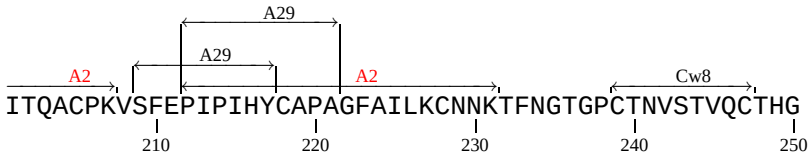


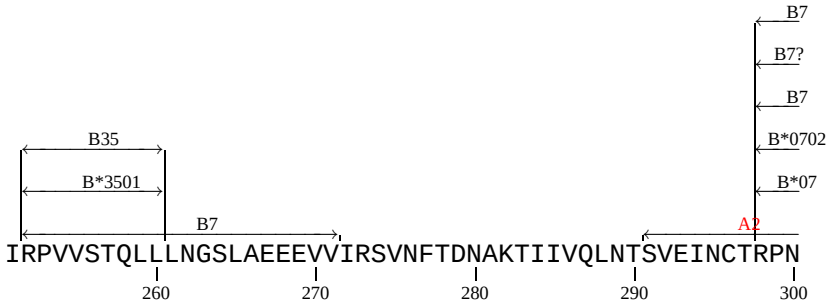
<- gp120 start

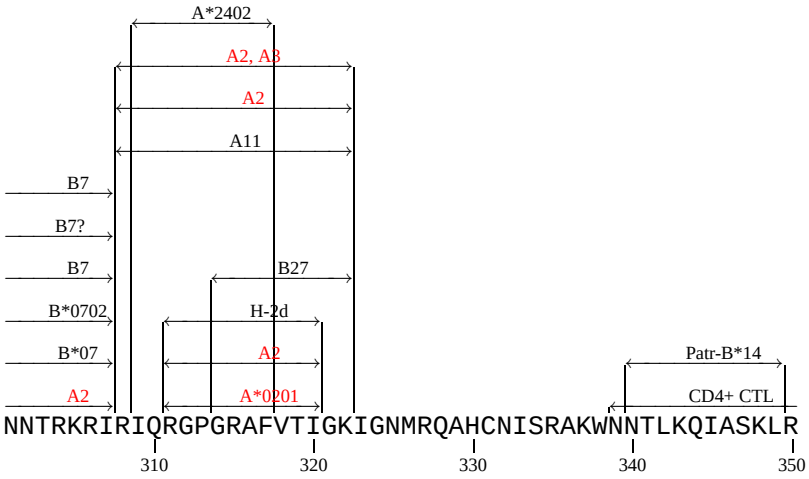


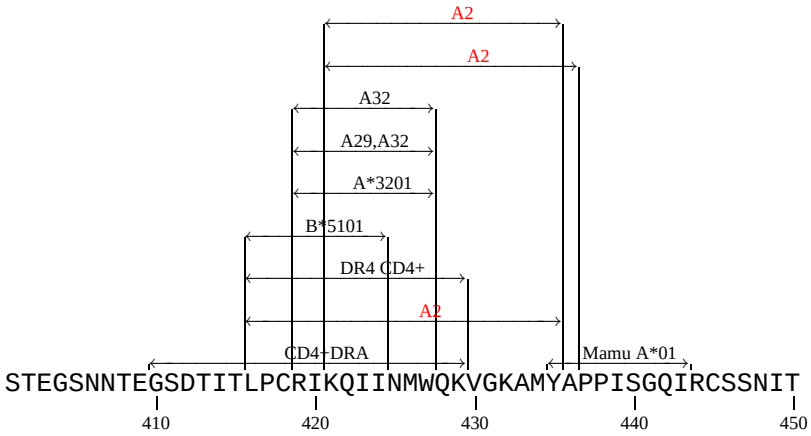


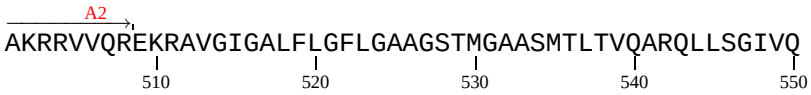




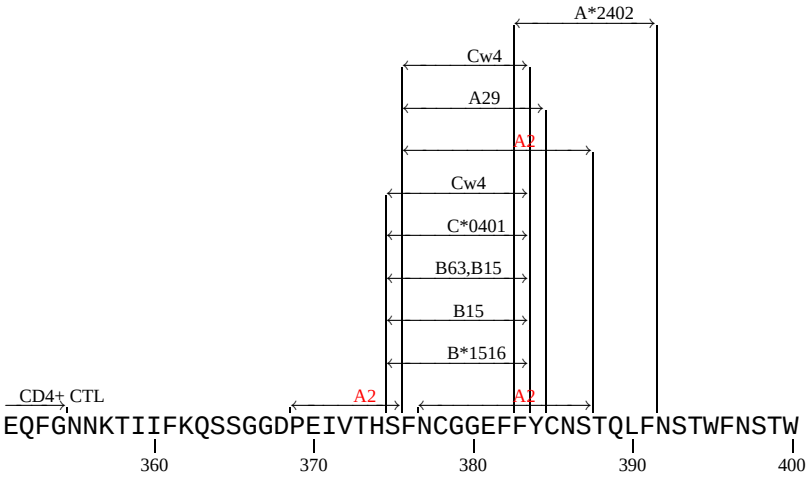


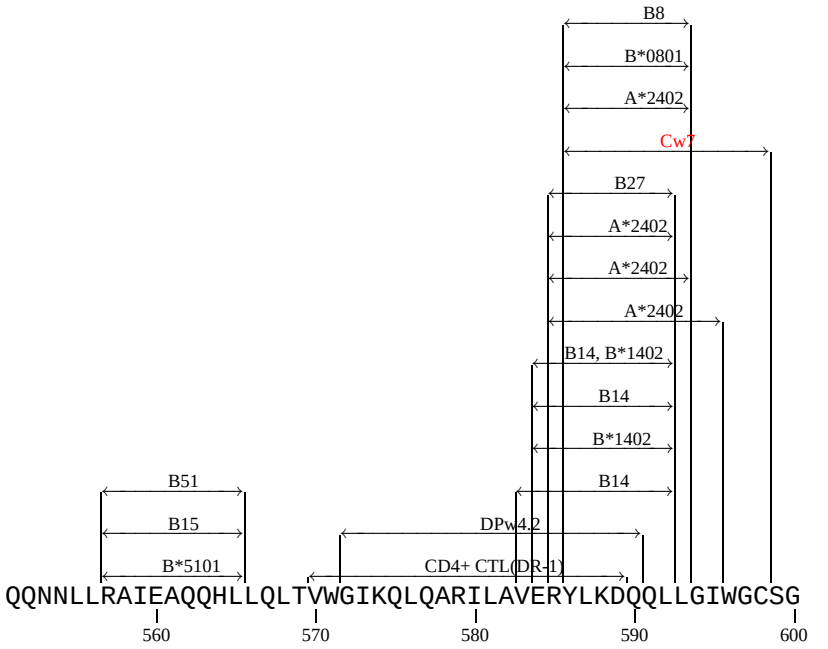


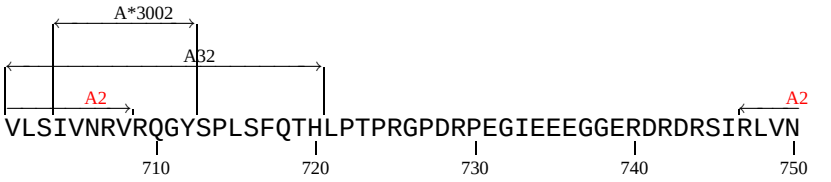


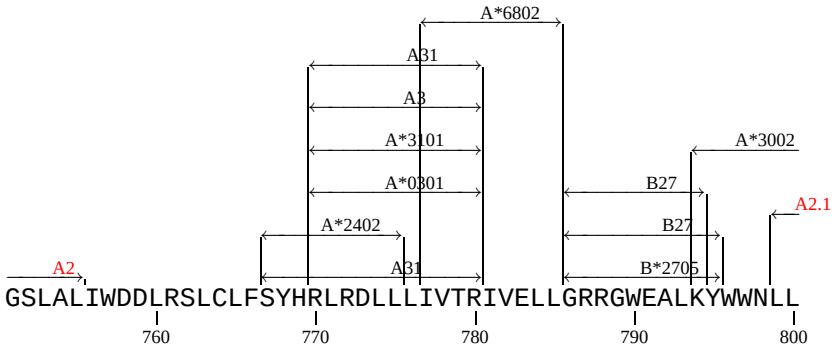


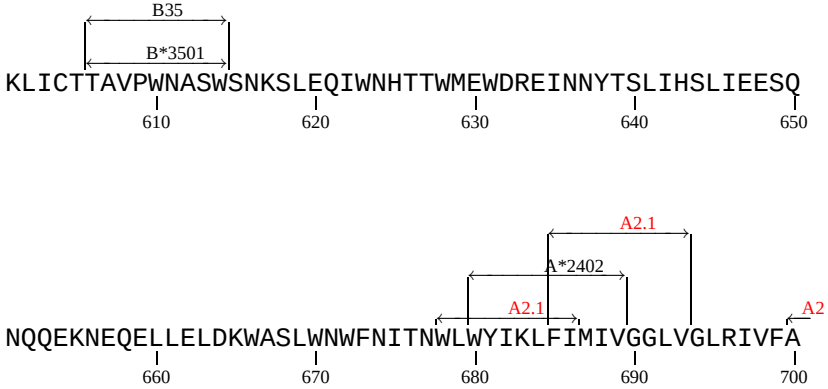
gp120 end <> gp41 start



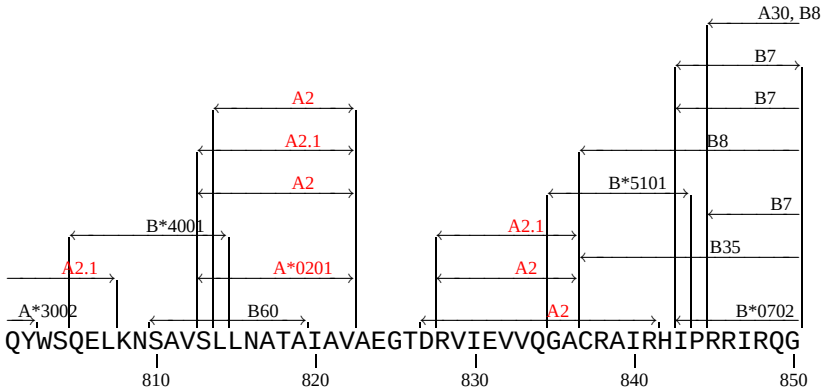

B8
B*0801
A*2402
Cw7
B27
A*2402
A*2402
A*2402
B14, B*1402
B14
B*1402
B14
B51
B15
B*5101
DPw4.2
CD4+ CTL(DR-1)
QQNNLLRAIEAQQHLLQLTVWGIKQLQARILAVERYLKDQQLLGIWGCSG
560
570
580
590
600

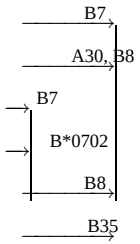

A*3002
A32
A2
A2
VLSIVNRVRQGYSPLSFQTHLPTPRGPDRPEGIEEEGGERDRDRSIRLVN
710
720
730
740
750


A*6802
A31
A3
A*3101
A*0301
A*2402
A31
A*3002
B27
B27
B*2705
A2.1
A2
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

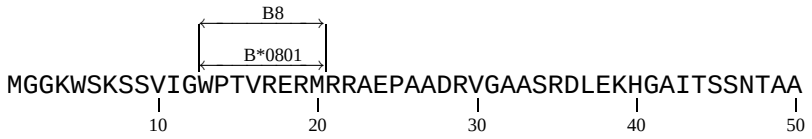

A30, B8
B7
B7
A2
A2.1
B8
A2
B*5101
B7
B*4001
A2.1
B35
A2.1
A*0201
A2
A*3002
B60
A2
B*0702
QYWSQELKNSAVSLLNATAIAVAEGTDRVIEVVQGACRAIRHIPRRIRQG
810
820
830
840
850

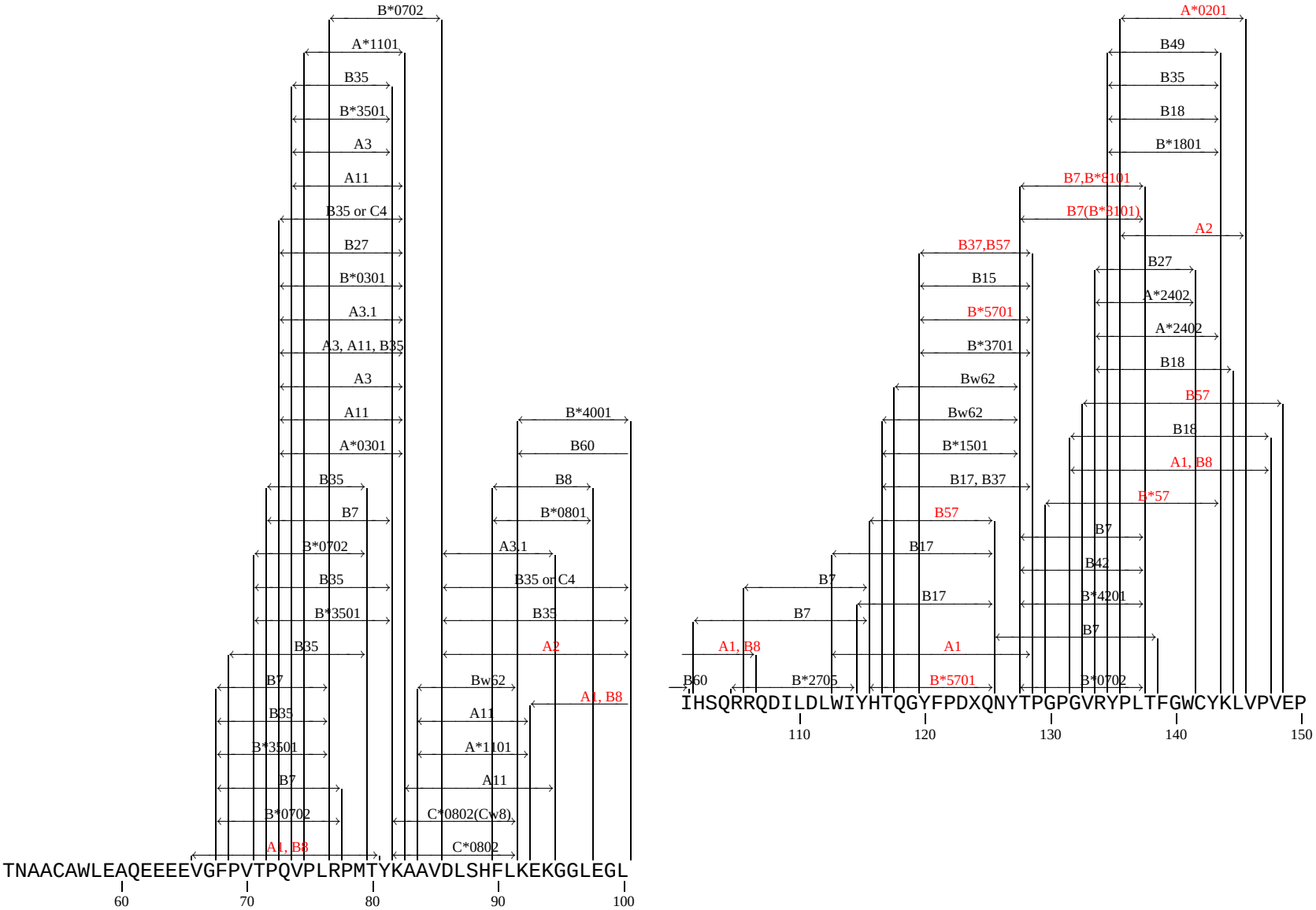


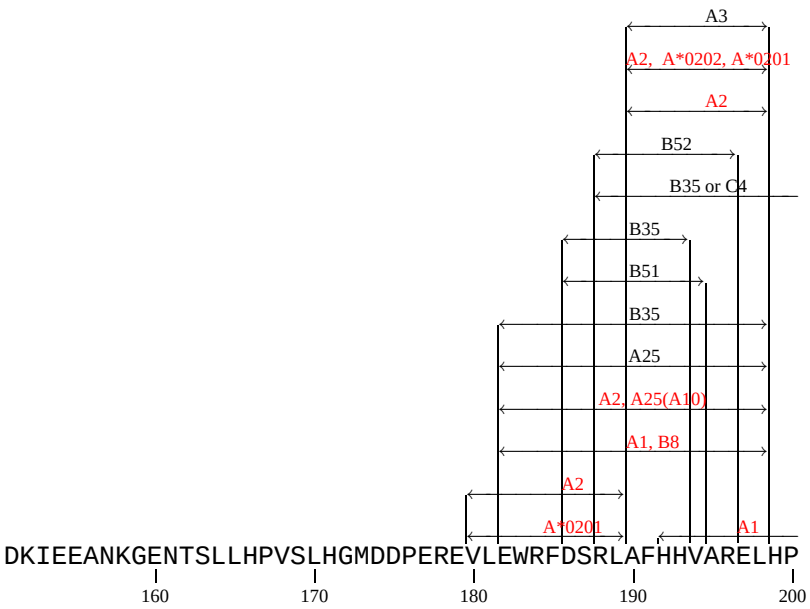
LERILL

-> 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
A1, B8
B*4001
B60
B8
B*0801
A3.1
B35 or C4
B35
A2
Bw62
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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[Brander (1996)] C. Brander, G. Corradin, T. Hasler, & W. Pichler. Peptide immunization in humans: a combined CD8+/CD4+ T cell-targeted vaccine restimulates the memory CD4 T cell response but fails to induce cytotoxic T lymphocytes (CTL). *Clin Exp Immunol* **105**:18–25, 1996. (Medline: 96280772).

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cytotoxic T lymphocytes generated from donors infected in Africa. *J Virol* **73**:1708–14, 1999. (Medline: 99099071).

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[Goulder (1996a)] P. Goulder, C. Conlon, K. McIntyre, & A. McMichael. Identi£cation of a novel human leukogen antigen A26-restricted epitope in a conserved region of Gag. *AIDS* **10(12)**:1441–1443, 1996a. (Medline: 97057743) Notes: This paper is correspondence brie¤y describing the identi£cation and characterization of an immuno-dominant A26-CTL epitope in an asymptomatic HIV+ individual.

[Goulder (2000a)] P. J. Goulder, C. Brander, K. Annamalai, N. Mngqundaniso, U. Govender, Y. Tang, S. He, K. E. Hartman, C. A. O'Callaghan, G. S. Ogg, M. A. Altfeld, E. S. Rosenberg, H. Cao, S. A. Kalams, M. Hammond, M. Bunce, S. I. Pelton, S. A. Burchett, K. McIntosh, H. M. Coovadia, & B. D. Walker. Differential narrow focusing of immunodominant human immunode£ciency virus gag-speci£c cytotoxic T-lymphocyte responses in infected African and caucasoid adults and children. *J Virol* **74**:5679–90, 2000a. (Medline: 20283828).

[Goulder (1997)] P. J. Goulder, M. Bunce, G. Luzzi, R. E. Phillips, & A. J. McMichael. Potential underestimation of HLA-C-restricted cytotoxic T-lymphocyte responses. *AIDS* **11(15)**:1884–1886, 1997. (Medline: 98074190).

[Goulder (2000b)] P. J. Goulder, Y. Tang, S. I. Pelton, & B. D. Walker. HLA-B57-Restricted cytotoxic T-lymphocyte activity in a single infected subject toward two optimal epitopes, one of which is entirely contained within the other. *J Virol* **74**:5291–9, 2000b. (Medline: 20261752).

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