Table 1: **p17** 

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(18-26 IIIB)	p17(18-26) <b>NOTES:</b>	KIRLRPGGK	HIV-1 infection	human(A3)	[Walkerpercom96]
	<ul><li>Epitope of study</li><li>KIRLRP</li></ul>	Epitope defined in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transr study  KIRLRPGGR and RIRLRPGGR, naturally occurring variants, were found in mother, and are not recognized	IDS Foundation ARIEL Pring variants, were found	roject, a mother-infant HIV transmission in mother, and are not recognized	ransmission gnized
p17(18-26 IIIB)	p17(18-26) <b>NOTES:</b>	KIRLRPGGK	HIV-1 infection	human(A3)	[Goulder97, Goulder97e]
	<ul><li>Identical</li><li>One had</li><li>[Goulder</li></ul>	Identical twin hemophiliac brothers were both infected with the same batch of factor VIII One had a response to this epitope, the other did not [Goulder97] is a review of immune escape that summarizes this study	fected with the same batcl not ummarizes this study	n of factor VIII	
p17(18-26 LAI)	p17(18-26)	KIRLRPGGK	HIV-1 infection	human(A3.1)	[Harrer96b]
p17(18-27 LAI)	p17(18-27) <b>NOTES:</b> • D. Lewin	(18-27) KIRLRPGGKK TES: D. Lewinsohn, pers. comm.		human(B27)	[Brander96]
p17(18-27)	p17(18-27) <b>NOTES:</b>	KIRLRPGGKK	HIV-1 infection	human(B27)	[Birk98]
	A study of evolution	A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is influenced by immune pressure from CTLs	epitopes and individuals CTLs	with known HLA types rev	ealed that p17
p17(18-31)	p17(18-31) <b>NOTES:</b>	KIRLRPGGKKKYKL	HIV-1 infection	human(B62)	[Lubaki97]
	• 82 HIV-1 response	82 HIV-1-specific CTL clones from 5 long term non-progressors were isolated response	non-progressors were is	olated and analyzed for breadth of CTL	eadth of CTL
	<ul> <li>A sustain a polyclo</li> </ul>	A sustained Gag, Env and Nef response was observed, and clones were restricted by multiple HLA epitopes, indicating a polyclonal response  A subject who was HI A B62+ had CTI that recomined this pentide and 22/1 GI NKINDMYS and one additional	ved, and clones were restri	cted by multiple HLA epitopes, indicating	pes, indicating
	unknown epitope	epitope	0		

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(18-31)	p17(18-31) <b>NOTES:</b> • A study of the study o	7(18-31) KIRLRPGGKKKYKL HIV-1 infection human(A3) [Birk98]  OTES:  • A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17	HIV-1 infection epitopes and individuals	human(A3) with known HLA types re	[Birk98] vealed that p17
p17(18-42 IIIB)	p17(18-42)  NOTES:  • Enitone 1	(18-42) KIRLRPGGKKKYKLKHI- VWASRELE TES: Enitone recognized by CTL clone derived from CSF	HIV-1 infection	human(A3)	[Jassoy92]
p17(18-42 BH10)	p17(18-42) NOTES: • Gag CTI	7(18-42) KIRLRPGGKKKYKLKHI- VWASRELE <b>YTES:</b> Gag CTL response was studied in three individuals	HIV-1 infection	human(Bw62)	[Johnson91]
p17(18-42 PV22)	p17(18-42) NOTES: • HIV-1 sp	(18-42) KIRLRPGGKKKYKLKHI- HI VWASRELE TES: HIV-1 specific CTLs release $\gamma$ -IFN, and $\alpha$ - and $\beta$ -TNF	HIV-1 infection	human(A3)	[Jassoy93]
p17(90-105 SF2)	p17(20-35) NOTES: • Of 25 par • 12 subjec • One of th	(20-35) CLRPGGKKKYKLKHIV HIV-1 infection TES: Of 25 patients, most had CTL specific for more than 1 HIV-1 protein 12 subjects had CTL that could recognize vaccinia expressed LAI gag One of these 12 had CTL response to this peptide The responding subject was HLA A-2, A-24, B-13, B-35	HIV-1 infection han 1 HIV-1 protein ia expressed LAI gag e 13, B-35	human	[Lieberman97]
p17(19-27 LAI)	p17(19-27) <b>NOTES:</b> • D. Lewir	(19-27) IRLRPGGKK  TES:  D. Lewinsohn, pers. comm.		human(B27)	[Brander96]

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(20-29 IIIB)	p17(20-29) <b>NOTES:</b>	RLRPGGKKKY	HIV-1 infection	human(B42)	[Walkerpercom96]
	<ul><li>Epitope of study</li><li>RLRPGO</li><li>Binds HI</li></ul>	Epitope defined in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV to study study RLRPGGKKRY, a naturally occurring variant, was found in non-transmitting mother and is recognized Binds HLA-A3 and Bw62 as well	AIDS Foundation ARIEL was found in non-transmi	Project, a mother-infant HIV transmission ting mother and is recognized	Y transmission zed
p17(20-29)	p17(20-29) <b>NOTES:</b> • Unpublis	7(20-29) RLRPGGKKKY OTES:  Unpublished, C. Jassoy and Beatrice Culman, pers comm	HIV-1 infection ers comm	human(A3.1)	[Brander95a]
p17(20-29 LAI)	p17(20-29) <b>NOTES:</b>	RLRPGGKKKY		human(Bw62)	[McMichael94]
	<ul><li>Review of Also P. J</li></ul>	Review of HIV CTL epitopes Also P. Johnson, per. comm.			
p17(20-28)	p17(20-28) NOTES:  • Identical • One had • [Goulder	(20-28) RLRPGGKKK HIV-1 infection human(A*03) [Goulder97, TES: Identical twin hemophiliac brothers were both infected with the same batch of factor VIII One had a response to gag A3 epitope RLRPGGKKK, the other non-responder carried the sequence RLRPGGKKC [Goulder97e] is a review of immune escape that summarizes this study	HIV-1 infection  Infected with the same bath of the same	human(A*03)  ch of factor VIII  onder carried the sequence	[Goulder97, Goulder97e] RLRPGGKKC
p17(20-28)	p17(20-28) <b>NOTES:</b>	RLRPGGKKK	HIV-1 infection	human(A3)	[Goulder97b]
	A contro	A control CTL line that reacts with this peptide was included in the study	was included in the study		
p17(20-28)	p17(20-28) <b>NOTES:</b> • The cons • The cons	(20-28) RLRPGGKKK HIV-1 infection huma  TES:  The consensus peptide of A, B, and D clade viruses is RLRPGGKKK  The consensus peptide of C clade viruses is RLRPGGKKH and is equally reactive	HIV-1 infection uses is RLRPGGKKK RPGGKKH and is equally	human(A3) reactive	[Cao97]

p17(24-31) p17(24-31) GGKKKYKL  NOTES:  • The crystal structure of this peptide bound to HLA-B8 was used to predict new epitopes an epitope variation  • The predictions were experimentally confirmed  • The anchors for HLA-B8 epitopes, as defined by peptide elution data, are P3 (K), P5 (K/R), a  • Structural data suggests that a positive charge at P5 is essential, but that the constraints on P3	p17(91-105 SF2) p17(21-35) LRPGGKKKYKLKHIV HIV-1 infection human NOTES:  Of 25 patients, most had CTL specific for more than 1 HIV-1 protein  12 subjects had CTL that could recognize vaccinia expressed LAI gag One of these 12 had CTL response to this peptide  The responding subject was HLA-A1, A2, B50, B57	p17(21-35) p17(21-35) LRPGGKKKYKLKHIV HIV-1 infection human(not B8)  NOTES:  • Unknown HLA specificity, but not B8	p17(21-35) p17(21-35) LRPGGKKKYKLKHIV human(B8)  NOTES:  • Two CTL epitopes defined (see also p24(191-205))	Location WEAU Sequence Immunogen Species(HLA)
Page 131) GGKKKYKL human(B8) [Goulder97c]  The crystal structure of this peptide bound to HLA-B8 was used to predict new epitopes and the consequences of epitope variation  The predictions were experimentally confirmed  The anchors for HLA-B8 epitopes, as defined by peptide elution data, are P3 (K), P5 (K/R), and P8 (L)  Structural data suggests that a positive charge at P5 is essential, but that the constraints on P3 may be less severe Small hydrophobic residues at P2 may be favorable for binding  A spacious F-pocket favors mid-sized hydrophobic residues in the C-term anchor  MCAdam98  MCAdam98	09	human(not B8)	human(B8)	Species(HLA)
[Goulder97c]  sequences of  less severe  [McAdam98]	[Lieberman97]	[vanBaalen96]	[Nixon91]	References

Location	WEAU	Sequence	Immunogen	${\bf Species(HLA)}$	References
p17(24-31 LAI)	p17(24-31) <b>NOTES:</b>	GGKKKYKL	HIV-1 infection	human(B8)	[Reid96]
	<ul><li>The variants 7R:</li><li>Crystal structure were determined</li></ul>	The variants 7R: GGKKKYRL, 7Q: GGKKKYQL, 5R: GGKKRYKL, and 3R: GGRKKYKL, were studied Crystal structures were obtained to study these peptides in the context of HLA-B8, and CTL binding and activity were determined	(QL, 5R: GGKKRYKL, au	nd 3R: GGRKKYKL, were f HLA-B8, and CTL bind	e studied ling and activity
	<ul><li>3R has be binding in 7Q and 7</li><li>Reactivity bound</li></ul>	3R has been detected in 3 patients, and it abolishes recognition causing extensive conformational changes upon binding including MHC main chain movement 7Q and 7R alter the TCR exposed surface, and retain some recognition Reactivity of 5R depends on the T cell clone, this amino acid is embedded in the C pocket of B8 when the peptide is bound	olishes recognition causing retain some recognition arise amino acid is embedded	g extensive conformational changes upon in the C pocket of B8 when the peptide is	al changes upon en the peptide is
	<ul><li>bound</li><li>Optimal p</li></ul>	bound Optimal peptide is 8-mer, not 9-mer, and positions 3, 5, and 8 are the anchor residues	ons 3, 5, and 8 are the anc	nor residues	
p17(24-31 LAI)	p17(24-31) <b>NOTES:</b>	GGKKKYKL	HIV-1 infection	human(B8)	[Price97]
	<ul><li>A weak CTI</li><li>Sequences f was present</li></ul>	A weak CTL response to the index peptide was observed in an HLA-B8+ infected Sequences from the earliest available time point showed that a variant at position 5, was present	s observed in an HLA-B8+ t showed that a variant at po	infected individual sition 5, an anchor residue, GGKKQYKL,	, GGKKQYKL,
p17(24-32 LAI)	p17(24-32) <b>NOTES:</b> • Explorati	17(24-32) GGKKKYKLK HIV-1 informs:  • Exploration of HLA-B8 binding motif through peptide elution	HIV-1 infection peptide elution	human(B8)	[Sutton93]
p17(24-32 LAI)	p17(24-32) <b>NOTES:</b>	GGKKKYKLK	HIV-1 infection	human(B8)	[RowlandJones93a]
	<ul> <li>Study of :</li> </ul>	<ul> <li>Study of an individual with partially defective antigen processing</li> </ul>	antigen processing		
p17(24-32)	p17(24-32) <b>NOTES:</b>	GGKKKYKLK	HIV-1 infection	human(B8)	[Klenerman94]
	<ul> <li>Naturally</li> </ul>	<ul> <li>Naturally occurring variants GGKKKYQLK and GGKKRYRLK may act as antagonists</li> </ul>	nd GGKKRYRLK may act	as antagonists	
p17(24-32)	p17(24-32) <b>NOTES:</b>	GGKKKYKLK	HIV-1 infection	human(B8)	[Klenerman95]
	<ul> <li>Naturally</li> </ul>	<ul> <li>Naturally occurring antagonist GGKKKYQLK found in viral PBMC DNA and R</li> </ul>	found in viral PBMC DN	A and RNA	
p17(24-32)	p17(24-32) <b>NOTES:</b>	GGKKKYKLK	HIV-1 infection	human(B8)	[Nowak95]
	• Longitudi reactive	Longitudinal study of CTL response and immune escape – the variant GGRKKY reactive	ne escape – the variant G	GRKKYKLK binds to HLA-B8 but is not	.A-B8 but is not

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(25-35 SF2)	p17(24-35) <b>NOTES:</b>	GGKKKYKLKHIV	HIV-1 infection	human(B8)	[Phillips91, Goulder97e]
	<ul><li>Longituc relative t</li><li>[Goulden and that</li></ul>	Longitudinal study of CTL escape mutants – little variation was observed in the immunodominant B27 epitope, relative to B8 epitopes, which varied over time, in people with the appropriate HLA types [Goulder97e] is a review of immune escape that points out that there may be a protective effect associated with B27, and that HLA-B8 individuals tend to progress more rapidly than HLA B27 patients	little variation was observe, in people with the appropriate points out that there may more rapidly than HLA B2	yed in the immunodominal riate HLA types be a protective effect associng patients	nt B27 epitope, iated with B27,
p17(25-35)	p17(24-35) <b>NOTES:</b>	GGKKKYKLKHIV	HIV-1 infection	human(B8)	[Birk98]
	<ul> <li>A study evolution</li> </ul>	A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is influenced by immune pressure from $CTLs$	7 epitopes and individuals m CTLs	with known HLA types re	vealed that p17
p17(28-36 LAI)	p17(28-36) <b>NOTES:</b> • D. Lewir	.7(28-36) KYKLKHIVW  OTES:  • D. Lewinsohn, pers. comm.		human(A24)	[Brander96]
p17(35-43 LAI)	p17(36-44)	WASRELERF	HIV-1 infection	human(B*3501)	[Goulder97a]
	<ul><li>NOTES:</li><li>Optimal</li><li>Dominar</li><li>The Phe</li></ul>	Optimal epitope defined from within p17(30-44), LKHIVWASRELERFA  Dominant CTL response in an HIV+ asymptomatic donor was to this epitope  The Phe in the C-term anchor is distinct from the previously defined Tyr for B*3501 C-term anchors	4), LKHIVWASRELERFA natic donor was to this epi he previously defined Tyr :	tope for B*3501 C-term anchors	
p17(36-44)	p17(36-44) <b>NOTES:</b>	WASRELERF	HIV-1 infection	human(B35)	[Birk98]
	A study evolution	A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is influenced by immune pressure from CTLs	7 epitopes and individuals m CTLs	with known HLA types re	vealed that p17
p17(69-93 BH10)	p17(69-93)	QTGSEELRSLYNTVATL- YCVHQRIE	HIV-1 infection	human(A2)	[Johnson91]
	NOTES: • Gag CTI	TES: Gag CTL response studied in three individuals			

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(71-85 SF2)	p17(71-85) <b>NOTES:</b>	GSEELRSLYNTVATL	HIV-1 infection	human	[Lieberman97]
	<ul><li>Of 25 pat</li><li>12 subjec</li><li>One of th</li><li>The response</li></ul>	Of 25 patients, most had CTL specific for more than 1 HIV-1 protein 12 subjects had CTL that could recognize vaccinia expressed LAI gag One of these 12 had CTL response to this peptide The responding subject was HLA-A1, A11, B8, B27	than 1 HIV-1 protein nia expressed LAI gag de , B27		
p17(71-79 LAI)	p17(71-79) <b>NOTES:</b> • P. Goulde	17(71-79) GSEELRSLY OTES: • P. Goulder, pers. comm.		human(A1)	[Brander96]
p17(71-79)	p17(71-79) <b>NOTES:</b>	GSEELRSLY	HIV-1 infection	human(A1)	[Birk98]
	A study of evolution	• A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is influenced by immune pressure from CTLs	7 epitopes and individuals m CTLs	with known HLA types rev	ealed that p17
p17	p17(74-82) <b>NOTES:</b> • Defined:	(74-82) ELRSLYNTV TES: Defined in a study of the R8 binding motif		human(B8)	[Goulder97c]
p17(74-82)	p17(74-82) <b>NOTES:</b>	ELRSLYNTV	HIV-1 infection	human(B8)	[Birk98]
	A study of evolution	A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is influenced by immune pressure from CTLs	7 epitopes and individuals m CTLs	with known HLA types rev	ealed that p17
p17(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A*0201)	[Altman96]
	<ul> <li>This paper sion of spe sion of spe SLYNTVA</li> <li>The highes patient wh not the Pol</li> <li>Reviewed</li> </ul>	This paper introduces the tetramer methodology which permits quantification of specific CTL based on expression of specific TCRs – HLA-A2 tetramers were prepared that can stain CTL lines specific for ILKEPVHGV and SLYNTVATL, and quantitate HIV-specific CD8+ cell lines in freshly isolated PBMCs  The highest frequency of tetramer staining was found to the Pol epitope, 0.77% of the CD8+ lymphocytes in one patient who also had cells specific for the Gag epitope (0.28%) – three other patients only stained the Gag epitope, not the Pol  Reviewed in [McMichael98]	gy which permits quantificate prepared that can stain of the cell lines in freshly isolates found to the Pol epitope, epitope (0.28%) – three other cells are considered to the polecy of the cells of the polecy of the cells of the polecy of the cells of the property of the cells of the property of the permits of the perm	ation of specific CTL based on expresation of specific for ILKEPVHGV and ted PBMCs 0.77% of the CD8+ lymphocytes in one ner patients only stained the Gag epitope,	sed on expres- EPVHGV and hocytes in one e Gag epitope,

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(77-85)	p17(77-85) <b>NOTES:</b> • A study c evolution	(77-85) SLYNTVATL HIV-1 infection human(A2) [Birk98]  TES: A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is influenced by immune pressure from CTLs	HIV-1 infection 17 epitopes and individuals m CTLs	human(A2) with known HLA types re	[Birk98] vealed that p17
p17(77-85 SF2)	p17(77-85) <b>NOTES:</b> • CTL fron	7(77-85) SLYNTVATL HIV-1 infection human(A*0201)  OTES:  CTL from a patient infected with clade B virus did not recognize the clade A analog of this epitope	HIV-1 infection did not recognize the clace	human(A*0201) le A analog of this epitope	[McAdam98]
p17(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A*0201)	[Wilson98]
	• HIV+ inc and clona • Seven HI uninfecte • Three par increases • An A2-G patient's	HIV+ individuals were followed longitudinally using MHC tetramers in combination with 14 anti-BV chain MAbs, and clonal expansion of HIV-specific T cells was followed <i>in vivo</i> Seven HIV+ people were studied, and all showed expansions of particular TCR BV clones, often several, relative to uninfected controls  Three patients were followed in detail, TCR VB expansions persisted for 2 to 3 years, with occasional transient increases  An A2-Gag specific line from one patient was found to be BV8, and at its highest level represented 17.5% of the patient's CD8+ T cells	as followed <i>in vivo</i> ed expansions of particula VB expansions persisted 1 found to be BV8, and at	r TCR BV clones, often several, relative to or 2 to 3 years, with occasional transient its highest level represented 17.5% of the	V chain MAbs, eral, relative to sional transient d 17.5% of the
p17(77-85)	p17(77-85) <b>NOTES:</b> • Included	.7(77-85) SLYNTVATL  OTES:  • Included as a negative control in a tetramer study of A2-EBV CTL response	HIV-1 infection dy of A2-EBV CTL respo	human(A2)	[Callan98]
p17(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A*0201)	[Ogg98]
	<ul> <li>HLA-tetrameric of revealing an inversion of both restricted activity</li> <li>No correlation was</li> </ul>	HLA-tetrameric complexes were used in a cross-sectional study of 14 untreated HLA A*0201 positive individuals, revealing an inverse relationship between HIV Gag and Pol specific CTL effector cells (CTLe) and viral load Inclusion of both the p17 SLYNTVATL and RT ILKEPVHGV epitopes gives a good representation of HLA A*0201-restricted activity  No correlation was observed between the CTLe and CD4 count or clearance rate of productively infected cells	ss-sectional study of 14 un Gag and Pol specific CTL ILKEPVHGV epitopes gi e and CD4 count or cleara	treated HLA A*0201 positive individuals, effector cells (CTLe) and viral load ves a good representation of HLA A*0201-nce rate of productively infected cells	iral load HLA A*0201-

P17(77-85) SLYNTVATL  NOTES:  CTL specific for HIV epitopes were used to show that the mediators of both the cytolytic (granzyme A was used as the marker) and non-cytolytic (HIV-1 inhibitory chemokines MIP-1 \(\alpha\) and RANTES were used as markers) and inon-cytolytic (HIV-1 inhibitory chemokines MIP-1 \(\alpha\) and RANTES were used as markers) anti-viral responses are localized within the CTL's cytotoxic granules  P17(77-85) SLYNTVATL  NOTES:  Not CTL clones recognize this epitope, but not the NL4-3 form of the epitope SLYNTIAVL  Nef down-regulates MHC class I molecules, which inhibits CTL killing, and this down-regulation can be partially compensated for by adding excess soluble peptide  P17(77-85) SLYNTVATL  NOTES:  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 vaccinita  Pol reactivity: 7/8 reacted with A subtype, and 7/8 to B subtype, and HIV-2 Gag  Not reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef  Env 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, 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 Nef  Env reactivity: 3/8 reacted with A subtype, 1/8 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 Nef  Env reactivity: 3/8 reacted with A subtype, 1/8 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 Nef  Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, 1/8 with HIV-2 Nef  Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, 1/8 with HIV-2 Nef  Env reactivity: 3/8
man(A2)  man(A2)  man(A2)  man(A2)  man(A2)  LYNTIAVL  is down-regulation can  man(A2)  man(A2)  man(A2)  man(A2)  patients from Bangui, ((ing in France originally vas not tested  IV-2 Nef 2 Env  Tag SLYNTVATL and I  man(A2)  man(A2)  man(A2)  man(A2)  man(A2)  man(A2)  man(A2)  ced with rgp160 MN or form the sequence of the sequence of table CTL response — the table CTL response — the sequence of table CTL respons
man(A2)  man(A2)  cytolytic (granzyme A ES were used as marke)  man(A2)  LYNTIAVL is down-regulation can  man(A2)  patients from Bangui, (( ing in France originally ing in France originally vas not tested  V-2 Nef Env Fag SLYNTVATL and P Ged with rgp 160 MN or A ed with rgp 160 MN or A ed with sequence a table CTL response – the
References  [Wagner98b] anzyme A was used as d as markers) anti-viral  [Collins98]  [Durali98] Bangui, (6 A subtype, originally from Togo, defending and Nef PLTFG-  [Kundu98]  60 MN or A2 restricted howed increase only in sequence as their HIV seponse – the other two

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(77-85 IIIB)	p17(77-85) <b>NOTES:</b> • HIV IIIE	(77-85) SLYNTVATL HIV-1 infection hum  TES:  HIV IIIB proteins were used to define the range of CTL epitopes recognized by 3	HIV-1 infection e of CTL epitopes recogni	human(A2) [Sipsas97] zed by 3 lab workers accidentally infected	[Sipsas97] entally infected
	with HIV-1 IIIB • SLYNTVAVL, a • SLFNTVAVL, a	with HIV-1 IIIB SLYNTVAVL, a variant found in HIV-1 MANC, was also recognized SLFNTVAVL, a variant found in HIV-1 NY5CG, was also recognized	C, was also recognized G, was also recognized		
p17	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A2)	[RowlandJones98]
	<ul> <li>A CTL 1 epitopes and conf</li> <li>The A su</li> <li>The D su</li> </ul>	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 subtype consensus is SLfNtvatL  The D subtype consensus is SLfNtvATL	infected prostitutes from D clades – such cross-reabtypes are circulating	Nairobi using previously defined B clade ctivity could protect against both A and D	t both A and D
p17(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	none	human(A*0201)	[Walter97]
	<ul><li>HLA-A2</li><li>The HL./</li><li>Suggests gens</li></ul>	HLA-A2 heavy chain and β2-microglobulin expressed in <i>E. coli</i> were refolded in the presence of this peptide The HLA-A2-peptide complex elicited HLA-A2 peptide specific CTL response in cells lacking HLA-A2 Suggests that preformed HLA-peptide complexes could provide an alternate to intracellular processing for immunogens	pressed in <i>E. coli</i> were ref 2 peptide specific CTL reses could provide an altern	olded in the presence of this peptide sponse in cells lacking HLA-A2 ate to intracellular processing for important to intracellular processing for important to the second	h-A2 ng for immuno-
p17	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A2)	[Sewell97]
	<ul><li>Naturally</li><li>The follo</li></ul>	Naturally occurring variants of this epitope escaped killing and acted as antagonists  The following variants were found in HIV-1 infected patients who mounted a strong response against this epitope: F,FV-,S, -SF,L,I-V-,	aped killing and acted as a fected patients who moun	ntagonists ted a strong response against this epito	ist this epitope: $I-V-$ ,
	FI • All varia • Antagoni	FI,FI-V-,F-A All variants bound to A2 with at least half the affinity of SLYNTVATL except the triple mutant:FI-V- Antagonism could be observed at low concentrations, abrogating lysis at an antagonist:agonist ratio of 1:10 – the antagonism was observed in one SLYNTVATL specific CTL line but not another	uffinity of SLYNTVATL exrations, abrogating lysis a specific CTL line but not	ccept the triple mutant:FI-V-t an antagonist:agonist ratio of 1:10 another	o of 1:10 – the

Location p17(77-85 HXB2)	<b>WEAU</b> p17(77-85)	Sequence SLYNTVATL	Immunogen HIV-1 infection	Species(HLA) human(A2)	References [Yang97b]
	<ul> <li>A chime signaling</li> <li>The respondence occuring</li> <li>A CTL c</li> </ul>	A chimeric universal T-cell receptor was created by linking CD4 or an HIV-specific anti-gp41 Ig sequence to the signaling domain of the T cell receptor chain ζ, and transducing into CD8+ cells  The response using universal-receptor-bearing CD8+ cells to lyse infected cells <i>in vitro</i> was comparable to the natural occuring responses of CTL-clones from HIV+ individuals in terms of kinetics and efficiency  A CTL clone specific for this epitope was used for the comparison	ted by linking CD4 or an F, and transducing into CD8. CD8+ cells to lyse infected c individuals in terms of kine I for the comparison	IIV-specific anti-gp41 Ig + cells ells <i>in vitro</i> was compara tics and efficiency	sequence
p17(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	in vitro stimulation	human(A2)	[Stuhler97]
	Keyhole was requ	Keyhole limpit hemocyanin or tetanus toxoid Th epitope co-expression with peptide CTL epitopes on the same APC was required for induction of peptide specific CTL	Th epitope co-expression wit	h peptide CTL epitopes o	on the same A
p17(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A201)	[Lalvani97]
	<ul> <li>A peptid</li> <li>import</li> <li>could be</li> <li>This pep</li> </ul>	A peptide based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-specific CTLp counts could be obtained via staining with peptide-Class I tetramers  This peptide was one of the test peptides for optimizing the protocol	stimulation of CTLp using o a primary response, only so ass I tetramers otimizing the protocol	ptimized peptide and IL- xondary – peptide-speci	7 concentration fic CTLp cour
p17(76-84)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	in vitro stimulation	human(A*0201)	[vanderBurg96]
	<ul><li>Slow dis</li><li>CTL gen</li></ul>	Slow dissociation rate is associated with immunogenicity CTL generated by <i>in vitro</i> stimulation of PBMC derived from uninfected individual	nogenicity C derived from uninfected i	ndividual	
gag(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A2)	[Yang96]
	<ul><li>CD4+ ce</li><li>Clones sj</li><li>The disti</li><li>CTL can</li></ul>	CD4+ cell lines acutely infected with HIV were studied to determine their susceptibility to lysis by CTL Clones specific for RT lysed HIV-1 infected cells at lower levels than Env or Gag specific clones. The distinction was thought to be due to lower expression of RT relative to Env and Gag CTL can lyse infected cells early after infection, possibly prior to viral production.	e studied to determine their lls at lower levels than Env expression of RT relative to possibly prior to viral pro	susceptibility to lysis by or Gag specific clones Env and Gag duction	CTL

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
gag(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A2)	[Yang97]
	<ul><li>CTL inh</li><li>CTL pro</li><li>CTL sup</li></ul>	CTL inhibit HIV-1 replication at effector cell concentrations comparable to those found <i>in vivo</i> CTL produced HIV-1-suppressive soluble factors – MIP-1 $\alpha$ , MIP-1 $\beta$ , RANTES, after antigen-specific activation CTL suppress HIV replication more efficiently in HLA-matched cells	centrations comparable to $-$ MIP-1 $\alpha$ , MIP-1 $\beta$ , RA1 HLA-matched cells	o those found <i>in vivo</i> NTES, after antigen-specif	ic activation
p17(77-85 LAI)	p17(77-85) <b>NOTES:</b> • Examine	.7(77-85) SLYNTVATL HIV-1 infection OTES:  • Examined in the context of motifs important for HLA-A2 binding	HIV-1 infection ILA-A2 binding	human(A2)	[Parker92, Parker94]
p17(77-85 LAI)	p17(77-85) <b>NOTES:</b> • Review (	(77-85) SLYNTVATL TES: Review of HIV CTL epitopes	HIV-1 infection	human(A2)	[McMichael94]
p17(77-85)	p17(77-85) <b>NOTES:</b> • CTL clo	7(77-85) SLYNTVATL  OTES:  CTL clones recognize naturally processed peptide	HIV-1 infection	human(A2)	[Tsomides94]
p17(77-85)	p17(77-85)  NOTES: • A three	(77-85) SLYNTVATL Peptide stimulation hum in vitro  TES: A three cell-type cluster consisting of APCs, Th, and CTLs is the minimal regu	Peptide stimulation in vitro  n, and CTLs is the mining	human(A2) [Stuhler97] nal regulatory unit required for Th cell-	[Stuhler97] ed for Th cell-
p17(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A2)	[Cao97]
	<ul><li>The cons</li><li>The cons</li></ul>	The consensus peptide of B and D clade viruses and some Cs have the sequence SLYNTVAT The consensus peptide of A and some C strains is SLFNTVATL, a form that is cross-reactive	nd some Cs have the sequence SLFNTVATL, a form the	uence SLYNTVATL at is cross-reactive	

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(A*0201)	[Goulder97, Goulder97e]
	<ul><li>Identical twin</li><li>One had a responsible</li><li>Viral sequenci</li><li>SLHNAVAVL</li></ul>	twin hemophiliac brothers were a response to gag A2 epitope SL quencing from the twin that had /AVL	Identical twin hemophiliac brothers were both infected with the same batch of factor VIII One had a response to gag A2 epitope SLYNTVATL, the other to pol A2 epitope ILKEPVHGV Viral sequencing from the twin that had no response to SLYNTVATL indicated his virus had the substituted form SLHNAVAVL	th of factor VIII epitope ILKEPVHGV dicated his virus had the	substituted form
	<ul> <li>71% of a</li> <li>VATL</li> </ul>	n additional set of 22 HIV-1 infe	71% of an additional set of 22 HIV-1 infected HLA-A*0201 positive donors preferentially responded to gag SLYNT-VATL	rs preferentially responde	ed to gag SLYNT-
	<ul><li>Those individ</li><li>An additional</li><li>SLFNTVATL</li></ul>	dividuals with a pol ILKEPVHG ional subject went from SLYNT /ATL	Those individuals with a pol ILKEPVHGV response tended to have mutations in or around SLYNTVATL An additional subject went from SLYNTVATL responder to non-responder coincident with a switch to the variant SLFNTVATL	tions in or around SLYNTVATL ler coincident with a switch to t	TVATL tch to the variant
	• [Goulder	[Goulder97e] is a review of immune escape that summarizes this study	ape that summarizes this study		
p17(77-85)	p17(77-85) <b>NOTES:</b>	SLYNTVATL	HIV-1 infection	human(B62)	[Goulder97e]
	<ul> <li>This pap to SLYN</li> </ul>	This paper is a review of CTL and immune evasion, but to SLYNTVATL, to a B62 response to GLNKIVRMY	This paper is a review of CTL and immune evasion, but it presents a study of a shift from an HLA-A*0201 response to SLYNTVATL, to a B62 response to GLNKIVRMY	of a shift from an HLA-	A*0201 response
	• As long dominan	as a strong CTL response to SLY ted the viral population – eventu	As long as a strong CTL response to SLYNTVATL was evident, the epitope variants SLFNTVATL or SLYNTIATL dominanted the viral population – eventually the CTL response to the index peptide became undetectable, the CTL	pe variants SLFNTVATI lex peptide became under	or SLYNTIATL lectable, the CTL
	the domi	the dominant form	the dominant form	INT ACTE Office again established fisch as	taonsnea nsen as
p17(84-92)	p17(84-92) <b>NOTES:</b>	TLYCVHQRI	HIV-1 infection	human(A11)	[Brander95a]
	• Epitope study	defined in the context of the Ped	Epitope defined in the context of the Pediatric AIDS Foundation ARIEL Project, study		a mother-infant HIV transmission
p17(84-92)	p17(84-92) <b>NOTES:</b>	TLYCVHQRI	HIV-1 infection	human(A11)	[Birk98]
	A study evolution	A study of p17 variation considering known p17 epitopevolution is influenced by immune pressure from CTLs	A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is influenced by immune pressure from CTLs	with known HLA types	revealed that p17

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(88-115 ARV)	p17(88-115) <b>NOTES:</b> • B cell epit	88-115) VHQRIEIKDTKEALDKI- EEEQNKSKKKA FES: B cell epitope HGP-30 also serves as a CTL epitope	HIV-1 infection	human(A2)	[Achour90]
p17(88-115 ARV)	p17(88-115) <b>NOTES:</b> • B cell epit  • Vaccine cc  • IL-12 expr	(88-115) VHQRIEIKDTKEALDKI- EEEQNKSKKKA  TES:  B cell epitope HGP-30 also serves as a CTL epitope Vaccine combined HGP-30, V3 loop peptide variants, and CD4 binding site peptide IL-12 expression plasmid included with the vaccination enhanced the CTL response	Combination peptide vaccine  e e its, and CD4 binding site ation enhanced the CTL i	murine BALB/c (H- $2^d$ ) peptide response	[Hamajima97]
p17(91-105 SF2)	p17(90-105) <b>NOTES:</b> • CTL expa	(7(90-105) CRIDVKDTKEALEKIE HIV-1 infection OTES:  • CTL expanded <i>ex vivo</i> were later infused into HIV-1 infected patients	HIV-1 infection I infected patients	human	[Lieberman97b]
p17(91-105 SF2)	p17(91-105) NOTES:  Of 25 pati  12 subject  One of the	(91-105) RIDVKDTKEALEKIE HIV-1 infection TES: Of 25 patients, most had CTL specific for more than 1 HIV-1 protein 12 subjects had CTL that could recognize vaccinia expressed LAI gag One of these 12 had CTL response to this peptide The responding subject was HLA-A3, A24, B8, B55	HIV-1 infection n 1 HIV-1 protein expressed LAI gag	human	[Lieberman97]
p17	p17(92-101) NOTES: • CTL speci the marker responses	(92-101) IEIKDTKEAL HIV-1 infection human(B60) [Wagner98t TES: CTL specific for HIV epitopes were used to show that the mediators of both the cytolytic (granzyme A was used as the marker) and non-cytolytic (HIV-1 inhibitory chemokines MIP-1 $\alpha$ and RANTES were used as markers) anti-viral responses are localized within the CTL's cytotoxic granules	HIV-1 infection hat the mediators of both mokines MIP-1 $\alpha$ and R. granules	human(B60) [Wagner98t the cytolytic (granzyme A was used as ANTES were used as markers) anti-viral	[Wagner98b] A was used as ærs) anti-viral
p17(93-101)	p17(93-101) <b>NOTES:</b> • Examined	(93-101) EIKDTKEAL no CTL shown hum  TES:  Examined in the context of motifs important for HLA-B8 binding, predicted epitor	no CTL shown  A-B8 binding, predicted	human(B8) [DiBrino9.1 epitope based on Achour et al. above	[DiBrino94a] et al. above

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(93-101)	p17(93-101) <b>NOTES:</b>	EIKDTKEAL	HIV-1 infection	human(B8)	[Birk98]
	<ul> <li>A study of evolution is</li> </ul>	A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is influenced by immune pressure from CTLs	itopes and individuals wi	th known HLA types reve	ealed that p17
p17(93-101 LAI)	p17(93-101) <b>NOTES:</b>	EIKDTKEAL		human(B8,B60)	[Brander97]
	• Per. comm.	• Per. comm. from A. Trocha and S. Kalaams to C. Brander and B. Walker	Brander and B. Walker		
p17(121-132 HXB2R)	p17(121-132)	DTGHSNQVSQNY	HIV-1 infection	human(A33)	[Buseyne93]
A STATE OF	NOTES: • Clustering	OTES: <ul> <li>Clustering of Gag p24 CTL epitopes recognized in 29 HIV infected people</li> </ul>	29 HIV infected people		
p17(124-132 LAI)	p17(124-132) <b>NOTES:</b> • Review of l	17(124-132) NSSKVSQNY OTES: • Review of HIV CTL epitopes	HIV-1 infection	human(B35)	[McMichael94]
P17(124-132)	P17(124-132) <b>NOTES:</b>	NSSKVSQNY	HIV-1 infection	human(B35)	[Birk98]
	A study of evolution is	A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is influenced by immune pressure from CTLs	itopes and individuals wi	th known HLA types reve	ealed that p17
p17(124-132 LAI)	p17(124-132)	NSSKVSQNY	HIV-1 or -2 infection	human(B35)	[RowlandJones95]
	NOTES: • Established by titration	by titration			
p17(124-132 LAI)	p17(124-132) <b>NOTES:</b>	NSSKVSQNY	none	human(B35)	[Lalvani97]
	<ul> <li>A peptide b</li> <li>important</li> <li>could be ob</li> <li>This peptid</li> </ul>	A peptide based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-specific CTLp counts could be obtained via staining with peptide-Class I tetramers  This peptide was one of the B35 presented test peptides used in control experiments showing that the assay gave no	lation of CTLp using opt mary response, only secutetramers ides used in control expe	mized peptide and IL-7 concentrations and ary – peptide-specific CTLp counts ciments showing that the assay gave no	oncentrations CTLp counts assay gave no

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(127-135 HIV-	p17(127-135)	p17(127-135) QVSQNYPIV		human(A*6802)	[Dong98]
i Cauc D)	NOTES: • Predicted o	OTES: Predicted on binding motif, no truncations analyzed			
p17(132-140 SF2)	p17(131-132) NYPIVQNL NOTES:	NYPIVQNL	HIV-1 infection	human(A*2402)	[IkedaMoore97]
	<ul> <li>The epitope</li> <li>Defined usi anchors in anchors in anchors in NYPIVQN</li> <li>NYPIVQN</li> </ul>	The epitope starts in p17 and ends in p24 Defined using reverse immunogenetics – 59 HLA-A*2402 binding peptides were predicted by searching for A*2402 anchors in HIV proteins, (Tyr at 2, and Phe, Leu or IIe at the C term) – 53 of the 59 peptides bound A*2402 This peptide induced CTL in 1/4 HIV-1+ people tested NYPIVQNL bound to A*2402 with medium strength, and the epitope can be processed in a vaccinia construct and presented – no CTL clone was obtained	*2402 binding peptides v lle at the C term) – 53 of ted th, and the epitope can b	vere predicted by searching for the 59 peptides bound A*2402 e processed in a vaccinia const	g for A*2402 *2402 construct and