

VQMAVFIHNFKRKGGIGGYS

QUERY VQMAVFIHNFKRKGGIGGYS

CONSENSUS_A ----- I-GY
 A.KE.Q23-CXC-CG ----- I-GY
 A.SE.SE6594 ----- I-GY
 A.SE.SE7253 ----- I-GY
 A.SE.SE7535 -----R-- I-GY
 A.SE.SE8131 ----- I-GY
 A.SE.SE8538 ----- I-GY
 A.SE.SE8891 ----- I-GY
 A.UG.92UG037 ----- I-GY
 A.UG.U455 ----- I-GY

CONSENSUS_B -----?I-gY
 B.-.NL43E9 -H----- I-GY
 B.AU.MBC18 ----- I-GY
 B.AU.MBC200 ----- I-DY
 B.AU.MBC925 ----- I-GY
 B.AU.MBCC54 -----Y---- I-GY
 B.AU.MBCC98 -----I---- I-GY
 B.AU.MBCD36 ----- I-GY
 B.CN.RL42 ----- I-GY
 B.DE.D31 ----- I-GY
 B.DE.HAN ----- I-GY
 B.FR.HXB2 ----- I-GY
 B.GA.OYI ----- I-GY
 B.GB.CAM1 ----- I-GY
 B.GB.MANC ----- I-GY
 B.NL.3202A21 ----- I-GY
 B.TW.LM49 -----#I-GY
 B.US.AD8 ----- I-GY
 B.US.BC ----- I-GY
 B.US.DH123 ----- I-GY
 B.US.JRCSF ----- I-GY
 B.US.JRFL ----- I-GY
 B.US.MNCG ----- I-GY
 B.US.NY5CG ----- I-GY
 B.US.P896 ----- I-GY
 B.US.RF ----- I-GY
 B.US.SF2 ----- I-GY
 B.US.WEAU160 ----- I-GY
 B.US.WR27 ----- I-GY
 B.US.YU2 ----- I-GY

CONSENSUS_C ----- I-GY
 C.BR.92BR025 -----#-- I-GY
 C.BW.96BW01B03 -----R-- I-GY
 C.BW.96BW0402 ----- I-GY
 C.BW.96BW0502 ----- I-GY
 C.BW.96BW1104 ----- I-GY
 C.BW.96BW1210 ----- I-GY
 C.BW.96BW15B03 ----- I-GY
 C.BW.96BW1626 ----- I-GY
 C.BW.96BW17A09 -----L-- I-GY
 C.ET.ETH2220 -----R-- I-GY
 C.IN.21068 ----- I-GY

C.IN.301904 ----- I-GY
 C.IN.301905 ----- I-GY
 C.IN.301999 --V-----R-- I-GY
 C.IN.94IN11246 ----- I-GY

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

CONSENSUS_F1 ----- I-GY
 F1.BE.VI850 ----- I-GY
 F1.BR.93BR020.1 ----- I-GY
 F1.FI.FIN9363 ----- I-GY
 F1.FR.MP411 ----- I-GY

CONSENSUS_F2 ----- I-GY
 F2.CM.MP255 ----- I-GY
 F2.CM.MP257 ----- I-GY

CONSENSUS_G ----- I-GY
 G.BE.DRCBL ----- I-GY
 G.FI.HH8793 ----- I-GY
 G.NG.92NG083 ----- I-GY
 G.SE.SE6165 ----- I-GY

CONSENSUS_H ----- I-GY
 H.BE.VI991 ----- I-GY
 H.BE.VI997 ----- I-GY
 H.CF.90CF056 ----- I-GY

CONSENSUS_J ----- I-GY
 J.SE.SE9173 ----- I-GY
 J.SE.SE9280 ----- I-GY

CONSENSUS_K ----- I-GY
 K.CD.EQTB11C ----- I-GY
 K.CM.MP535 ----- I-GY
 N.CM.YBF30 ----- I-GY

CONSENSUS_O -----V----- I-GY
 O.CM.ANT70C -----V----- I-GY
 O.CM.MVP5180 -----V----- I-GY
 AC.ET.E3099G ----- I-GY
 AC.IN.21301 ----- I-GY
 AC.RW.92RW009 ----- I-GY
 AC.SE.SE9488 ----- I-GY
 AC.ZM.ZAM184 ----- I-GY
 ACD.SE.SE8603 ----- I-GY
 AD.SE.SE6954 ----- I-GY
 AD.SE.SE7108 ----- I-GY
 ADU.CD.MAL ----- I-GY
 AG.NG.G3 ----- I-GY
 AG.SE.SE7812 -----L-----R-- I-GY
 AGHU.GA.VI354 ----- I-GY
 AGHU.NO.NOGIL3 x----- I-GY

AGJ.AU.BFP90 ----- I-GY
 AGJ.ML.95ML8 ----- I-GY
 AGU.CD.Z321 ----- I-GY
 BF.BR.93BR029.4 --T----- I-GY
 CRF01_AE.CF.90CF40 ----- I-EY
 CRF01_AE.TH.93TH25 ----- I-GY
 CRF01_AE.TH.CM240 ----- I-GY
 CRF01_AE.TH.TH022 ----- I-GY
 CRF01_AE.TH.TH047 ----- I-GY
 CRF02_AG.FR.DJ263 ----- I-GY
 CRF02_AG.FR.DJ264 ----- I-GY
 CRF02_AG.NG.IBNG ----- I-GY
 CRF03_AB.RU.KAL153 ----- I-GY
 CRF04_CPX.CY.94CY0 ----- I-GY
 CRF04_CPX.GR.97PVC ----- I-GY
 CRF04_CPX.GR.97PVM ----- IEGY
 DF.CD.VI961 ----- I-GY
 U.CD.VI1126 ----- I-GY

CONSENSUS_CPZ ----- I-GY
 CPZ.CD.CPZANT --V--H----- I-GY
 CPZ.GA.CPZGAB ----- I-GY
 CPZ.US.CPZUS ----- I-GY

SPAIFQSSMTKILEPFRKQN

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

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

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

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

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

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

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

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

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

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

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

 CONSENSUS_O -----D---??-
 O.CM.ANT70C -----D---RD-
 O.CM.MVP5180 -----D---S-
 AC.ET.E3099G -----E-----TK-
 AC.IN.21301 -----A-----A--
 AC.RW.92RW009 -----N-----A--
 AC.SE.SE9488 -----A--S-
 AC.ZM.ZAM184 --S-----D---SK-
 ACD.SE.SE8603 -----L-----SK-
 AD.SE.SE6954 -----
 AD.SE.SE7108 -----SK-
 ADU.CD.MAL -----TK-
 AG.NG.G3 -----TE-
 AG.SE.SE7812 -----A-----TK-
 AGHU.GA.VI354 -----
 AGHU.NO.NOGIL3 -----C-----AK-

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

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

TPKFKLPIQKETWETWWTEY

QUERY **TPKFKLPIQKETWETWWTEY**
 CONSENSUS_A -----md-
 A.KE.Q23-CXC-CG ----R-----D---MD-
 A.SE.SE6594 -----MD-
 A.SE.SE7253 -----MD-
 A.SE.SE7535 --RS-----ID-
 A.SE.SE8131 -----D-D---MD-
 A.SE.SE8538 -----V-----M--
 A.SE.SE8891 -----MD-
 A.UG.92UG037 -----MD-
 A.UG.U455 I---R-----A--M--
 CONSENSUS_B -----a-----
 B.-.NL43E9 -----A-----
 B.AU.MBC18 ----R-----DA-----
 B.AU.MBC200 ----R-----A-----
 B.AU.MBC925 I-----A-----
 B.AU.MBCC54 -----R---A-----
 B.AU.MBCC98 -----A-----
 B.AU.MBCD36 -----A---K-----
 B.CN.RL42 -----A-----
 B.DE.D31 -----A-----
 B.DE.HAN ----R-----A-----
 B.FR.HXB2 -----A-----
 B.GA.OYI -----A-----
 B.GB.CAM1 -----DA--ID-
 B.GB.MANC I-----DA-----
 B.NL.3202A21 -----A-----
 B.TW.LM49 -----A-----
 B.US.AD8 -----A--M--
 B.US.BC ----R-----A-----
 B.US.DH123 ----R-----A-----
 B.US.JRCSF I-----A-----
 B.US.JRFL I-----A-----
 B.US.MNCG ----R-----A-----
 B.US.NY5CG -----A-----
 B.US.P896 -----A--D-----
 B.US.RF -----A-----
 B.US.SF2 I-----A--M--
 B.US.WEAU160 -----A-----
 B.US.WR27 --xI-----SR-----
 B.US.YU2 -----A-----
 CONSENSUS_C ----R-----d-----
 C.BR.92BR025 ----R-----A--D-----
 C.BW.96BW01B03 #---R-----D-----
 C.BW.96BW0402 ----R-----D-----
 C.BW.96BW0502 ----R-----D-----
 C.BW.96BW1104 I---R-----A--A--D-----
 C.BW.96BW1210 ----R-----A--D-----
 C.BW.96BW15B03 ----R-----A-----
 C.BW.96BW1626 ----R-----D-----
 C.BW.96BW17A09 ----R-----D-----
 C.ET.ETH2220 ----R-----A--D-----
 C.IN.21068 ----R-----D-----

C.IN.301904 ----R-----D-----
 C.IN.301905 ----R-----D-----
 C.IN.301999 ----R-----A--D-----
 C.IN.94IN11246 ----R-----D-----
 CONSENSUS_D ----r-----i-----
 D.CD.84ZR085 ----R-----ID-----
 D.CD.ELI ----R-----A-----
 D.CD.NDK ----R-----I-----
 D.CD.Z2Z6 ----R-----V-----
 D.UG.94UG1141 ----R-----A-----
 CONSENSUS_F1 ----?---l---d---?---
 F1.BE.VI850 S-----L---D---D---
 F1.BR.93BR020.1 ----R---L---D---
 F1.FI.FIN9363 ----L---D---
 F1.FR.MP411 S---R-----A--D-----
 CONSENSUS_F2 ?---R-----I-----
 F2.CM.MP255 I---R-----I-----
 F2.CM.MP257 V---R-----I-----
 CONSENSUS_G ?-----r---v-----
 G.BE.DRCBL I-----K---V-----
 G.FI.HH8793 ----R---V-----
 G.NG.92NG083 I-----R---V-----
 G.SE.SE6165 ----R---I---D-----
 CONSENSUS_H I---r-----h-----
 H.BE.VI991 I---R-----H-----
 H.BE.VI997 I-----H-----
 H.CF.90CF056 I---R-----A-----
 CONSENSUS_J ----R---?-----D-----
 J.SE.SE9173 ----R-----D-----
 J.SE.SE9280 ----R---R-----D-----
 CONSENSUS_K ----R-----G-----
 K.CD.EQTB11C ----R-----A-----
 K.CM.MP535 ----R---V---V---A---DH
 N.CM.YBF30 ----R-----A-----
 CONSENSUS_O L---?--VTR-----A?--
 O.CM.ANT70C L-----VTR-----AD-
 O.CM.MVP5180 L---R--VTR-----A--
 AC.ET.E3099G ----R-----D-----
 AC.IN.21301 ----R-----D-----
 AC.RW.92RW009 ----R-----D-----
 AC.SE.SE9488 ----R-----MD-----
 AC.ZM.ZAM184 PK.-R-----D-----
 ACD.SE.SE8603 A-----MD-----
 AD.SE.SE6954 ----R-----A-----
 AD.SE.SE7108 ----R-----A-----
 ADU.CD.MAL ----R-----A-----
 AG.NG.G3 V-----R---V-----
 AG.SE.SE7812 ----R---R---A--M--
 AGHU.GA.VI354 I-----DH-----
 AGHU.NO.NOGIL3 ----R-----D-----

AGJ.AU.BFP90 ----R-----A-----
 AGJ.ML.95ML8 I---R-----AD-----
 AGU.CD.Z321 ----R-----A--I-----
 BF.BR.93BR029.4 I-----A-----
 CRF01_AE.CF.90CF40 ----R---R-----M-----
 CRF01_AE.TH.93TH25 ----R---R-----M-----
 CRF01_AE.TH.CM240 ----R---R-----M-----
 CRF01_AE.TH.TH022 ----R-----M-----
 CRF01_AE.TH.TH047 ----R-----M-----
 CRF02_AG.FR.DJ263 ----R---R---A--M-----
 CRF02_AG.FR.DJ264 ----S---R---A--M-----
 CRF02_AG.NG.IBNG ----R---R-----M-----
 CRF03_AB.RU.KAL153 ----R-----D-----
 CRF04_CPX.CY.94CY0 ----R-----D-----
 CRF04_CPX.GR.97PVC ----R-----D-----
 CRF04_CPX.GR.97PVM ----R-----D-----
 DF.CD.VI961 ----R-----D-----
 U.CD.VI1126 ----R-----D-----
 CONSENSUS_CPZ v---?--v---?--a--s--
 CPZ.CD.CPZANT V---Q---TR---DA---SD-
 CPZ.GA.CPZGAB ----R---V---S---A---A---
 CPZ.US.CPZUS V-----LV---V-----S--

Study Subject ID:01RCH21

Study Subject Clone:

Study Subject HLA:A2,A31,B7,B72,Cw2,Cw7

Sequence: Known reactive 20Mer0: VQMAVFIHNFKRKGGIGGYS Integrase(176-195)

Possible HLA

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*0222
A31 A*3101,A*3104,A*3201,A*3202
B7 B*07,B*0702,B*0703,B*0704,B*0705,B*0706,B*0707,B*0709,B*0711
B72 B*1503,B*1546
Cw2 Cw*0202
Cw7 Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(4-12) AVFIHNFKR A*3101
(5-12) VFIHNFKR A*3101
(3-12) MAVFIHNFKR A*3101
(2-10) QMAVFIHNF Cw*0702
(11-19) KRKGGIGGY Cw*0702
(3-10) MAVFIHNF Cw*0702
(12-19) RKGGIGGY Cw*0702
(1-10) VQMAVFIHNF Cw*0702
(10-19) FKRKGGIGGY Cw*0702

Anchor Residues Searched

A*0201 X[LM]XXXXXX[VL]
A*0201 X[LM]XXXXXX[VL]
A*0201 X[LM]XXXXXXXX[VL]
A*0202 X[L]XXXXXX[LV]
A*0202 X[L]XXXXXX[LV]
A*0202 X[L]XXXXXXXX[LV]
A*0204 X[L]XXXXXX[L]
A*0204 X[L]XXXXXX[L]
A*0204 X[L]XXXXXXXX[L]
A*0205 X[VLMQ]XXXXXX[L]
A*0205 X[VLMQ]XXXXXX[L]
A*0205 X[VLMQ]XXXXXXXX[L]
A*0206 X[V]XXXXXX[V]
A*0206 X[V]XXXXXX[V]
A*0206 X[V]XXXXXXXX[V]
A*0207 X[L][D]XXXXXX[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]XXXXXX[LV]
A*0214 X[VQL]XXXXXX[LV]
A*3101 XXXXXXXX[R]
A*3101 XXXXXXXX[R]
A*3101 XXXXXXXX[R]
B7 X[P]XXXXXX[LF]
B7 X[P]XXXXXX[LF]
B7 X[P]XXXXXX[LF]
B*0702 X[P]XXXXXX[L]
B*0702 X[P]XXXXXX[L]
B*0702 X[P]XXXXXX[L]
B*0703 X[P]XXXXXX[L]
B*0703 X[P]XXXXXX[L]
B*0703 X[P]XXXXXX[L]
B*0705 X[P]XXXXXX[L]
B*0705 X[P]XXXXXX[L]
B*0705 X[P]XXXXXX[L]
Cw*0702 XXXXXXXX[YFL]
Cw*0702 XXXXXXXX[YFL]
Cw*0702 XXXXXXXX[YFL]

Study Subject ID:01RCH21

Study Subject Clone:

Study Subject HLA:A2,A31,B7,B72,Cw2,Cw7

Sequence: Known reactive 20Mer1: SPAIFQSSMTKILEPFRKQN RT(156-175)

Possible HLA

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*0221
A31 A*3101,A*3104,A*3201,A*3202
B7 B*07,B*0702,B*0703,B*0704,B*0705,B*0706,B*0707,B*0709,B*0711
B72 B*1503,B*1546
Cw2 Cw*0202
Cw7 Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(5-13) FQSSMTKIL A*0205
(5-13) FQSSMTKIL A*0214
(9-17) MTKILEPFR A*3101
(10-17) TKILEPFR A*3101
(8-17) SMTKILEPFR A*3101
(5-13) FQSSMTKIL Cw*0702
(8-16) SMTKILEPF Cw*0702
(6-13) QSSMTKIL Cw*0702
(9-16) MTKILEPF Cw*0702
(4-13) IFQSSMTKIL Cw*0702
(7-16) SSMTKILEPF Cw*0702

Anchor Residues Searched

A*0201 X[LM]XXXXXX[VL]
A*0201 X[LM]XXXXXX[VL]
A*0201 X[LM]XXXXXX[VL]
A*0202 X[L]XXXXXX[LV]
A*0202 X[L]XXXXXX[LV]
A*0202 X[L]XXXXXX[LV]
A*0202 X[L]XXXXXX[LV]
A*0204 X[L]XXXXXX[L]
A*0204 X[L]XXXXXX[L]
A*0204 X[L]XXXXXX[L]
A*0204 X[L]XXXXXX[L]
A*0205 X[VLIMQ]XXXXXX[L]
A*0205 X[VLIMQ]XXXXXX[L]
A*0205 X[VLIMQ]XXXXXX[L]
A*0206 X[V]XXXXXX[V]
A*0206 X[V]XXXXXX[V]

A*0206 X[V]XXXXXXXX[V]
A*0207 X[L][D]XXXXXX[L]
A*0207 X[L][D]XXXXX[L]
A*0207 X[L][D]XXXXXXXX[L]
A*0214 X[VQL]XXXXXXXX[LV]
A*0214 X[VQL]XXXXXX[LV]
A*0214 X[VQL]XXXXXXXX[LV]
A*3101 XXXXXXXXX[R]
A*3101 XXXXXXXXX[R]
A*3101 XXXXXXXXX[R]
B7 X[P]XXXXXXX[LF]
B7 X[P]XXXXXX[LF]
B7 X[P]XXXXXXXX[LF]
B*0702 X[P]XXXXXXX[L]
B*0702 X[P]XXXXXX[L]
B*0702 X[P]XXXXXXXX[L]
B*0703 X[P]XXXXXXX[L]
B*0703 X[P]XXXXXX[L]
B*0703 X[P]XXXXXXXX[L]
B*0705 X[P]XXXXXXX[L]
B*0705 X[P]XXXXXX[L]
B*0705 X[P]XXXXXXXX[L]
Cw*0702 XXXXXXXXX[YFL]
Cw*0702 XXXXXXXXX[YFL]
Cw*0702 XXXXXXXXX[YFL]

Study Subject ID:01RCH21

Study Subject Clone:

Study Subject HLA:A2,A31,B7,B72,Cw2,Cw7

Sequence: Known reactive 20Mer2: TPKFKLPIQKETWETWWTEY RT(386-405)

Possible HLA

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*0221,A*0222,A*0223,A*0224,A*0225,A*0226,A*0227,A*0228,A*0229,A*0230,A*0231,A*0232,A*0233,A*0234,A*0235,A*0236,A*0237,A*0238,A*0239,A*0240,A*0241,A*0242,A*0243,A*0244,A*0245,A*0246,A*0247,A*0248,A*0249,A*0250,A*0251,A*0252,A*0253,A*0254,A*0255,A*0256,A*0257,A*0258,A*0259,A*0260,A*0261,A*0262,A*0263,A*0264,A*0265,A*0266,A*0267,A*0268,A*0269,A*0270,A*0271,A*0272,A*0273,A*0274,A*0275,A*0276,A*0277,A*0278,A*0279,A*0280,A*0281,A*0282,A*0283,A*0284,A*0285,A*0286,A*0287,A*0288,A*0289,A*0290,A*0291,A*0292,A*0293,A*0294,A*0295,A*0296,A*0297,A*0298,A*0299,A*0300,A*0301,A*0302,A*0303,A*0304,A*0305,A*0306,A*0307,A*0308,A*0309,A*0310,A*0311,A*0312,A*0313,A*0314,A*0315,A*0316,A*0317,A*0318,A*0319,A*0320,A*0321,A*0322,A*0323,A*0324,A*0325,A*0326,A*0327,A*0328,A*0329,A*0330,A*0331,A*0332,A*0333,A*0334,A*0335,A*0336,A*0337,A*0338,A*0339,A*0340,A*0341,A*0342,A*0343,A*0344,A*0345,A*0346,A*0347,A*0348,A*0349,A*0350,A*0351,A*0352,A*0353,A*0354,A*0355,A*0356,A*0357,A*0358,A*0359,A*0360,A*0361,A*0362,A*0363,A*0364,A*0365,A*0366,A*0367,A*0368,A*0369,A*0370,A*0371,A*0372,A*0373,A*0374,A*0375,A*0376,A*0377,A*0378,A*0379,A*0380,A*0381,A*0382,A*0383,A*0384,A*0385,A*0386,A*0387,A*0388,A*0389,A*0390,A*0391,A*0392,A*0393,A*0394,A*0395,A*0396,A*0397,A*0398,A*0399,A*0400

A31 A*3101,A*3104,A*3201,A*3202

B7 B*07,B*0702,B*0703,B*0704,B*0705,B*0706,B*0707,B*0709,B*0711

B72 B*1503,B*1546

Cw2 Cw*0202

Cw7 Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(12-20) TWETWWTEY Cw*0702
(13-20) WETWWTEY Cw*0702
(11-20) ETWETWWTEY Cw*0702

Anchor Residues Searched

A*0201 X[LM]XXXXXX[VL]
A*0201 X[LM]XXXXXX[VL]
A*0201 X[LM]XXXXXXXX[VL]
A*0202 X[L]XXXXXX[LV]
A*0202 X[L]XXXXXX[LV]
A*0202 X[L]XXXXXXXX[LV]
A*0204 X[L]XXXXXX[L]
A*0204 X[L]XXXXXX[L]
A*0204 X[L]XXXXXXXX[L]
A*0205 X[VLIMQ]XXXXXX[L]
A*0205 X[VLIMQ]XXXXXX[L]
A*0205 X[VLIMQ]XXXXXXXX[L]
A*0206 X[V]XXXXXX[V]
A*0206 X[V]XXXXXX[V]
A*0206 X[V]XXXXXXXX[V]
A*0207 X[L][D]XXXXXX[L]
A*0207 X[L][D]XXXXXX[L]
A*0207 X[L][D]XXXXXXXX[L]
A*0214 X[VQL]XXXXXX[LV]
A*0214 X[VQL]XXXXXX[LV]
A*0214 X[VQL]XXXXXXXX[LV]
A*3101 XXXXXXXX[R]

A*3101 XXXXXXX[R]
A*3101 XXXXXXX[R]
B7 X[P]XXXXXX[LF]
B7 X[P]XXXXXX[LF]
B7 X[P]XXXXXX[LF]
B*0702 X[P]XXXXXX[L]
B*0702 X[P]XXXXXX[L]
B*0702 X[P]XXXXXX[L]
B*0703 X[P]XXXXXX[L]
B*0703 X[P]XXXXXX[L]
B*0703 X[P]XXXXXX[L]
B*0705 X[P]XXXXXX[L]
B*0705 X[P]XXXXXX[L]
B*0705 X[P]XXXXXX[L]
Cw*0702 XXXXXXX[YFL]
Cw*0702 XXXXXXX[YFL]
Cw*0702 XXXXXXX[YFL]

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

Protein	Epitope in Database	Epitope in Ref. strain	Epitope in Consensus B	HLA	Notes
p17(22–31)	RPGGKKRYKL	RPGGKKKYKL	RPGGKKKYKL	B7	
p17(77–85)	SLFNTVATL	SLYNTVATL	SLYNTVATL	A*0201	
p24(223–231)	GPSHKARVL	GPGHKARVL	GPGHKARVL	B7	
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)	KLTSNTSV	RLISNTSV	RLISNTSV	A2	
gp160(192–200)	TLTSNTSV	RLISNTSV	RLISNTSV	A2	
gp160(192–200)	TLTSNTSV	RLISNTSV	RLISNTSV	A2.1	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B*07	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B*0702	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B7	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B7?	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B7	
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(419–427)	RIKQIINMW	KIKQIINMW	RIKQIINMW	A*3201	
gp160(700–708)	AVLSVVNRV	AVLSIVNRV	AVLSIVNRV	A2	
gp160(747–755)	RLVNGSLAL	RLVHGFLAI	RLVDGFLAL	A2	
gp160(770–778)	RLRDLLIV	HHRDLLIA	RLRDLLIV	A*0201	
gp160(770–780)	RLRDLLIVTR	HHRDLLIAAR	RLRDLLIVTR	A*3101	
gp160(770–780)	RLRDLLIVTR	HHRDLLIAAR	RLRDLLIVTR	A31	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A*0201	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2.1	
gp160(814–822)	LLNATDIAV	LLNATAIAV	LLNATAIAV	A2	
gp160(843–851)	IPRRIRQGL	IPTRIRQGL	IPRRIRQGL	B*0702	
gp160(843–851)	IPRRIRQGL	IPTRIRQGL	IPRRIRQGL	B7	
Nef(77–85)	RPMTYKAAL	RPMTYKAAV	RPMTYKAAV	B*0702	

Nef(136-145)	PLTFGWCFKL	PLTFGWCYKL	PLTFGWCFKL	A2
Nef(175-184)	DPEKEVLQWK	DPEREVLEWR	DPEKEVLVWK	B7

Table 1: **p17**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(22–31)	Gag(22–31)	RPGGKKRYKL	HIV-1 infection	human(B7)	[Jin (2000)]
	<ul style="list-style-type: none"> • This B7 epitope is one of three subdominant CTL responses detected in a long-term non-progressor • A dominant B7 epitope was defined using conventional methods, and three additional sub-dominant HLA B7 epitopes were defined by first using a non-anchor based strategy, EpiMatrix, to identify 2078 possible epitopes in the autologous HIV-1, followed by B7 anchor residue prediction to narrow the set to 55 peptides for experimental testing 				
p17(77–85)	p17(77–85 Clade A)	SLFNTVATL	HIV-1 infection	human(A*0201)	[Dorrell (1999)]
	<ul style="list-style-type: none"> • Epitope SL9: CTL responses in three individuals with non-clade B infections were studied, 2 with subtype A infections, 1 with subtype C – their infections all originated in East Africa • This epitope is most commonly SLYNTVATL in B subtype, and CTL from the C subtype infection did not recognize B clade gag or the 3Y form of the epitope, but do recognize the predominant A and C clade form, SLFNTVATL 				

Table 2: **p24**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(223–231)	p24()	GPSHKARVL	HIV-1 infection	human(B7)	[Goulder (2000)]
	<ul style="list-style-type: none"> • The CTL-dominant response was focused on this epitope in a HIV+ Caucasian living in Boston – this epitope did not fall within the three most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLLK(p17 16-30) contained the dominant Gag-specific 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), SALSEGATPQDLNMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-specific epitopes in 32 out of 37 C-clade infected subjects from South Africa 				

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

Table 4: **gp160**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(121–129)	gp120(121–129)	KLTPLCVSL	<i>in vitro</i> stimulation	human(A2)	[Zarling (1999)]
					<ul style="list-style-type: none"> • This study compares the ability of macrophages and dendritic cells to stimulate primary responses in CD8+ lymphocytes isolated from HLA-appropriate HIV-uninfected donors using peptide-pulsed APC – the dendritic cells performed better as APC for the stimulation of primary responses • Strong CTL responses were elicited by the epitopes DRFYKTLRA and GEIYKRWII when presented by either immature or mature dendritic cells – macrophages were not able to prime a CTL response against DRFYKTLRA • A weak response to KLTPLCVSL was stimulated using macrophages as the APC • No detectable response was observed for the following previously-defined HIV epitopes: KIRLRPGGK, ILKEPVHGV, IRLRPGGK, GPKVKQWPL
gp160(192–200)	gp120(192–199 HXB2R)	KLTSNTSV	HIV-1 infection	human(A2)	[Brander (1995)]
					<ul style="list-style-type: none"> • 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)]
					<ul style="list-style-type: none"> • Crystallization of HLA-A2 molecules complexed with antigenic peptides – refers to Dadaglio <i>et al</i> 1991
gp160(192–200)	gp120(199–207)	TLTSCNTSV	peptide immunization and HIV-1 infection	human(A2.1)	[Brander (1996)]
					<ul style="list-style-type: none"> • 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(298–307)	gp120(298–307)	RPNNNTRKSI	HIV-1 infection	human(B*07)	[Ferris (1999), Hammond (1995)]
					<ul style="list-style-type: none"> • The processing of this epitope is TAP1/2-dependent, as are most Env epitopes, and it contains an N-linked glycosylation site that is glycosylated in Env • Peptide that had been deglycosylated, a process that changes asparagine (N) to aspartic acid (D) (RPNDNTRKSI) was recognized a 100-fold more efficiently than either glycosylated or non-glycosylated RPNNNTRKSI • Position 5 is not involved with HLA B*07 binding, so is probably important for TCR recognition • HIV-1 Env epitopes are typically processed by a TAP1/2 dependent mechanism, which involves cotranslational translocation into the ER, glycosylation, export back into the cytosol, and deglycosylation for processing, and retransport into the ER for the association with class I molecules • The particular pathway of generating an epitope may have an impact on the presentation of that epitope, quantitatively as well as qualitatively

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(298–307)	gp120(302–312 HXB2) • C. Brander notes this is a B*0702 epitope	RPNNNTRKSI	HIV-1 infection	human(B*0702)	[Brander & Goulder(2001)]
gp160(298–307)	gp120(302–312 HXB2) • CTL from two acute seroconversion cases	RPNNNTRKSI	HIV-1 infection	human(B7)	[Safrit (1994b)]
gp160(298–307)	gp120(303–312 IIIB) • Epitope defined in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • RPNNNTRKDI and RPNNNTRKGI, naturally occurring variants, were found in non-transmitting mother – ability to recognize these variants has not yet been determined	RPNNNTRKSI	HIV-1 infection	human(B7?)	[Wilson (1996)]
gp160(298–307)	gp120(302–311 Clade B) • The extent of CTL interclade cross-reactivity from CTL isolated from individuals newly infected with B clade virus was studied, and extensive cross-reactivity was observed • Two HLA B7 individuals had CTL response to B_LAI, A_92UG037 and C_92BR025 gp160, but were B clade strain MN non-responders – the authors note that the B7 epitope RPNNNTRKSI is immunodominant, conserved between the LAI and clade A and C strains, but is very divergent in MN (RPNYNKRKRI), and that this epitope might be dominating the specificity of the response in the HLA B7 individuals	RPNNNTRKSI	HIV-1 infection	human(B7)	[Wilson (1998)]
gp160(311–320)	gp160(318–327 IIIB) • This immunogenic peptide does not have the known binding motif for A2.1 • The same optimal peptide for this human HLA-A2.1 epitope was observed for a murine H-2 D ^d epitope	RGPGRAFVTI	CTL line from HIV- donor	human(A*0201)	[Alexander-Miller (1996)]
gp160(311–320)	gp160(318–327 IIIB) • Individual was immunized with rec vaccinia gp160 IIIB and boosted with purified gp160 • Lysis only occurs with IIIB P18 peptide pulsed onto autologous targets; MN, RF, SIMI P18 peptides fail to stimulate CTL • Restimulating immune cells from gp160 IIIB vaccinees with MN, RF, or SIMI P18 did not enhance the MN, RF, or SIMI specific CTL response	RGPGRAFVTI	vaccinia IIIB gp160	human(A2)	[Achour (1996)]

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(311–320)	gp160(318–327 SIMI)	MGPKRAFYAT	vaccinia SIMI gp160	human(A2)	[Achour (1996)]
					<ul style="list-style-type: none"> • Individual was immunized with rec vaccinia gp160 SIMI and boosted with purified recombinant gp160 SIMI • P18 MN and RF peptides were able to stimulate the HIV-specific CTL that arose in response to the SIMI vaccination, thus the P18 MN peptide (IGPGRAFYTT) and the P18 RF peptide (KGPRVIYAT) could cross-react • The P18 IIIB peptide does not cross-react (RGPRAFVTI in the epitope region) • gp160 SIMI primed immune cells could generate a significantly broader specificity when stimulated with P18 MN or P18RF peptides, but not P18 IIIB
gp160(369–375)	gp120(374–380 BRU)	PEIVTHS	HIV-1 infection	human(A2)	[Dadaglio (1991)]
					<ul style="list-style-type: none"> • Defined through blocking CTL activity, and Env deletions
gp160(377–387)	gp120(377–387)	NSGGEFFYSNS		human(A2)	[Hickling (1990)]
					<ul style="list-style-type: none"> • Peptides recognized by class I restricted CTL can bind to class II
gp160(419–427)	gp120(424–432 HXB2)	RIKQIINMW		human(A*3201)	[Harrer (1996)]
					<ul style="list-style-type: none"> • C. Brander notes that this is an A*3201 epitope in the 1999 database
gp160(700–708)	gp41(705–714)	AVLSVVNRV	HIV-1 infection	human(A2)	[Ferris (1999)]
					<ul style="list-style-type: none"> • This epitope is processed by a TAP1/2 dependent mechanism
gp160(747–755)	gp41(747–755)	RLVNGSLAL	HIV-1 infection	human(A2)	[Parker (1992)]
					<ul style="list-style-type: none"> • Studied in the context of HLA-A2 peptide binding
gp160(770–778)	Env(679–777)	RLRDLLLIV	HIV-1 infection	human(A*0201)	[Kmieciak (1998)]
					<ul style="list-style-type: none"> • CTL responses in six patients to four Env epitopes were studied: D2: LLNATAIAV, 5.3: RLRDLLLIV, D1: KLTPLCVTL, and 4.3: QMHEDIISL – all have A2 anchor residues • The C terminal epitopes (D2 and 5.3) were highly variable and the variability was considered responsible for limited CTL response, while D1 and 4.3, N-terminal epitopes, were much more conserved and gave evidence of high levels of CTL response <i>in vitro</i> • Peptides 5.3 and D2 bound to HLA A*0201 with low affinity and were variable, particularly D2;
gp160(770–780)	gp41(770–780 BH10)	RLRDLLLIVTR	HIV-1 infection	human(A*3101)	[Safrit (1994a), Safrit (1994b)]
					<ul style="list-style-type: none"> • Recognized by CTL derived from acute seroconverter • C. Brander notes that this is an A*3101 epitope in the 1999 database

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(770–780)	gp41(770–780)	RLRDLLLIVTR	HIV-1 infection	human(A31)	[Ferris (1999), Hammond (1995)]
					<ul style="list-style-type: none"> • This epitope is processed by a TAP1/2 dependent mechanism
gp160(813–822)	gp41(814–823 LAI)	SLLNATDIAV	MN rec gp160	human(A*0201)	[Dupuis (1995)]
					<ul style="list-style-type: none"> • Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823 • Noted to be A*0201 in Brander <i>et al.</i>, 1999 database
gp160(813–822)	gp41(814–823)	SLLNATDIAV	HIV-1 infection	human(A2)	[Kundu (1998b)]
					<ul style="list-style-type: none"> • Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-specific CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • SLLNATDIAV is a conserved HLA-A2 epitope included in this study – 4/6 patients had this sequence as their HIV direct sequence, and 3 of these had a detectable CTL response – the other two had either the sequence 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)]
					<ul style="list-style-type: none"> • Ten HIV-1+ HLA A2 asymptomatic individuals were given two courses of HIV-1 MN rgp160 vaccine over a 2 year period • Two hundred and fifty three HIV-1 peptides of 9 or 10 aa possessing the HLA-A2.1 binding motif (Leu at position 2, Val at the C terminus) were identified in gp160, of which 25 had a high or intermediate binding affinity • Eleven peptides were studied that had high HLA-A2 binding affinity – a CTL response was detected to 9/11 peptides in at least 1 individual • CTL responses after reimmunization may include recall responses – only individuals with vaccine cross-reactive sequences prior to vaccination showed detectable CTL responses • CTL to overlapping peptides in this region gave a positive response in the greatest number of patients • ALTERNATIVE EPITOPES: LLNATDIAV and LLNATDIAVA – CTL were induced by vaccine in those that had the sequence SLLNATAIAVA in their own infection, but not in those with: NLLNTAIAVA or NLFNTTAIAVA or SLLNATAITVA
gp160(814–822)	gp41(815–823 LAI)	LLNATDIAV	MN rec gp160	human(A2)	[Dupuis (1995)]
					<ul style="list-style-type: none"> • Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823
gp160(843–851)	gp41(848–856 LAI)	IPRRIRQGL		human(B*0702)	[Brander & Goulder(2001)]
					<ul style="list-style-type: none"> • C. Brander notes this is a B*0702 epitope
gp160(843–851)	gp41(848–856 LAI)	IPRRIRQGL		human(B7)	[Brander & Walker(1995)]
					<ul style="list-style-type: none"> • Epitope defined in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study

Table 5: **Nef**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(77–85)	Nef(77–85 LAI)	RPMTYKAAL	HIV-1 infection	human(B*0702)	[Bauer (1997)]
					<ul style="list-style-type: none"> • Structural constraints on the Nef protein may prevent escape • Noted in Brander 1999, this database, to be B*0702
Nef(136–145)	Nef(136–145)	PLTFGWCFKL	HIV-1 infection	human(A2)	[Durali (1998)]
					<ul style="list-style-type: none"> • Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • Patient B18 had the greatest breadth and diversity of response, and recognized Gag SLYNTVATL and Nef PLTFGWCFKL
Nef(175–184)	Nef(175–184)	DPEKEVLQWK	HIV-1 infection	human(B7)	[Jin (2000)]
					<ul style="list-style-type: none"> • This a B7 epitope, a subdominant CTL response, was defined by an un-conventional approach used to predict epitopes in an HLA B7+ long-term non-progressor • Three additional sub-dominant HLA B7 epitopes were defined using EpiMatrix, a non-anchor based strategy for defining potential epitopes, which highlighted 2078 possible epitopes in the autologous HIV-1 derived from the study subject, followed by B7 anchor residue prediction which narrowed the set to 55 peptides, three of which could serve as functional CTL epitopes

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

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Integrase(179–188)	Integrase(179–188 LAI)	AVFIHNFKRK		human(A*1101)	[Brander & Goulder(2001), Fukada (1999)]
	<ul style="list-style-type: none"> • C. Brander notes this is an A*1101 epitope 				

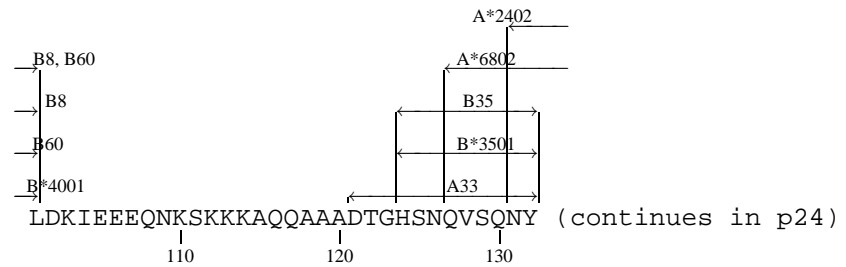
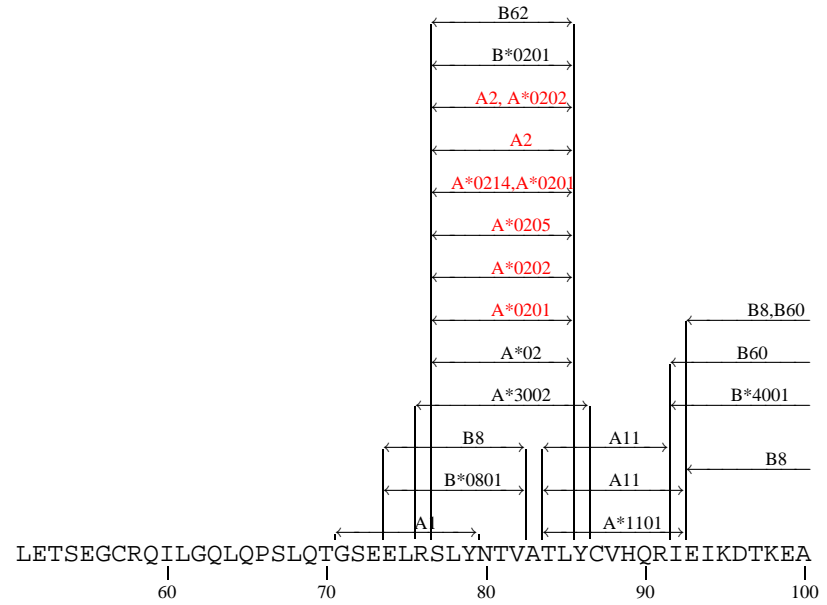
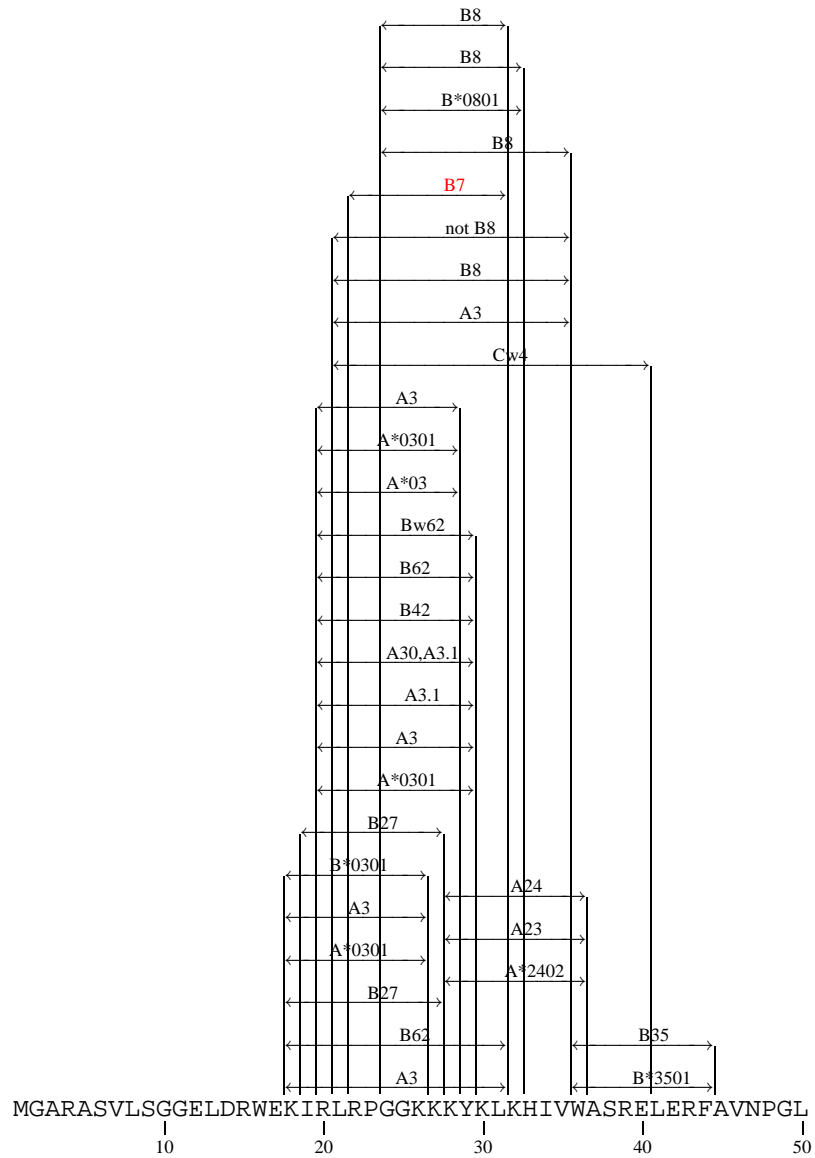
Table 7: **All Defined Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Integrase(179–188)	Integrase(179–188 LAI)	AVFIHNFKRK		human(A*1101)	[Brander & Goulder(2001), Fukada (1999)]
	<ul style="list-style-type: none"> • C. Brander notes this is an A*1101 epitope 				

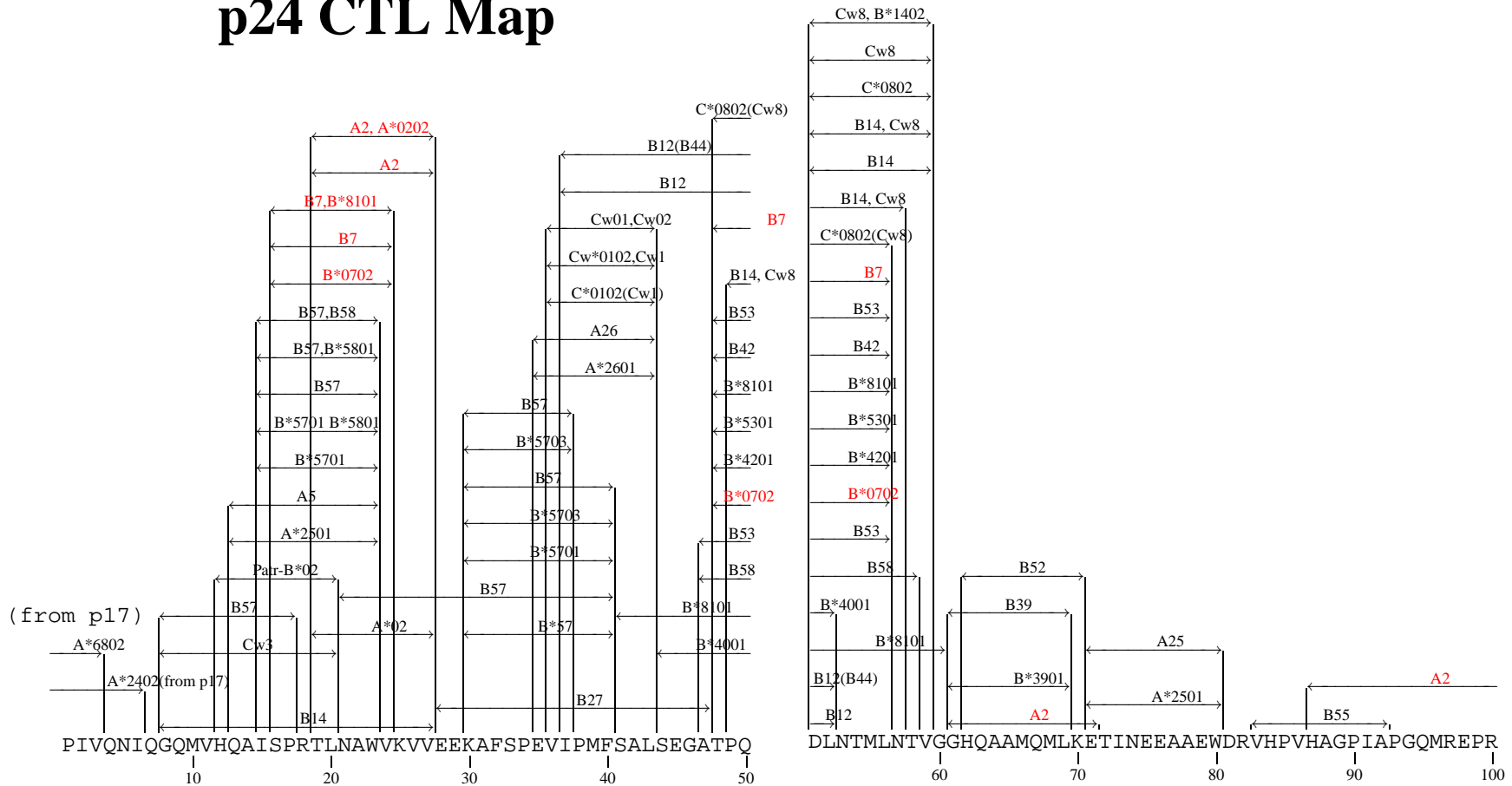
Table 8: **All Defined Epitopes within the 20mer, regardless of HLA type**

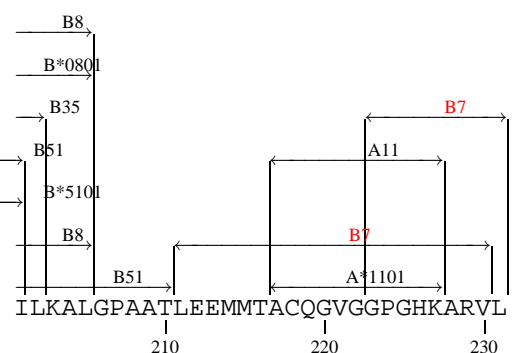
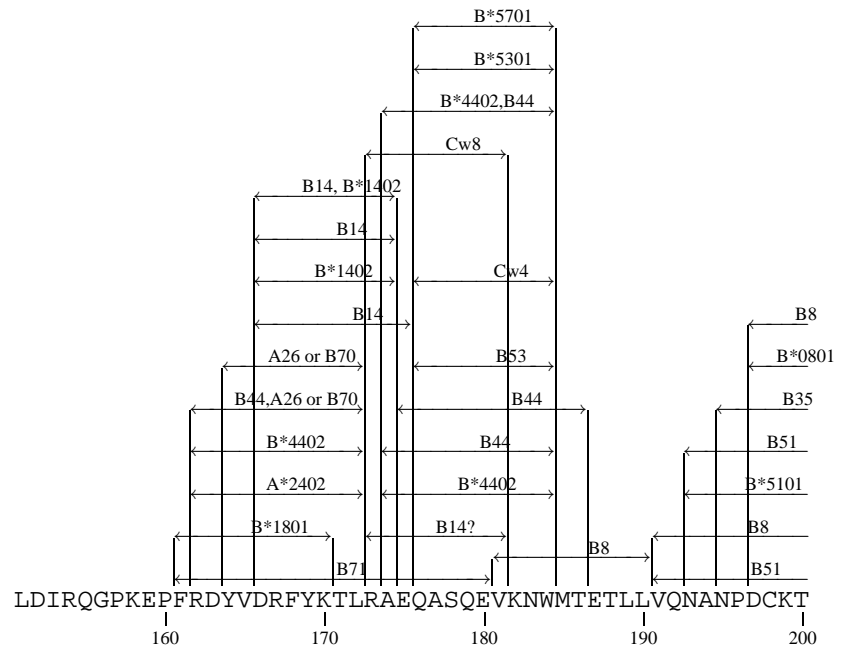
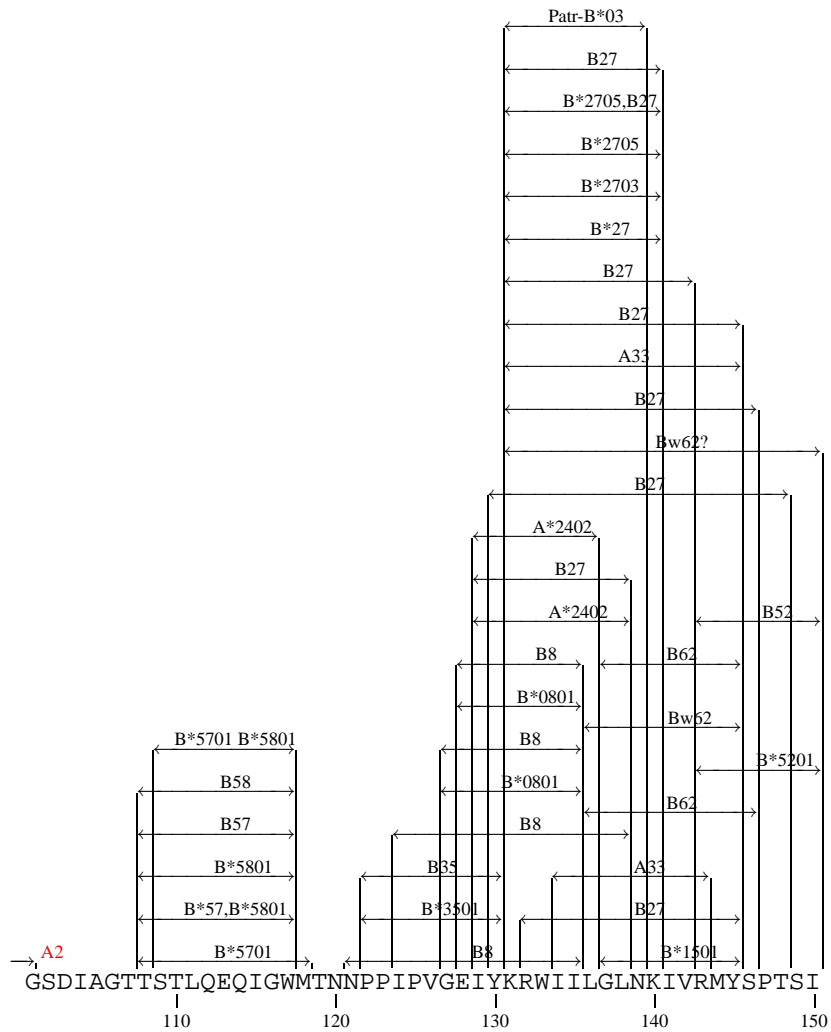
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Integrase(179–188)	Integrase(179–188 LAI)	AVFIHNFKRK		human(A*1101)	[Brander & Goulder(2001), Fukada (1999)]
	<ul style="list-style-type: none"> • C. Brander notes this is an A*1101 epitope 				

p17 CTL Map

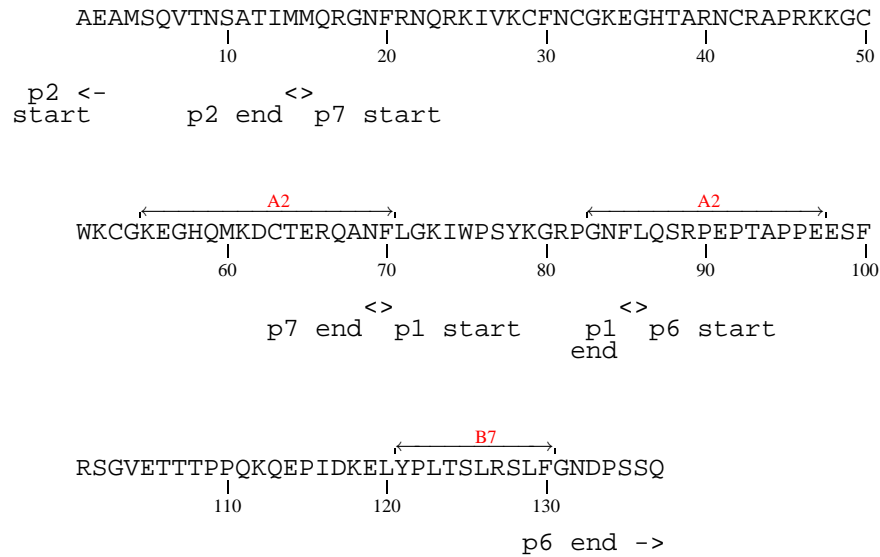


p24 CTL Map





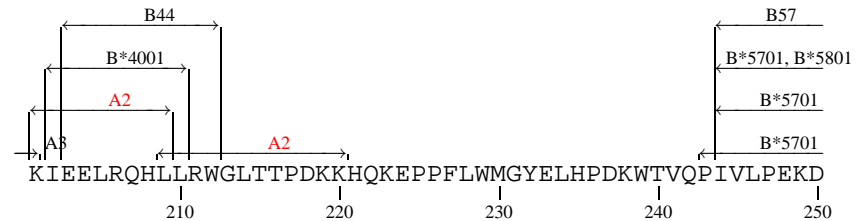
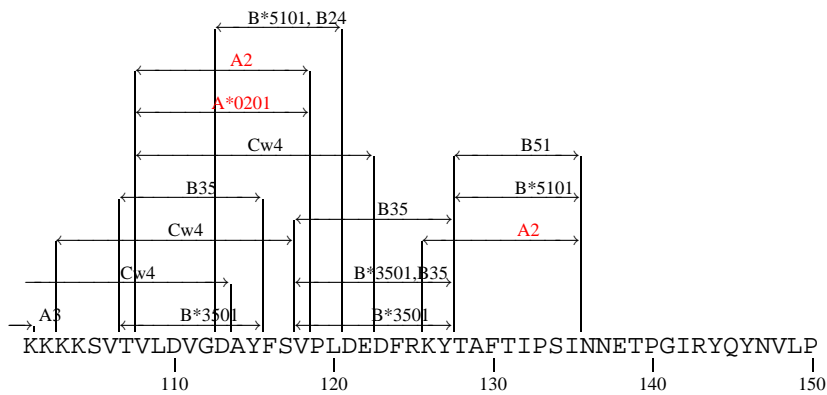
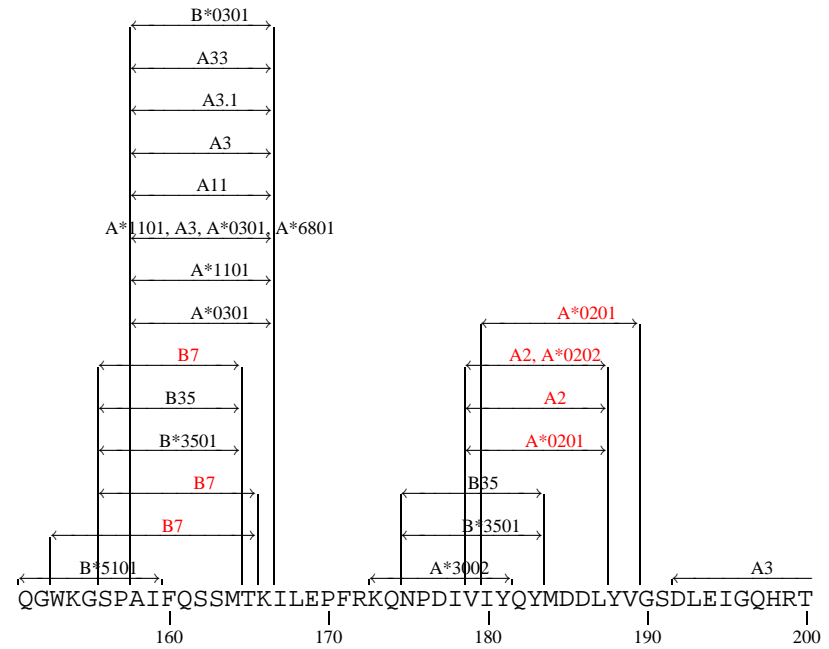
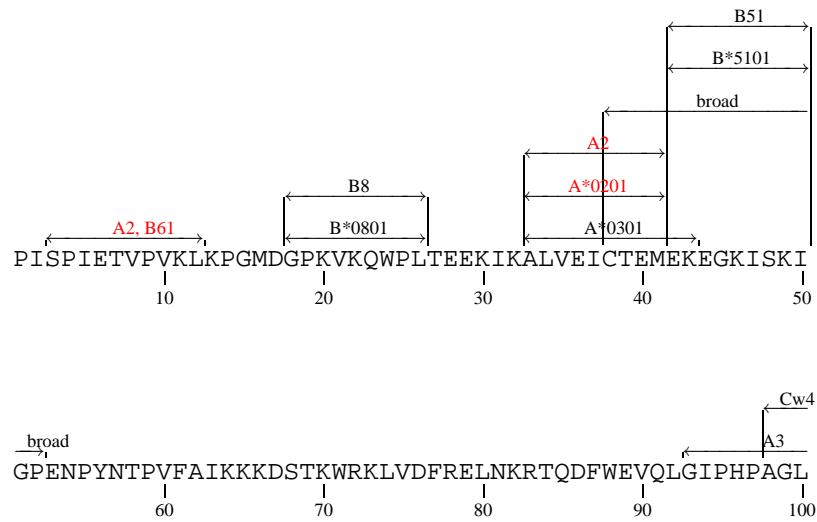
p2p7p1p6 CTL Map

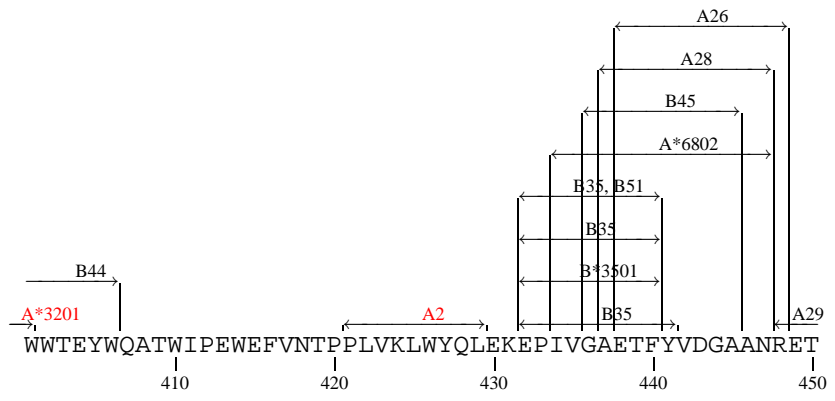
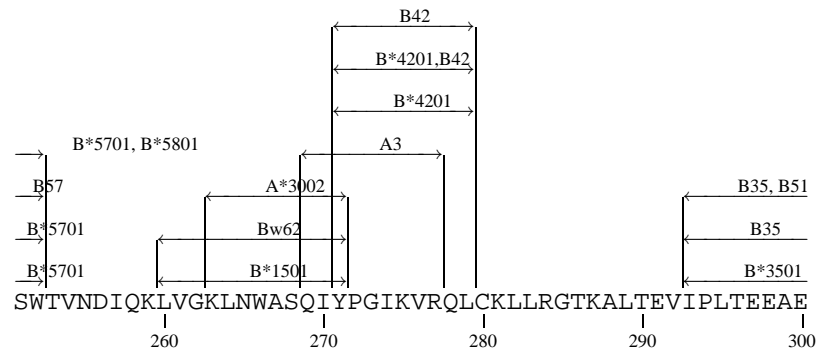


Protease CTL Map

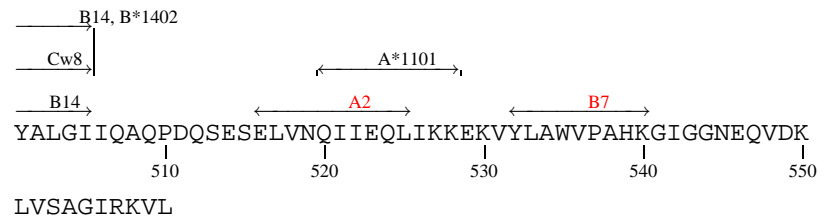
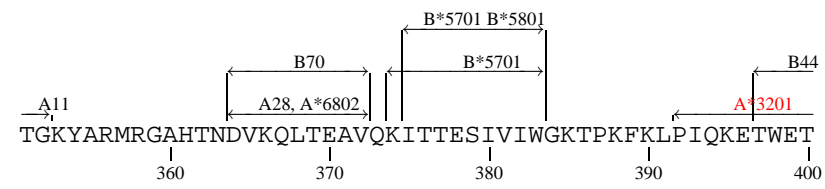
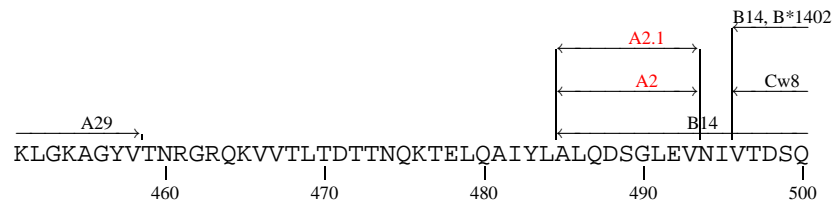
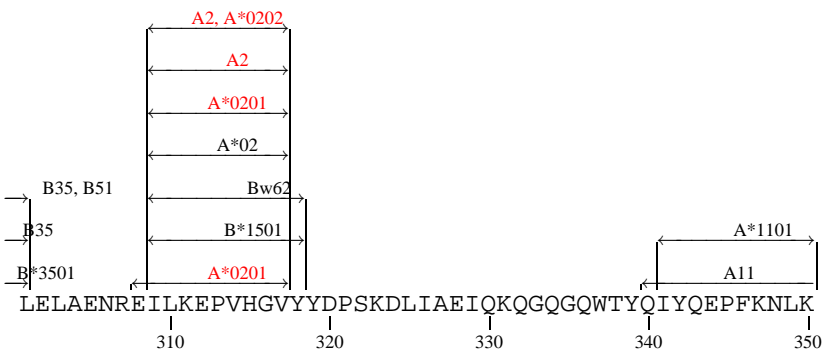


RT CTL Map



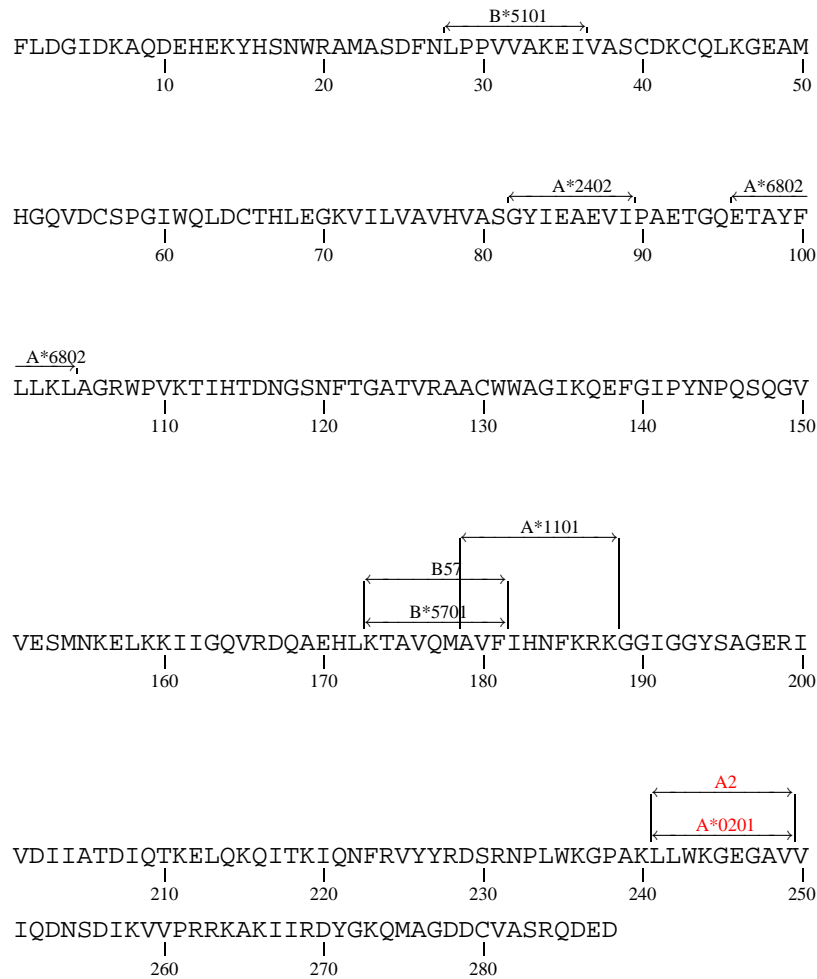


p15 RNase start <-

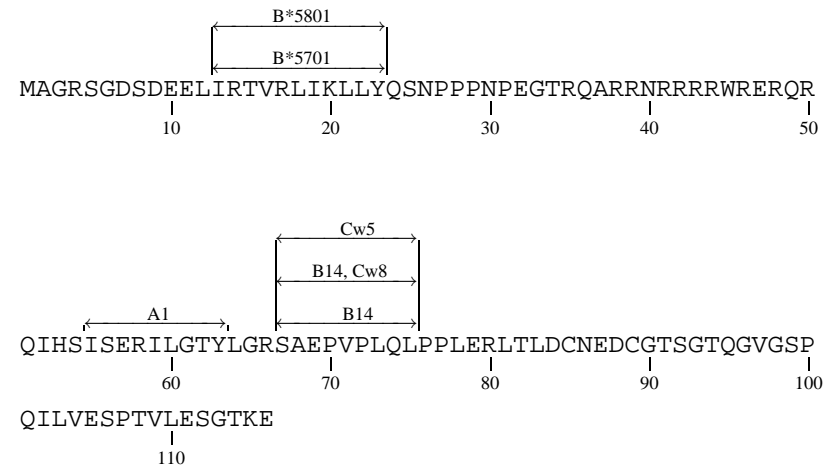


-> p15 RNase end

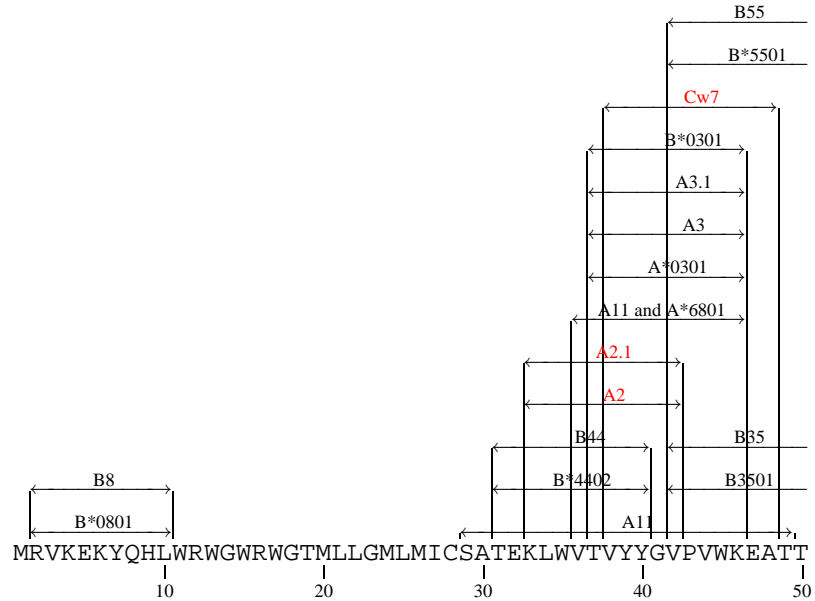
Integrase CTL Map



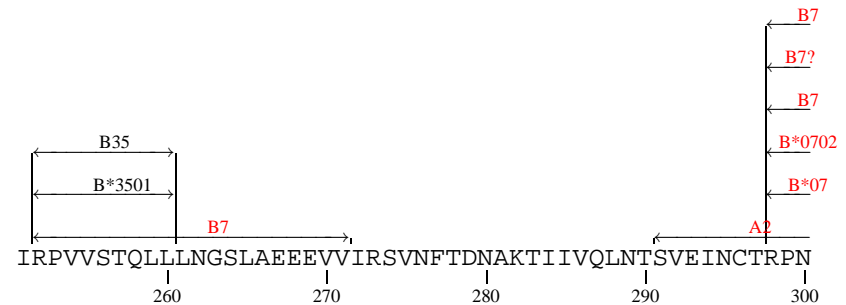
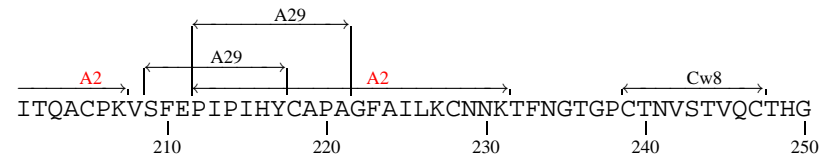
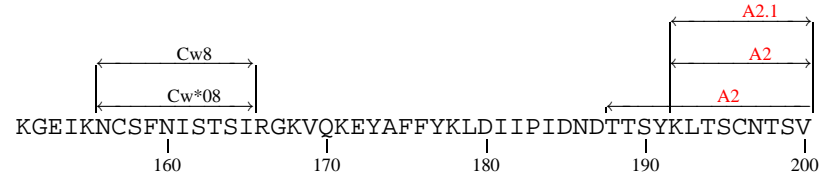
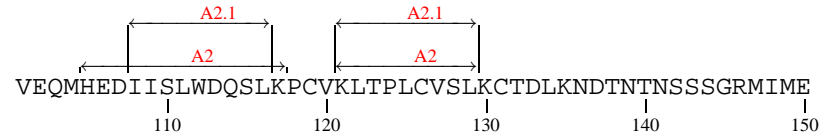
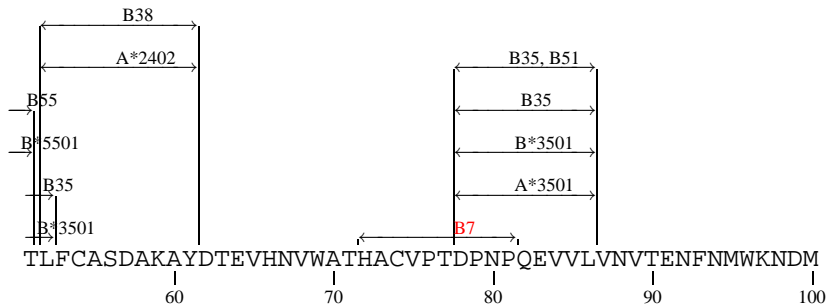
Rev CTL Map

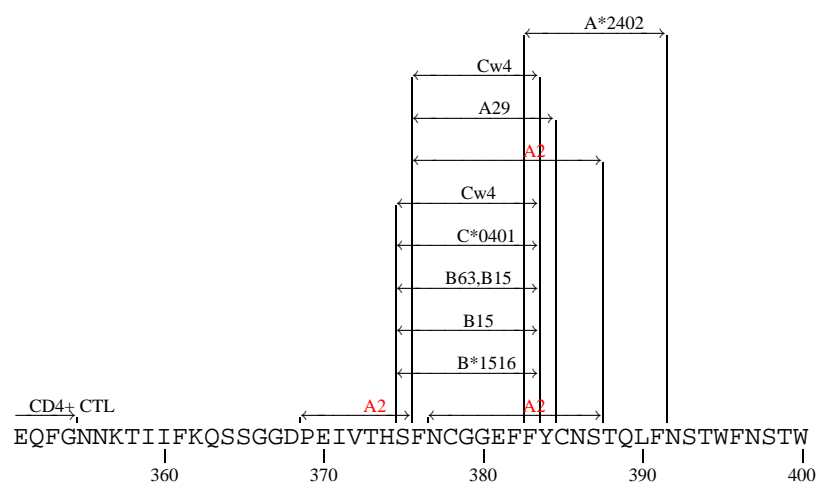
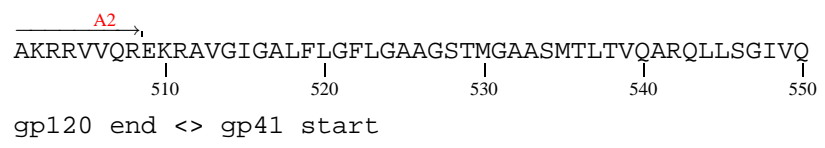
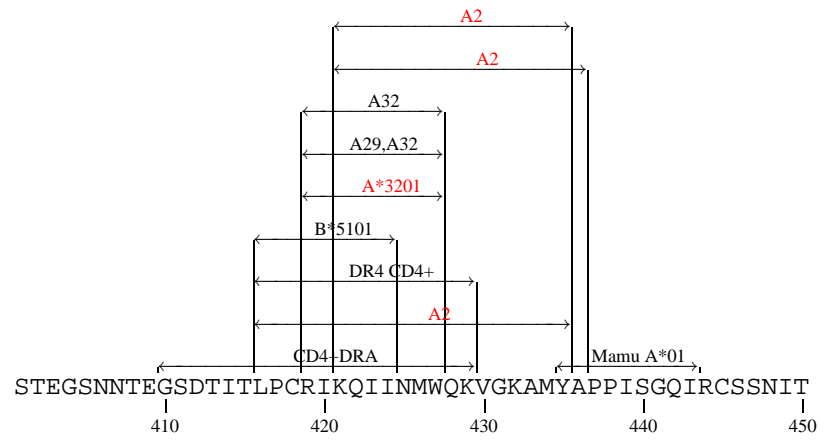
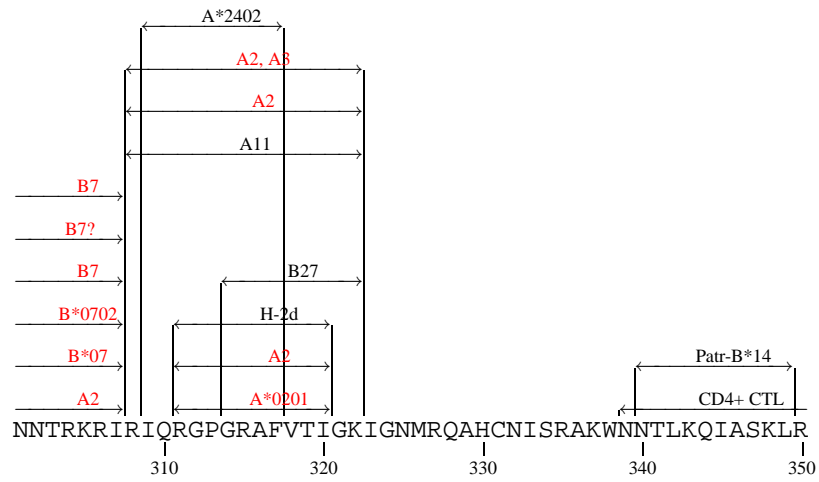


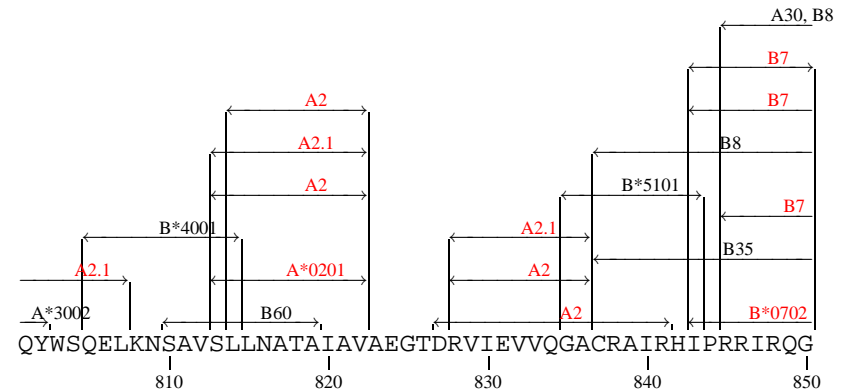
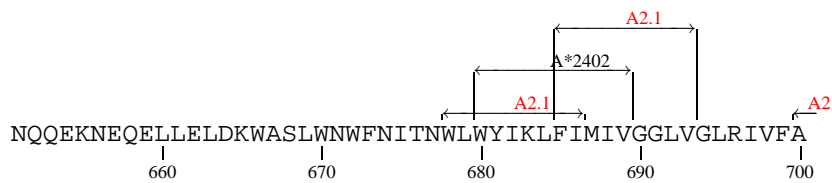
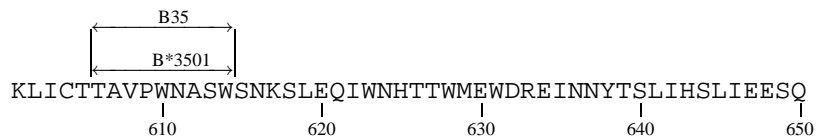
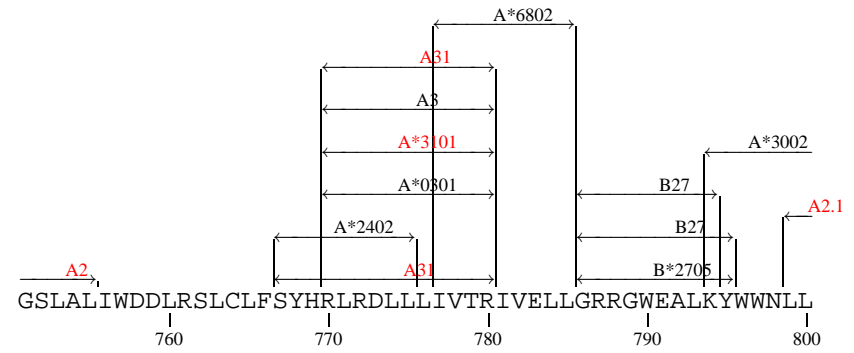
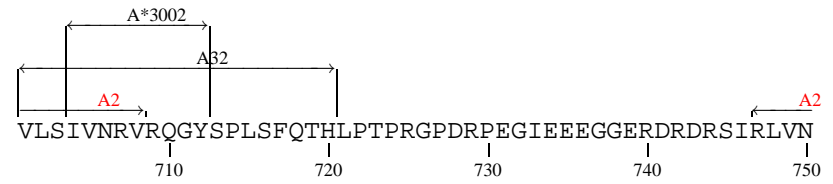
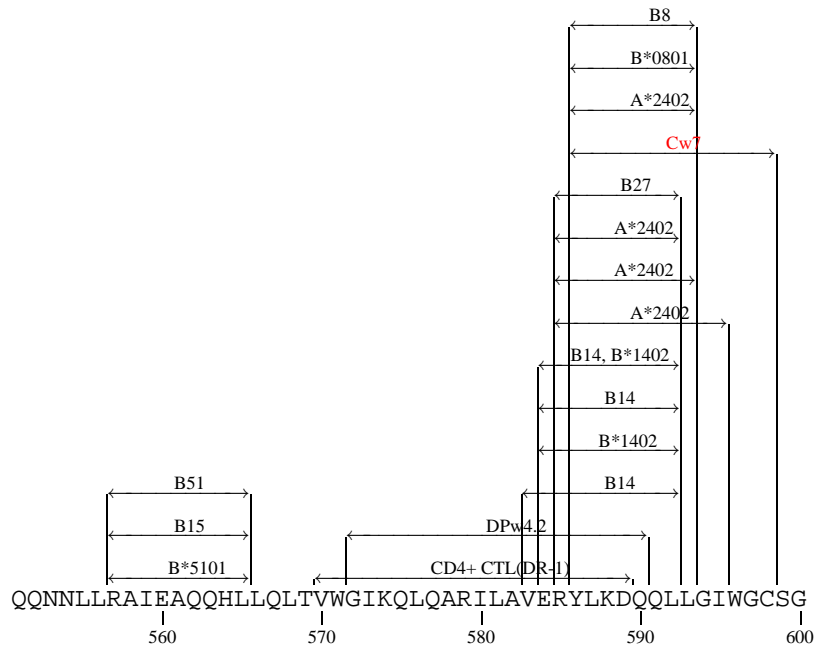
gp160 CTL Map



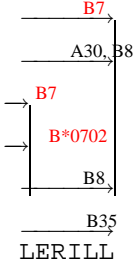
<- gp120 start



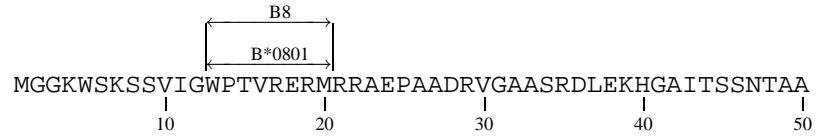


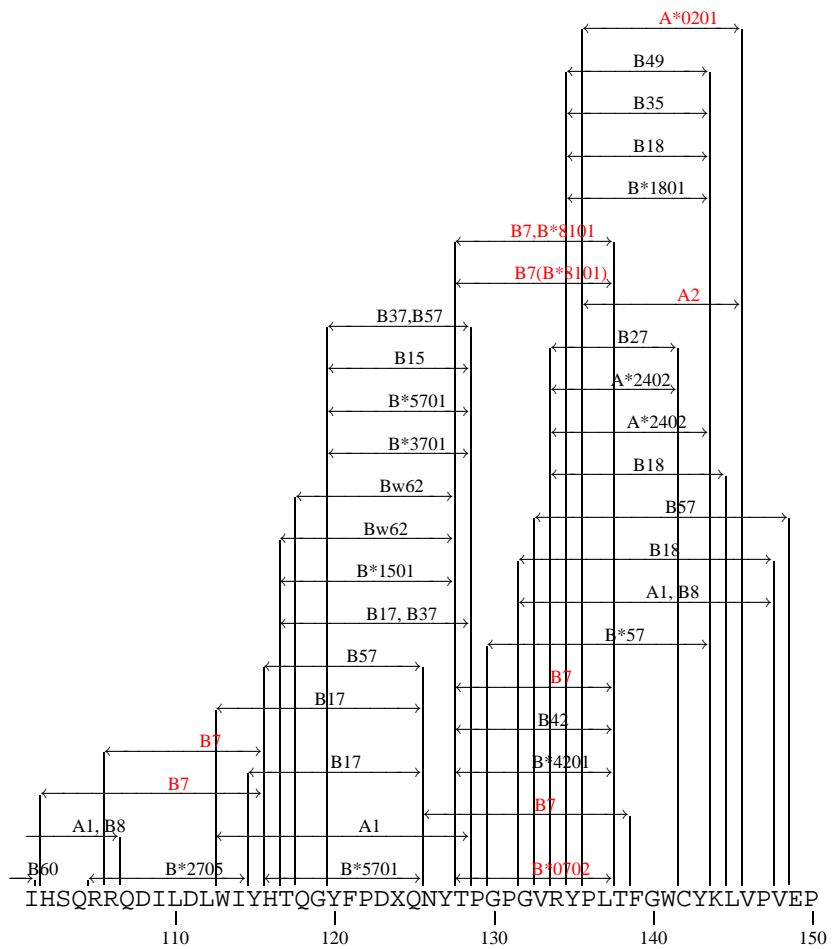


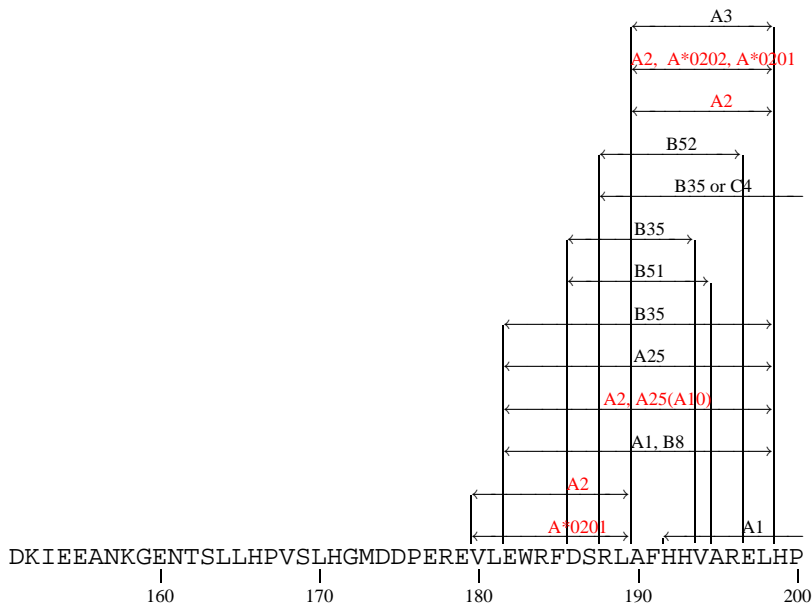
Nef CTL Map



-> gp41 end







$\xrightarrow{A1}$
 EYFKNC

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