**Table 2: CTL epitopes conserved among HIV-1 proteomes**

| EPITOPE | C-(1) | Source (2) | Position (3) | HLA I Restriction (4) | Predicted HLA I Restriction (5) | PPC(6) |
|---|---|---|---|---|---|---|
| SPRTLNAWV | K | GAG:p24 | 148–156:16–24 | B0702 | B0702 B3501 B5101 B5102 B5103 B5301 B5401 B5502 | 0.35 |
| AVFIHNFKR | K | POL:Integrase | 894–902:179–187 | A0301 | A0301 A1101 A3101 A3301 A6601 A6801 | 0.35 |
| TLFCASDAK | A | ENV:gp160 | 51–59 | A0301 | A0301 A1101 A3101 A3301 A6801 | 0.32 |
| FPVRPQVPL | R | NEF | 68–76 | B3501 | A2902 B0702 B3501 B5101 B5102 B5103 B5301 B5401 | 0.32 |
| RAMASDFNL | P | POL:Integrase | 735–743:20–28 | A0201 | A0201 B2709 C0304 | 0.31 |
| TLNAWVKVI | E | GAG:p24 | 151–159:19–27 | A0201 | A0201 A0202 A0203 A0204 A0206 | 0.29 |
| VIYQYMDDL | Y | POL: Reverse Transcriptase | 334–342:179–187 | A0201 | A0201 A0205 A0207 A0214 | 0.28 |
| LVGPTPVNI | I | POL:Protease | 132–140:76–84 | A0201 | A0201 A0202 A0205 A0209 B1501 B1516 | 0.27 |
| TVLDVGDAY | F | POL: Reverse Transcriptase | 262–270:107–115 | B3501 | B1501 B3501 B5701 C0304 | 0.26 |
| PLVKLWYQL | E | POL: Reverse Transcriptase | 576–584:421–429 | A0201 | A0201 A0202 A0203 | 0.26 |
| TLNFPISPI | E | POL: Protease | 152–160:96–1004 | | A0201 A0207 | 0.23 |
| NTPVFAIKK | K | POL: Reverse Transcriptase | 212–220:57–65 | A0301 | A0301 A6601 C0102 | 0.22 |
| EKEGKISKI | G | POL: Reverse Transcriptase | 197–205:42–50 | B5101 | B2701 B3801 B39011 B3909 B4402 B5101 B8 | 0.19 |
| LLWKGEGAV | V | POL: Reverse Transcriptase | 956–964:241–249 | A0201 | A0201 A0204 A0205 A0209 | 0.18 |
| LTFGWCFKL | V | NEF | 137–145 | A0201 | A0201 | 0.18 |
| YQYMDDLYV | G | POL: Reverse Transcriptase | 336–344:181–189 | A0201 | A0201 | 0.18 |
| GPKVKQWPL | T | POL: Reverse Transcriptase | 173–181:18–26 | B0801 | B0702 B0801 B3501 B8 | 0.17 |
| RAIEAQQHL | L | ENV:gp41 | 557–565:46–54 | B5101 B1501 C0304 | B1501 B1517 B5101 C0304 | 0.13 |
| GLNKIVRMY | S | GAG:p24 | 269–277:137–145 | B1501 | A0203 A1 B1501 | 0.13 |
| YFPDWQNYT | P | NEF | 120–128 | A1 B3701 B5701 | A1 B3701 B5701 | 0.07 |
| WYIKIFIMI | V | ENV:gp41 | 680–688:680–688 | A2402 | A0203 A0206 A2402 | 0.05 |
| YVDRFFKTL | R | GAG:p24 | 296–304:164–172 | A2601 | A0203 A0204 A0207 A2601 B3801 | 0.05 |
| FVNTPPLVK | L | POL: Reverse Transcriptase | 571–579:416–424 | A1101 | A1101 | 0.05 |
| KIQNFRVYY | R | POL:Integrase | 934–942:219–227 | A3002 | A1 A3002 | 0.03 |
| DRFFKTLRA | E | GAG:p24 | 298–306:166–174 | B1402 | B1402 B2701 B2702 B2703 B2704 B2705 B2709 | 0.03 |

1) Most proximal C-terminal flanking amino acid residue to the epitope
2) HIV-1 ORF
3) Position in mature protein.
4) HLA-1 restriction of the epitope as retrieved from the Los Alamos HIV database
5) Predicted HLA-1 binding profiles of epitopes were obtained using PSSMs [16].
6) Protection Population Coverage (PPC) was computed for 5 ethnic groups (Black, Caucasian, Hispanic, North American Natives and Asian) in the USA population. Each PPC value shown in the table is that of the ethnic group with the lowest PPC for that specific HLA I combination.

LVGPTPVNI is predicted to bind to several alleles within the A2 supertype (A0201 A0202 A0205 A0209) but additionally to other non-related alleles of the HLA-B locus (B2701 and B3801).

In order to assess the accuracy of the predicted PPC, five peptide pools were created, each solubilized in DMSO and combining CTL epitopes (200 μM each) to provide a PPC ≥ 95 % (peptides included in the pools are shown in Table 3). Peptide 1 pool contained all 25 selected peptides whereas peptide pools #2 (15 peptides), #3 (13 peptides), #4 (11 peptides), #5 (7 peptides) comprising 55, 13, 11, 5, 2, respectively, of distinct 5-peptide combinations producing ≥ 95% PPC. Peptides pools 1–5 were designed so that peptides in the smaller pools are contained in the larger pools. CD8+ T cell responses to these pools were first checked by IFNγ ELISPOT assay using PBMC samples from a cohort of 47 HIV-1 infected patients. Patients were largely heterogeneous with regard to their HLA I background and included those diagnosed during acute or chronic HIV-1 with or without HAART therapy and some long-term non-progressors (Table 4). Furthermore, these