Table 1: **p17**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK	HIV-1 infection	human(A3)	[Wilson (1996)]
• 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 escape mutants					
p17(18–26)	p17(18–26)	KIRLRPGGK	*in vitro* stimulation	human(A3)	[Zarling (1999)]
• HIV and influenza virus CTL epitopes were used to study the relative abilities of different antigen presenting cells (macrophages, immature dendritic cells (iDC) and mature dendritic cells (mDC)) to prime CD8+ lymphocytes • Both types of dendritic cells were superior to macrophages in the primary stimulation of CTL					
p17(18–26)	Gag(18–26)	KIRLRPGGK	HIV-1 infection	human(A3)	[Brodie (1999)]
• The ability of CTL effector cells was studied by expanding autologous HIV-1 Gag-specific CTL *in vitro*, and adoptive transfer • The transferred CTLs migrated to the lymph nodes and transiently reduced circulating productively infected CD4+ T cells, showing that CTL move to appropriate target sites and mediate anti-viral effects					
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK	HIV-1 infection	human(A3)	[Wilson (1999a)]
• This study describes maternal CTL responses in the context of mother-to-infant transmission • Detection of CTL escape mutants in the mother was associated with transmission, but the CTL susceptible forms of the virus tended to be found in infected infants • KIRLRPGGR and RIRLRPGGR were escape mutants • This epitope was recognized and many escape mutants were detected in an HLA A3 transmitting mother, and was recognized but invariant in an HLA A3 non-transmitting mother					
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK	HIV-1 infection	human(A3)	[Goulder (1997b), Goulder (1997a)]
• 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. [Goulder (1997b)] is a review of immune escape that summarizes this study.					
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK		human(A*0301)	
• C. Brander notes that this is a A*0301 epitope in the 1999 database					
p17(18–27)	p17(18–27 LAI)	KIRLRPGGKK		human(B27)	[Brander & Walker(1997a)]
• D. Lewinsohn, pers. comm.					

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(18–27)	p17(18–27)	KIRLRPGGKK	HIV-1 infection	human(B27)	[Birk (1998)]
	• 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)	KIRLRPGGKKKYKL	HIV-1 infection	human(B62)	[Lubaki (1997)]
	• 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				
p17(18–31)	p17(18–31)	KIRLRPGGKKKYKL	HIV-1 infection	human(A3)	[Birk (1998)]
	• 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)	p17(18–42 IIIB)	KIRLRPGGKKKYKLKHI-VWASRELE	HIV-1 infection	human(A3)	[Jassoy (1992)]
	• Epitope recognized by CTL clone derived from CSF				
p17(18–42)	p17(18–42 BH10)	KIRLRPGGKKKYKLKHI-VWASRELE	HIV-1 infection	human(Bw62)	[Johnson (1991)]
	• Gag CTL response was studied in three individuals				
p17(18–42)	p17(18–42 PV22)	KIRLRPGGKKKYKLKHI-VWASRELE	HIV-1 infection	human(A3)	[Jassoy (1993)]
	• HIV-1 specific CTLs release γ-IFN, and α- and β-TNF				
p17(19–27)	p17(19–27 LAI)	IRLRPGGKK		human(B*2705,B27)	[Brander & Walker(1997a)]
	• Noted in Brander 1999, this database, to be B*2705, Pers. Comm. D. Lewinsohn				
p17(19–27)	p17(19–27 JR-CSF)	IRLRPGGKK	HIV-1 infection	scid-hu mouse(B*2705,B27)	[McKinney (1999)]
	• Epitope-specific CTL were infused in infected human PBL-SCID mice, and transient decreases in viral load were observed, however the CTL rapidly disappeared through target interaction • No escape mutants were observed • Control CTL were long lived in both infected and uninfected mice, showing the rapid loss of CTL was due to target interaction • Noted in Brander 1999, this database, to be B*2705, Pers. Comm. D. Lewinsohn				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A*0301)	[Goulder (1997b), Goulder (1997a)]
• 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 • [Goulder (1997a)] is a review of immune escape that summarizes this study • C. Brander notes that this is a A*0301 epitope in the 1999 database					
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A3)	[Goulder (1997f)]
• A control CTL line that reacts with this peptide was included in the study					
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A3)	[Cao (1997)]
• 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					
p17(20–29)	p17(20–29 IIIB)	RLRPGGKKKY	HIV-1 infection	human(B42)	[Wilson (1996)]
• Epitope defined in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • RLRPGGKKRY, 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)	RLRPGGKKKY	HIV-1 infection	human(A3.1)	[Brander & Walker(1995)]
• Unpublished, C. Jassoy and Beatrice Culman, pers. comm.					
p17(20–29)	p17(20–29 LAI)	RLRPGGKKKY		human(Bw62)	[McMichael & Walker(1994)]
• Review of HIV CTL epitopes • Also P. Johnson, pers. comm.					
p17(20–29)	p17(20–29 LAI)	RLRPGGKKKY	HIV-1 infection	human(A3.1)	[Wilkens & Ruhl(1999)]
• Pers. comm., B. Wilkens and D. Ruhl					
p17(20–35)	p17(90–105 SF2)	CLRPGGKKKYKLKHIV	HIV-1 infection	human( )	[Lieberman (1997a)]
• 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					

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(21–35)	p17(21–35)	LRPGGKKKYKLKHIV		human(B8)	[Nixon & McMichael(1991)]
	• Two CTL epitopes defined (see also p24(191-205))				
p17(21–35)	p17(21–35)	LRPGGKKKYKLKHIV	HIV-1 infection	human(not B8)	[van Baalen (1996)]
	• Unknown HLA specificity, but not B8				
p17(21–35)	p17(91–105 SF2)	LRPGGKKKYKLKHIV	HIV-1 infection	human( )	[Lieberman (1997a)]
	• 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(21–40)	p17(21–40 Clade A)	LRPGGKKKYRLKHLVWASRE	HIV-1 infection	human(Cw4)	[Dorrell (1999)]
	• 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 was defined in an A subtype infection – the B clade variant (LRPGGKKKYKLKHIVWASRE) has two mutations relative to the A subtype form, and the CTL from this patient were not A-B cross-reactive				
p17(24–31)	p17(24–31)	GGKKKYKL		human(B8)	[Goulder (1997g)]
	• 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)	p17(24–31 SF2)	GGKKKYKL	HIV-1 infection	human(B8)	[McAdam (1998)]
	• CTL from a patient infected with clade B virus did not recognize Ugandan variants of this epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(24–31)	p17(24–31 LAI)	GGKKKYKL	HIV-1 infection	human(B8)	[Reid (1996)]
	• 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)	p17(24–31 LAI)	GGKKKYKL	HIV-1 infection	human(B8)	[Price (1997)]
	• 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)	p17(24–32 LAI)	GGKKKYKLK	HIV-1 infection	human(B8)	[Sutton (1993)]
	• Exploration of HLA-B8 binding motif through peptide elution				
p17(24–32)	p17(24–32 LAI)	GGKKKYKLK	HIV-1 infection	human(B*0801,B8)	[Rowland-Jones (1993b)]
	• Study of an individual with partially defective antigen processing • Noted in Brander 1999, this database, to be B*0801				
p17(24–32)	p17(24–32)	GGKKKYKLK	HIV-1 infection	human(B8)	[Klenerman (1994)]
	• Naturally occurring variants GGKKKYQLK and GGKKRYRLK may act as antagonists				
p17(24–32)	p17(24–32)	GGKKKYKLK	HIV-1 infection	human(B8)	[Klenerman (1995)]
	• Naturally occurring antagonist GGKKKYQLK found in viral PBMC DNA and RNA				
p17(24–32)	p17(24–32)	GGKKKYKLK	HIV-1 infection	human(B8)	[Nowak (1995)]
	• Longitudinal study of CTL response and immune escape – the variant GGRKKYKLK binds to HLA-B8 but is not reactive				
p17(24–32)	p17(24–32)	GGKKKYKLK	HIV-1 infection	human(B8)	[Dyer (1999)]
	• CTL specific responses were measured over a 1.5- to 1.3-year period in members of the Sydney Blood Bank Cohort (SBBC) who had been infected with a natural attenuated strain of HIV-1 which was nef-defective. Some of these patients had prolonged high levels of CTL effector and memory cells despite low viral load.				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(24–32)	p17( )	GGKKKYKLK		human(B8)	[Rowland-Jones (1999)]
	• CTL responses in seronegative highly HIV-exposed African female sex workers in Gambia and Nairobi were studied – these women had no delta 32 deletion in CCR5 • In Gambia there is exposure to both HIV-1 and HIV-2, CTL responses to B35 epitopes in exposed uninfected women are cross-reactive, and the B35 allele seems to be protective • HIV-2 sequence: GGKKKYKMK – no cross-reactivity [Phillips (1991)]				
p17(24–35)	p17(25–35 SF2)	GGKKKYKLKHIV	HIV-1 infection	human(B8)	[Phillips (1991), Goulder (1997a)]
	• 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 • [Goulder (1997a)] 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(24–35)	p17(25–35)	GGKKKYKLKHIV	HIV-1 infection	human(B8)	[Birk (1998)]
	• 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)	p17(28–36 LAI)	KYKLKHIVW		human(A24)	[Brander & Walker(1997a)]
	• D. Lewinsohn, pers. comm. • C. Brander notes that this is a A*2402 epitope in the 1999 database				
p17(28–36)	p17(28–36 LAI)	KYKLKHIVW		human(A23)	[Goulder(1999)]
	• P. Goulder, pers. comm.				
p17(28–36)	p17(28–36 SF2)	KYKLKHIVW	HIV-1 INFECTION	human(A*2402)	[Ikeda-Moore (1998)]
	• Strong CTL activity to this peptide was detected in 2/3 HIV-infected individuals who were HLA A24+ • HLA A24 is very common in Japanese (70% carry it) and is common globally • This epitope was detected by looking for peptides with appropriate A24 anchor residues (Y at position 2, carb-term ILF or W) – 16/17 such peptides bound to A24 – KYKLKHIVW was found to be a naturally processed epitope that elicits a strong CTL response.				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(36–44)	p17(35–43 LAI)	WASRELERF	HIV-1 infection	human(B*3501)	[Goulder (1997d)]
• 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)	[Birk (1998)]
• 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)	p17(69–93 BH10)	QTGSEELRSLYNTVATL-YCVHQRIE	HIV-1 infection	human(A2)	[Johnson (1991)]
• Gag CTL response studied in three individuals					
p17(71–79)	p17(71–79 LAI)	GSEELRSLY		human(A1)	[Brander & Walker(1997a)]
• P. Goulder, pers. comm.					
p17(71–79)	p17(71–79)	GSEELRSLY	HIV-1 infection	human(A1)	[Birk (1998)]
• 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(71–85)	p17(71–85 SF2)	GSEELRSLYNTVATL	HIV-1 infection	human( )	[Lieberman (1997a)]
• 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(74–82)	p17( )	ELRSLYNTV		human(B*0801,B8)	[Goulder (1997g)]
• Defined in a study of the B8 binding motif • Noted in Brander 1999, this database, to be B*0801					
p17(74–82)	p17(74–82)	ELRSLYNTV	HIV-1 infection	human(B8)	[Birk (1998)]
• 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					

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Ogg (1999)]
	• CTL effector levels were measured after potent ARV therapy using HLA-tetramer complexes for the A*0201 epitopes SLYNTVATL and ILKEPVHGV in seven patients, and the B*3501 epitope DPNPQEVVL in one additional patient • Levels of CTL effectors typically decline for 5-7 days and then rebound, fluctuating during the first two weeks of therapy • After the early fluctuation, there was a steady exponential decay with a median half-life of 45 days				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Altman (1996)]
	• 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				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Birk (1998)]
	• 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 SF2)	SLYNTVATL	HIV-1 infection	human(A*0201)	[McAdam (1998)]
	• 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)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Wilson (1998a)]
	• 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 *in vivo* • 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 patients CD8+ T cells				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Callan (1998)]
	• Included as a negative control in a tetramer study of A2-EBV CTL response				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Ogg (1998b)]
	• 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				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17( )	SLYNTVATL	HIV-1 infection	human(A2)	[Wagner (1998a)]
• 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)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A2)	[Collins (1998)]
• Two 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)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Durali (1998)]
• Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • Patient B18 had the greatest breadth and diversity of response, and recognized Gag SLYNTVATL and Nef PLTFGWCFKL					
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Kundu (1998b)]
• Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2 restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-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 SLFSAVAAL and no detectable CTL response					
p17(77–85)	p17(77–85 IIIB)	SLYNTVATL	HIV-1 infection	human(A2)	[Sipsas (1997)]
• HIV IIIB proteins were used to define the range of CTL epitopes recognized by 3 lab workers accidentally infected 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					

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17( )	SLYNTVATL	HIV-1 infection	human(A2)	[Rowland-Jones (1998a)]
• 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)	[Walter (1997)]
• HLA-A2 heavy chain and β2-microglobulin expressed in *E. coli* 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(77–85)	p17( )	SLYNTVATL	HIV-1 infection	human(A2)	[Sewell (1997)]
• 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---`, `-----I-V-`, `--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					
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang (1997b)]
• 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 transduced into CD8+ cells • The response using universal-receptor-bearing CD8+ cells to lyse infected cells *in vitro* 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					
p17(77–85)	p17(77–85)	SLYNTVATL	*in vitro* stimulation	human(A2)	[Stuhler & Schlossman(1997)]
• 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(A*0201)	[Lalvani (1997)]
• A peptide based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-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					

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(76–84)	SLYNTVATL	*in vitro* stimulation	human(A*0201)	[van der Burg (1996)]
	• Slow dissociation rate is associated with immunogenicity • CTL generated by *in vitro* stimulation of PBMC derived from uninfected individual				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang (1996)]
	• 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				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang (1997a)]
	• CTL inhibit HIV-1 replication at effector cell concentrations comparable to those found *in vivo* • 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)	p17(77–85 LAI)	SLYNTVATL	HIV-1 infection	human(A2)	[Parker (1992), Parker (1994)]
	• Examined in the context of motifs important for HLA-A2 binding				
p17(77–85)	p17(77–85 LAI)	SLYNTVATL	HIV-1 infection	human(A2)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Tsomides (1994)]
	• CTL clones recognize naturally processed peptide				
p17(77–85)	p17(77–85)	SLYNTVATL	Peptide stimulation *in vitro*	human(A2)	[Stuhler & Schlossman(1997)]
	• 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)	[Cao (1997)]
	• 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				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Goulder (1997b), Goulder (1997a)]
• 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 • 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 • [Goulder (1997a)] is a review of immune escape that summarizes this study					
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(B62)	[Goulder (1997a)]
• 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 • 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 response shifted to a focus on GLNKIVRMY, and the index peptide SLYNTVATL once again established itself as the dominant form					
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Gray (1999)]
• Peptide-tetramer complexes of A*0201 and SLYNTVATL or ILKEPVHGV were used to study individuals receiving HAART to determine the frequency of Class I HLA-restricted anti-HIV CD8+ T cells • 17/18 asymptomatic patients had CTL to one or both epitopes – 72% had a CTL response to SLYNTVATL • After HAART, the majority of the epitope-specific CTL were apparently memory cells					
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Dyer (1999)]
• CTL specific responses were measured over a 1.5- to 1.3-year period in members of the Sydney Blood Bank Cohort (SBBC) who had been infected with a natural attenuated strain of HIV-1 which was nef-defective. Some of these patients had prolonged high levels of CTL effector and memory cells despite low viral load.					
p17(77–85)	p17( )	SLYNTVATL	HIV-1 exposure	human(A2, A*0202)	[Rowland-Jones (1998b)]
• 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 B and D clade viruses • The Clade A version of the epitope, SLFNTVATL, was preferentially recognized by CTL • This epitope was recognized by two different exposed seronegative prostitutes					

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85 Clade A)	SLFNTVATL	HIV-1 infection	human(A*0201)	[Dorrell (1999)]
• 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					
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Brander (1998a)]
• Of 17 infected HLA A*0201 subjects, 13 had CTL responses against the p17 SLYNTVATL, epitope, six recognized ILKEPVHGV and five recognized VIYQYMDDL, and there was no correlation between viral load and recognition of a specific epitope • Only one subject had CTL against all three • There was significant heterogeneity in the CTL response to this immunodominant epitope • The overall variation in this epitope among the 17 who had a CTL response and 11 non-HLA A*0201 HIV-1+ individuals was similar, suggesting a lack of immune pressure • Subjects were part of the San Fransisco City Clinic Cohort, the ARIEL project and from the Boston area					
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Harrer (1998)]
• Two overlapping epitopes were recognized in a long term survivor, restricted by two different HLA molecules, HLA-A11(TLYCVHQR) and -A2 (SLYNTVATL) • Viral sequence substitutions were present in this individual which did not affect viral replication and did not alter CTL-recognition of the A2 epitope, but reduced recognition of the A11 epitope, indicative of immune escape					
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Hay (1999)]
• CTL response to IPRRIRQGL was the immunodominant response in a rapid progressor – there was an subdominant response to SPAIFQSSM in Pol, and interestingly, no response to commonly immunodominant HLA A*0201 epitope SLYNTVATL, although this individual was HLA-A*0201 • The individual showed a strong initial CTL response at the time of the initial drop in viremia, but it was quicly lost, although memory cells persisted • Despite the initial narrow response to two epitopes, no other CTL responses developed • No HIV-specific lymphoproliferative responses were detected in this patient, and neutralizing antibody response was weak • A variant of this epitope was observed *in vivo* (`--F----V-`), but this mutation is recognized by SLYNTVATL-specific CTL, and in this case the patients own cells could present the peptide to SLYNTVATL-specific CTL					
p17(77–85)	p17(77–85)	SLYNTVATL		human(A*0202)	
• C. Brander *et al*. in this database 1999, note that this epitope can be presented by A*0201 and A*0202					

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(84–91)	p17(83–91)	TLYCVHQR	HIV-1 infection	human(A11)	[Harrer (1998)]
	• Two overlapping epitopes were recognized in a long term survivor, restricted by two different HLA molecules, HLA-A11(TLYCVHQR) and -A2 (SLYNTVATL) • Viral sequence substitutions were present in this individual which did not affect viral replication and did not alter CTL-recognition of the A2 epitope, but reduced recognition of the A11 epitope, indicative of immune escape • A Q90E substitution resulted in a loss of the ability of the peptide to induce lysis, a R91K substitution was still reactive, and a R91Q substitution show a reduced ability to stimulate lysis				
p17(84–92)	p17(84–92)	TLYCVHQRI	HIV-1 infection	human(A11)	[Brander & Walker(1995)]
	• 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)	[Birk (1998)]
	• 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 • C. Brander notes that this is a A*1101 epitope in the 1999 database				
p17(87–105)	p17(91–105 SF2)	CRIDVKDTKEALEKIE	HIV-1 infection	human( )	[Lieberman (1997b)]
	• CTL expanded *ex vivo* were later infused into HIV-1 infected patients				
p17(88–115)	p17(88–115 ARV)	VHQRIEIKDTKEALDKI-EEEQNKSKKKA	HIV-1 infection	human(A2)	[Achour (1990)]
	• B cell epitope HGP-30 also serves as a CTL epitope				
p17(88–115)	p17(88–115 ARV)	VHQRIEIKDTKEALDKI-EEEQNKSKKKA	Combination peptide vaccine	murine BALB/c(H-2^d)	[Hamajima (1997)]
	• 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)	p17(91–105 SF2)	RIDVKDTKEALEKIE	HIV-1 infection	human( )	[Lieberman (1997a)]
	• 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				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(92–101)	p17( )	IEIKDTKEAL	HIV-1 infection	human(B60)	[Wagner (1998a)]
• 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(92–101)	p17( )	IEIKDTKEAL	HIV-1 infection	human(B*4001,B60)	
• Noted by C. Brander *et al*., this database 1999, to be a B*4001,B60 epitope, Pers. Comm. A. Trocha and S. Kalams					
p17(93–101)	p17(93–101)	EIKDTKEAL	no CTL shown	human(B8)	[DiBrino (1994b)]
• Examined in the context of motifs important for HLA-B8 binding, predicted epitope based on Achour *et al*.					
p17(93–101)	p17(93–101)	EIKDTKEAL	HIV-1 infection	human(B8)	[Birk (1998)]
• 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)	p17(93–101 LAI)	EIKDTKEAL		human(B8, B60)	[Brander & Walker(1997b)]
• Pers. Comm. from A. Trocha and S. Kalaams to C. Brander and B. Walker					
p17(121–132)	p17(121–132 HXB2R)	DTGHSNQVSQNY	HIV-1 infection	human(A33)	[Buseyne (1993b)]
• Clustering of Gag p24 CTL epitopes recognized in 29 HIV infected people					
p17(121–132)	Gag(121–132 LAI)	DTGHSNQVSQNY	HIV-1 infection	human(A33)	[Buseyne (1993a)]
• Vertical transmission of HIV ranges from 13% to 39% • Primary assays showed cytotoxic activities against at least one HIV protein was detected in 70% of infected children • Epitopes recognized in five children were mapped using synthetic peptides and secondary cultures • Patient EM17 (CDC P2A+C+D2) had a CTL response to two epitopes in Gag					
p17(124–132)	p17(124–132 LAI)	NSSKVSQNY	HIV-1 infection	human(B35)	[McMichael & Walker(1994)]
• Review of HIV CTL epitopes					
p17(124–132)	p17(124–132)	NSSKVSQNY	HIV-1 infection	human(B35)	[Birk (1998)]
• 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					

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(124–132)	p17(124–132 LAI)	NSSKVSQNY	HIV-1 or -2 infection	human(B*3501)	[Rowland-Jones (1995)]
	• Established by titration • Noted in Brander 1999, this database, to be B*3501				
p17(124–132)	p17(124–132 LAI)	NSSKVSQNY	none	human(B35)	[Lalvani (1997)]
	• A peptide based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-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				
p17(124–132)	p17( )	NSSKVSQNY		human(B35)	[Rowland-Jones (1999)]
	• CTL responses in seronegative highly HIV-exposed African female sex workers in Gambia and Nairobi were studied – these women had no delta 32 deletion in CCR5 • In Gambia there is exposure to both HIV-1 and HIV-2, CTL responses to B35 epitopes in exposed uninfected women are cross-reactive, • HIV-2 version of this epitope is not conserved: PPSGKGGNY, but the CTLs are cross-reactive – this is one of five B35 CTL epitopes that are cross-reactive, see also [Rowland-Jones (1995)]				