

Table 1: **p17**

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(18-26 IIIB)	p17(18-26)	KIRLRPGGK	HIV-1 infection	human(A3)	[Walkerpercom96]
	NOTES:				
			<ul style="list-style-type: none"> • Epitope defined in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • KIRLRPGGR and RIRLRPGGR, naturally occurring variants, were found in mother, and are not recognized 		
p17(18-26 IIIB)	p17(18-26)	KIRLRPGGK	HIV-1 infection	human(A3)	[Goulder97, Goulder97e]
	NOTES:				
			<ul style="list-style-type: none"> • 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 		
p17(18-26 LAI)	p17(18-26)	KIRLRPGGK	HIV-1 infection	human(A3.1)	[Harrey96b]
p17(18-27 LAI)	p17(18-27)	KIRLRPGGKK		human(B27)	[Brandner96]
	NOTES:				
			<ul style="list-style-type: none"> • D. Lewinsohn, pers. comm. 		
p17(18-27)	p17(18-27)	KIRLRPGGKK	HIV-1 infection	human(B27)	[Birk98]
	NOTES:				
			<ul style="list-style-type: none"> • 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 		
p17(18-31)	p17(18-31)	KIRLRPGGKKYKL	HIV-1 infection	human(B62)	[Lubaki97]
	NOTES:				
			<ul style="list-style-type: none"> • 82 HIV-1-specific CTL clones from 5 long term non-progressors were isolated and analyzed for breadth of CTL response • 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 HLA-B62+ had CTL that recognized this peptide, and p24 LGLNKIVRMYS, and one additional unknown epitope 		

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(18-31)	p17(18-31) NOTES:	KIRLRPGGKKKYKL	HIV-1 infection	human(A3)	[Birk98]
	<ul style="list-style-type: none"> • 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 				
p17(18-42 IIB)	p17(18-42) NOTES:	KIRLRPGGKKKYKLKHI- VWASRELE	HIV-1 infection	human(A3)	[Jassoy92]
	<ul style="list-style-type: none"> • Epitope recognized by CTL clone derived from CSF 				
p17(18-42 BH10)	p17(18-42) NOTES:	KIRLRPGGKKKYKLKHI- VWASRELE	HIV-1 infection	human(Bw62)	[Johnson91]
	<ul style="list-style-type: none"> • Gag CTL response was studied in three individuals 				
p17(18-42 PV22)	p17(18-42) NOTES:	KIRLRPGGKKKYKLKHI- VWASRELE	HIV-1 infection	human(A3)	[Jassoy93]
	<ul style="list-style-type: none"> • HIV-1 specific CTLs release γ-IFN, and α- and β-TNF 				
p17(90-105 SF2)	p17(20-35) NOTES:	CLRPPGGKKKYKLLKHIV	HIV-1 infection	human	[Lieberman97]
	<ul style="list-style-type: none"> • 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 				
p17(19-27 LAI)	p17(19-27) NOTES:	IRLRPGGKK		human(B27)	[Brandt96]
	<ul style="list-style-type: none"> • D. Lewinsohn, pers. comm. 				

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(20-29 IIB)	p17(20-29) NOTES:	RLRPPGGKKKY	HIV-1 infection	human(B42)	[Walkerpercom96]
	<ul style="list-style-type: none"> Epitope defined in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study RLRPPGGKKRY, a naturally occurring variant, was found in non-transmitting mother and is recognized Binds HLA-A3 and Bw62 as well 				
p17(20-29)	p17(20-29) NOTES:	RLRPPGGKKKY	HIV-1 infection	human(A3.1)	[Brander95a]
	<ul style="list-style-type: none"> Unpublished, C. Jassoy and Beatrice Culman, pers comm 				
p17(20-29 LAI)	p17(20-29) NOTES:	RLRPPGGKKKY		human(Bw62)	[McMichael94]
	<ul style="list-style-type: none"> Review of HIV CTL epitopes Also P. Johnson, per. comm. 				
p17(20-28)	p17(20-28) NOTES:	RLRPPGGKKK	HIV-1 infection	human(A*03)	[Goulder97, Goulder97e]
	<ul style="list-style-type: none"> Identical twin hemophiliac brothers were both infected with the same batch of factor VIII One had a response to gag A3 epitope RLRPPGGKKK, the other non-responder carried the sequence RLRPPGGKKK [Goulder97e] is a review of immune escape that summarizes this study 				
p17(20-28)	p17(20-28) NOTES:	RLRPPGGKKK	HIV-1 infection	human(A3)	[Goulder97b]
	<ul style="list-style-type: none"> A control CTL line that reacts with this peptide was included in the study 				
p17(20-28)	p17(20-28) NOTES:	RLRPPGGKKK	HIV-1 infection	human(A3)	[Caoy97]
	<ul style="list-style-type: none"> The consensus peptide of A, B, and D clade viruses is RLRPPGGKKK The consensus peptide of C clade viruses is RLRPPGGKKH and is equally reactive 				

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(21-35)	p17(21-35) NOTES:	LRRPGGKKKKYKLIKHHV		human(B8)	[Nixon91]
					<ul style="list-style-type: none"> Two CTL epitopes defined (see also p24(191-205))
p17(21-35)	p17(21-35) NOTES:	LRRPGGKKKKYKLIKHHV	HIV-1 infection	human(not B8)	[vanBalen96]
					<ul style="list-style-type: none"> Unknown HLA specificity, but not B8
p17(91-105 SF2)	p17(21-35) NOTES:	LRRPGGKKKKYKLIKHHV	HIV-1 infection	human	[Lieberman97]
					<ul style="list-style-type: none"> 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(24-31)	p17(24-31) NOTES:	GGKKKKYKL		human(B8)	[Goulder97c]
					<ul style="list-style-type: none"> 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
p17(24-31 SF2)	p17(24-31) NOTES:	GGKKKKYKL	HIV-1 infection	human(B8)	[McAdam98]
					<ul style="list-style-type: none"> CTL from a patient infected with clade B virus did not recognize Ugandan variants of this epitope

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(24-31 LAI)	p17(24-31) NOTES:	GGKKKYKL	HIV-1 infection	human(B8)	[Reid96]
	<ul style="list-style-type: none"> 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 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 Optimal peptide is 8-mer, not 9-mer, and positions 3, 5, and 8 are the anchor residues 				
p17(24-31 LAI)	p17(24-31) NOTES:	GGKKKYKL	HIV-1 infection	human(B8)	[Price97]
	<ul style="list-style-type: none"> A weak CTL response to the index peptide was observed in an HLA-B8+ infected individual Sequences from the earliest available time point showed that a variant at position 5, an anchor residue, GGKKQYKL, was present 				
p17(24-32 LAI)	p17(24-32) NOTES:	GGKKKYKLK	HIV-1 infection	human(B8)	[Sutton93]
	<ul style="list-style-type: none"> Exploration of HLA-B8 binding motif through peptide elution 				
p17(24-32 LAI)	p17(24-32) NOTES:	GGKKKYKLK	HIV-1 infection	human(B8)	[RowlandJones93a]
	<ul style="list-style-type: none"> Study of an individual with partially defective antigen processing 				
p17(24-32)	p17(24-32) NOTES:	GGKKKYKLK	HIV-1 infection	human(B8)	[Klenerman94]
	<ul style="list-style-type: none"> Naturally occurring variants GGKKKYQLK and GGKKRYRLK may act as antagonists 				
p17(24-32)	p17(24-32) NOTES:	GGKKKYKLK	HIV-1 infection	human(B8)	[Klenerman95]
	<ul style="list-style-type: none"> Naturally occurring antagonist GGKKKYQLK found in viral PBMC DNA and RNA 				
p17(24-32)	p17(24-32) NOTES:	GGKKKYKLK	HIV-1 infection	human(B8)	[Nowak95]
	<ul style="list-style-type: none"> Longitudinal study of CTL response and immune escape – the variant GGRKKYKLK binds to HLA-B8 but is not reactive 				

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(25-35 SF2)	p17(24-35)	GGKKKYKLIKHIW	HIV-1 infection	human(B8)	[Phillips91, Goulder97e]
	NOTES:				
					<ul style="list-style-type: none"> • 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
p17(25-35)	p17(24-35)	GGKKKYKLIKHIW	HIV-1 infection	human(B8)	[Birk98]
	NOTES:				
					<ul style="list-style-type: none"> • 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
p17(28-36 LAI)	p17(28-36)	KYKLIKHIWV		human(A24)	[Brander96]
	NOTES:				
					<ul style="list-style-type: none"> • D. Lewinsohn, pers. comm.
p17(35-43 LAI)	p17(36-44)	WASRELERF	HIV-1 infection	human(B*3501)	[Goulder97a]
	NOTES:				
					<ul style="list-style-type: none"> • 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
p17(36-44)	p17(36-44)	WASRELERF	HIV-1 infection	human(B35)	[Birk98]
	NOTES:				
					<ul style="list-style-type: none"> • 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
p17(69-93 BH10)	p17(69-93)	QTGSEELRSLYNTVATL- YCVHORIE	HIV-1 infection	human(A2)	[Johnson91]
	NOTES:				
					<ul style="list-style-type: none"> • Gag CTL response studied in three individuals

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(71-85 SF2)	p17(71-85) NOTES:	GSEELRSLYNTVATL	HIV-1 infection	human	[Lieberman97]
	<ul style="list-style-type: none"> • 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 				
p17(71-79 LAI)	p17(71-79) NOTES:	GSEELRSLY		human(A1)	[Brander96]
	<ul style="list-style-type: none"> • P. Goulder, pers. comm. 				
p17(71-79)	p17(71-79) NOTES:	GSEELRSLY	HIV-1 infection	human(A1)	[Birk98]
	<ul style="list-style-type: none"> • 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 				
p17	p17(74-82) NOTES:	ELRSLYNTV		human(B8)	[Goulder97c]
	<ul style="list-style-type: none"> • Defined in a study of the B8 binding motif 				
p17(74-82)	p17(74-82) NOTES:	ELRSLYNTV	HIV-1 infection	human(B8)	[Birk98]
	<ul style="list-style-type: none"> • 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 				
p17(77-85)	p17(77-85) NOTES:	SLYNTVATL	HIV-1 infection	human(A*0201)	[Altman96]
	<ul style="list-style-type: none"> • 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] 				

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species/(HLA)	References
p17(77-85)	p17(77-85) NOTES:	SLYNTVATL	HIV-1 infection	human(A2)	[Birk98]
	<ul style="list-style-type: none"> • 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 				
p17(77-85 SF2)	p17(77-85) NOTES:	SLYNTVATL	HIV-1 infection	human(A*0201)	[McAdam98]
	<ul style="list-style-type: none"> • CTL from a patient infected with clade B virus did not recognize the clade A analog of this epitope 				
p17(77-85)	p17(77-85) NOTES:	SLYNTVATL	HIV-1 infection	human(A*0201)	[Wilson98]
	<ul style="list-style-type: none"> • 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 				
p17(77-85)	p17(77-85) NOTES:	SLYNTVATL	HIV-1 infection	human(A2)	[Callan98]
	<ul style="list-style-type: none"> • Included as a negative control in a tetramer study of A2-EBV CTL response 				
p17(77-85)	p17(77-85) NOTES:	SLYNTVATL	HIV-1 infection	human(A*0201)	[Ogg98]
	<ul style="list-style-type: none"> • 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 (CTL_e) 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 CTL_e and CD4 count or clearance rate of productively infected cells 				

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Wagner'98b]
	NOTES:				
					<ul style="list-style-type: none"> 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 α and RANTES were used as markers) anti-viral responses are localized within the CTL's cytotoxic granules
p17(77-85 HXB2)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Collins98]
	NOTES:				
					<ul style="list-style-type: none"> Two CTL clones recognize this epitope, but not the NL4-3 form of the epitope SLYNTTAVL 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
gag(77-85)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Dural'98]
	NOTES:				
					<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 PLTFG-WCFKL
gag(77-85)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Kundu98]
	NOTES:				
					<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 SLYNTVATL is a conserved HLA-A2 epitope included in this study – 3/6 patients had this sequence as their HIV direct sequence, one had the form SLYNTVAVL and all four of these had a detectable CTL response – the other two had either the sequence SLFSAVAVL or SLFSAVVAVL and no detectable CTL response

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(77-85 IIB)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Sipasas97]
	NOTES:				
					<ul style="list-style-type: none"> • HIV IIB proteins were used to define the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIB • SLYNTVAVL, a variant found in HIV-1 MANC, was also recognized • SLFNTVAVL, a variant found in HIV-1 NY5CG, was also recognized
p17	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[RowlandJones98]
	NOTES:				
					<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 subtype consensus is SLFNTVATL • The D subtype consensus is SLYNTVATL
p17(77-85)	p17(77-85)	SLYNTVATL	none	human(A*0201)	[Walter97]
	NOTES:				
					<ul style="list-style-type: none"> • 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
p17	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Sewell97]
	NOTES:				
					<ul style="list-style-type: none"> • 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-----, --F-----V-, --S-----, --SF-----, --L-----, -----I-V-, --F-----, --F--I-V-, --F-A----- --F--I-----, --F--I-V-, --F-A----- • All variants bound to A2 with at least half the affinity of SLYNTVATL except the triple mutant: --F--I-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

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(77-85 HXB2)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang97b]
	NOTES:		<ul style="list-style-type: none"> • 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 occurring 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 		
p17(77-85)	p17(77-85)	SLYNTVATL	<i>in vitro</i> stimulation	human(A2)	[Stuhler97]
	NOTES:		<ul style="list-style-type: none"> • 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 		
p17(77-85)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A201)	[Lalvani97]
	NOTES:		<ul style="list-style-type: none"> • A peptide based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-specific CTLp counts could be obtained via staining with peptide-Class I tetramers • This peptide was one of the test peptides for optimizing the protocol 		
p17(76-84)	p17(77-85)	SLYNTVATL	<i>in vitro</i> stimulation	human(A*0201)	[vanderBurg96]
	NOTES:		<ul style="list-style-type: none"> • Slow dissociation rate is associated with immunogenicity • CTL generated by <i>in vitro</i> stimulation of PBMC derived from uninfected individual 		
gag(77-85)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang96]
	NOTES:		<ul style="list-style-type: none"> • 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 		

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
gag(77-85)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang97]
	NOTES:		<ul style="list-style-type: none"> • 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-1α, MIP-1β, RANTES, after antigen-specific activation • CTL suppress HIV replication more efficiently in HLA-matched cells 		
p17(77-85 LAD)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Parker92, Parker94]
	NOTES:		<ul style="list-style-type: none"> • Examined in the context of motifs important for HLA-A2 binding 		
p17(77-85 LAD)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[McMichael94]
	NOTES:		<ul style="list-style-type: none"> • Review of HIV CTL epitopes 		
p17(77-85)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Tsonides94]
	NOTES:		<ul style="list-style-type: none"> • CTL clones recognize naturally processed peptide 		
p17(77-85)	p17(77-85)	SLYNTVATL	Peptide stimulation <i>in vitro</i>	human(A2)	[Stuhler97]
	NOTES:		<ul style="list-style-type: none"> • A three cell-type cluster consisting of APCs, Th, and CTLs is the minimal regulatory unit required for Th cell-dependent induction of CTLs 		
p17(77-85)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A2)	[Cao97]
	NOTES:		<ul style="list-style-type: none"> • The consensus peptide of B and D clade viruses and some Cs have the sequence SLYNTVATL • The consensus peptide of A and some C strains is SLFNTVATL, a form that is cross-reactive 		

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(77-85)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Goulder97, Goulder97e]
	NOTES:				
					<ul style="list-style-type: none"> • 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 SLHNNAVAVL • 71% of an additional set of 22 HIV-1 infected HLA-A*0201 positive donors preferentially responded to gag SLYNTVATL • 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 • [Goulder97e] is a review of immune escape that summarizes this study
p17(77-85)	p17(77-85)	SLYNTVATL	HIV-1 infection	human(B62)	[Goulder97e]
	NOTES:				
					<ul style="list-style-type: none"> • 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 GLNKIVRMV • As long as a strong CTL response to SLYNTVATL was evident, the epitope variants SLFNTVATL or SLYNTIATL dominated the viral population – eventually the CTL response to the index peptide became undetectable, the CTL response shifted to a focus on GLNKIVRMV, and the index peptide SLYNTVATL once again established itself as the dominant form
p17(84-92)	p17(84-92)	TLYCVHQRI	HIV-1 infection	human(A11)	[Brander95a]
	NOTES:				
					<ul style="list-style-type: none"> • Epitope defined in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study
p17(84-92)	p17(84-92)	TLYCVHQRI	HIV-1 infection	human(A11)	[Birk98]
	NOTES:				
					<ul style="list-style-type: none"> • 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 CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(88-115 ARV)	p17(88-115)	VHQRIEIKDTKEALDKI- EEEQNKSKKKA	HIV-1 infection	human(A2)	[Achour90]
	NOTES:				
					<ul style="list-style-type: none"> • B cell epitope HGP-30 also serves as a CTL epitope
p17(88-115 ARV)	p17(88-115)	VHQRIEIKDTKEALDKI- EEEQNKSKKKA	Combination pep- tide vaccine	murine BALB/c (H- 2 ^d)	[Hamajima97]
	NOTES:				
					<ul style="list-style-type: none"> • 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
p17(91-105 SF2)	p17(90-105)	CRIDVKDTKEALEKIE	HIV-1 infection	human	[Lieberman97b]
	NOTES:				
					<ul style="list-style-type: none"> • CTL expanded <i>ex vivo</i> were later infused into HIV-1 infected patients
p17(91-105 SF2)	p17(91-105)	RIDVKDTKEALEKIE	HIV-1 infection	human	[Lieberman97]
	NOTES:				
					<ul style="list-style-type: none"> • 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 HL-A-A3, A24, B8, B55
p17	p17(92-101)	IEIKDTKEAL	HIV-1 infection	human(B60)	[Wagner98b]
	NOTES:				
					<ul style="list-style-type: none"> • 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 α and RANTES were used as markers) anti-viral responses are localized within the CTL's cytotoxic granules
p17(93-101)	p17(93-101)	EIKDTKEAL	no CTL shown	human(B8)	[DiBriño94a]
	NOTES:				
					<ul style="list-style-type: none"> • Examined in the context of motifs important for HL-A-B8 binding; predicted epitope based on Achour et al. above

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(93-101)	p17(93-101)	EIKDTKEAL	HIV-1 infection	human(B8)	[Birk98]
	NOTES:				
					<ul style="list-style-type: none"> • 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
p17(93-101 LAI)	p17(93-101)	EIKDTKEAL		human(B8,B60)	[Brander97]
	NOTES:				
					<ul style="list-style-type: none"> • Per. comm. from A. Trocha and S. Kalams to C. Brander and B. Walker
p17(121-132 HXB2R)	p17(121-132)	DTGHSNQVSONY	HIV-1 infection	human(A33)	[Buseyne93]
	NOTES:				
					<ul style="list-style-type: none"> • Clustering of Gag p24 CTL epitopes recognized in 29 HIV infected people
p17(124-132 LAI)	p17(124-132)	NSSKVSQNY	HIV-1 infection	human(B35)	[McMichael94]
	NOTES:				
					<ul style="list-style-type: none"> • Review of HIV CTL epitopes
p17(124-132)	P17(124-132)	NSSKVSQNY	HIV-1 infection	human(B35)	[Birk98]
	NOTES:				
					<ul style="list-style-type: none"> • 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
p17(124-132 LAI)	p17(124-132)	NSSKVSQNY	HIV-1 or -2 infection	human(B35)	[RowlandJones95]
	NOTES:				
					<ul style="list-style-type: none"> • Established by titration
p17(124-132 LAI)	p17(124-132)	NSSKVSQNY	none	human(B35)	[Lalvani97]
	NOTES:				
					<ul style="list-style-type: none"> • A peptide based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-specific CTLp counts could be obtained via staining with peptide-Class I tetramers • This peptide was one of the B35 presented test peptides used in control experiments showing that the assay gave no activity using lymphocytes from 21 healthy B35 seronegative donors

HIV CTL Epitopes

Location	WEAU	Sequence	Immunogen	Species(HLA)	References
p17(127-135 HIV-1 Clade D)	p17(127-135)	QVVSQNYPIV		human(A*6802)	[Dong98]
<p>NOTES:</p> <ul style="list-style-type: none"> • Predicted on binding motif, no truncations analyzed 					
p17(132-140 SF2)	p17(131-132)	NYPIVQNL	HIV-1 infection	human(A*2402)	[IkedaMoore97]
<p>NOTES:</p> <ul style="list-style-type: none"> • The epitope starts in p17 and ends in p24 • Defined using reverse immunogenetics – 59 HLA-A-A*2402 binding peptides were predicted by searching for A*2402 anchors in HIV proteins, (Tyr at 2, and Phe, Leu or Ile at the C term) – 53 of the 59 peptides bound A*2402 • This peptide induced CTL in 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 					