Supporting Information

Peptidomimetic Inhibitors Targeting the CCR5-Binding Site on the Human Immunodeficiency Virus Type-1 gp120 Glycoprotein Complexed to CD4

Markus Seitz, Peter Rusert, Kerstin Moehle, Alexandra Trkola
and John A. Robinson

Contribution from the Institute of Organic Chemistry, University of Zurich, Winterthurerstrasse 190, 8057 Zurich, and Institute of Medical Virology, University of Zurich, Winterthurerstrasse 190, 8057 Zurich, Switzerland.

1. Peptide synthesis

Peptide 1

The tyrosine sulfated peptide **1** was synthesized by solid phase methods using standard Fmoc chemistry and an Applied Biosystems 433A peptide synthesizer. Fmoc-Pro-OH (0.25 mmol) was coupled to 2-chlorotritylchloride resin (1000 mg, loading = 0.39 mmol/g) in the presence of diisopropylethylamine (DIPEA, 4 eq.) in CH_2Cl_2 (10 mL). The unreacted sites on the resin were capped by washing with a mixture of CH_2Cl_2/MeOH/DIPEA (17:2:1) followed by MeOH. After removal of the Fmoc-group using 20% piperidine in N-methyl-2-pyrrolidinone (NMP), chain elongation was performed with Fmoc-D-Pro-OH, Fmoc-Ile-OH, Fmoc-Glu(tBu)-OH, Fmoc-Phe-OH, Fmoc-Thr(tBu)-OH, Fmoc-Gly-OH, Fmoc-Tys-OH, Glu(tBu)-OH and Fmoc-Tys-OH (each 1 mmol, except Fmoc-Tys-OH 0.5 mmol), using 20% piperidine/NMP for Fmoc deprotection, 2-(1H-benzotriazole-1-yl)-1,1,3,3-tetramethyluronium hexafluoro-phosphate/1-hydroxybenzotriazole (HBTU/HOBt) for activation, DIPEA as base and NMP as solvent. After assembly of the linear peptide chain, the peptide was transferred with CH_2Cl_2 into a sintered glass funnel. The peptide was cleaved from the resin by adding 5 times ice cold 0.8 % CF_3COOH in CH_2Cl_2 (5 mL) for one minute. The eluate was neutralized immediately with DIPEA (1 mL). The resin was washed sequentially with CH_2Cl_2, MeOH, CH_2Cl_2, MeOH, CH_2Cl_2. Subsequently the solvent was removed under high vacuum. For cyclization, the resulting crude product was dissolved in dimethylformamide (DMF, 200 mL) and 2-(7-aza-1H-benzotriazole-1-yl)-1,1,3,3-tetramethyluronium hexafluorophosphate (HATU, 0.75 mmol, 285.2 mg, 3 eq.), 1-hydroxy-7-azabenzotriazole (HOAt, 0.75 mmol, 102 mg, 3 eq.) and DIPEA (1.5 mmol, 130 μl, 6 eq.) were added. The reaction was stirred under argon for 18 hours then the DMF was removed under high vacuum. To remove excess of coupling agents the crude product was dissolved in CH_2Cl_2 (20 ml) and extracted with 10 % MeCN in water. After evaporation, the crude peptide was cooled on ice before adding pre-cooled ice cold 90 % CF_3COOH, 10 % H_2O mixture (10 mL). The peptide was stirred for 5.5 h in an ice-water bath and then precipitated with ice cold diisopropyl ether (100 mL). After washing the precipitate twice with diisopropyl ether, the peptide was dried and purified by preparative HPLC on a *Waters XBridge*™ (C18, 50 x 19 mm, 5 μm, 135 Å) column with a

gradient of 15-60 % MeCN in H_2O (0.1 M NH_4OAc) in 10 column volumes. Retention time on analytical RP-HPLC (*GraceVydac 218TP54*, C18, 250 x 4.6 mm, 5 μm, 300 Å) with a linear gradient from 5-100 % MeCN in H_2O (0.1 M NH_4OAc) in 25 min at a flow rate of 1 ml/min was 10.1 minutes. ESI MS (*neg.*): *m/z*: 1355.5 $[M-H]^-$, calc. 1356.5 (monoisotopic).

Peptide 2

The peptide **2** was synthesized by solid phase methods using standard Fmoc chemistry and an Applied Biosystems 433A peptide synthesizer. Fmoc-Pro-OH (0.25 mmol) was coupled to 2-chlorotritylchloride resin (1000 mg, loading = 0.39 mmol/g) in the presence of DIPEA (4 eq) in CH_2Cl_2 (10 mL). The unreacted sites on the resin were capped by washing with a mixture of CH_2Cl_2/MeOH/DIPEA (17:2:1) followed by MeOH. After removal of the Fmoc-group using 20% piperidine in NMP, chain elongation was performed with Fmoc-D-Pro-OH, Fmoc-Ile-OH, Fmoc-Glu(tBu)-OH, Fmoc-Phe-OH, Fmoc-Thr(tBu)-OH, Fmoc-Gly-OH, Fmoc-Tyr(tBu)-OH, Glu(tBu)-OH and Fmoc-Tyr(tBu)-OH (each 1 mmol), using 20% piperidine/NMP for Fmoc deprotection, HBTU/HOBt for activation, DIPEA as base and NMP as solvent. After assembly of the linear peptide chain, the peptide was transferred with CH_2Cl_2 into a sintered glass funnel. The peptide was cleaved from the resin by adding 5 times ice cold 0.8 % TFA in DCM (5 mL) for one minute. The eluate was neutralized immediately with DIEA (1 mL). The resin was washed sequentially with CH_2Cl_2, MeOH, CH_2Cl_2, MeOH, CH_2Cl_2. Subsequently the solvent was removed under high vacuum. For cyclization, the resulting crude product was dissolved in DMF (200 mL) and HATU (0.75 mmol, 285.2 mg, 3 eq.), HOAt (0.75 mmol, 102 mg, 3 eq.) and DIPEA (1.5 mmol, 130 μl, 6 eq.) were added. The reaction was stirred under argon for 18 h then the DMF was removed under high vacuum. Side chain deprotection was carried out by adding CF_3COOH/H_2O/triisopropysilane (95:2.5:2.5) (10 mL) and shaking for 3 h at room temperature. After evaporation under high vacuum and peptide precipitation with ice cold Et_2O the peptide was dried and purified by preparative HPLC on a *Waters XBridge*™ (C18, 50 x 19 mm, 5 μm, 135 Å) column with a gradient of 15-60 % MeCN in H_2O (0.1 M NH_4OAc) in 10 column volumes. Retention time on analytical RP-HPLC (*GraceVydac 218TP54*, C18, 250 x 4.6 mm, 5 μm, 300 Å) with a linear gradient from 5-100 % MeCN in H_2O (0.1 M NH_4OAc) in 25 min at a flow rate of 1 ml/min was 14.1 min. ESI MS (*neg.*): *m/z*: 1195.6 $[M-H]^-$, calc. 1196.5 (monoisotopic). For more analytical data see below.

2. ^{1}H NMR solution conformation of peptide 1

^{1}H NMR experiments were performed on a Bruker AV-600 spectrometer at a peptide concentration of 5 mg/ml in either 90% H_2O-10% D_2O, or 100% D_2O, with a pH of 4.9 at 300 K. Water suppression was achieved by presaturation. 2D DQF-COSY, TOCSY and ROESY spectra were recorded to determine assignments (given in Table-S1. Distance restraints were obtained from ROESY with mixing time of 250 ms. Spectra were typically collected with 2048 x 512 complex data points zero-filled prior to Fourier transformation to 2048 x 1024, and transformed with a cosine-bell weighting function. Information on the relative rates of slowly exchanging amide protons was obtained by acquisition of a series of 1D spectra upon dissolution of **1** in D_2O. $^3J_{HN\text{-}Ha}$ coupling constants were determined from one-dimensional spectra. A series of 1D spectra were measured at different temperatures over the range of 278-330 K and temperature coefficients of the amide protons were calculated. Spectra were processed using TOPSPIN (*Bruker*) and further analysed using the program XEASY[1].

Table S1. ^{1}H NMR assignments for peptide 1 (in 90% H_2O:10% D_2O solution, pH 5.0, 300 K)

Residue	NH	C(α)-H	C(β)-H	Others
Tys[1]	7.98	4.54	3.04, 2.82	C(δ)H=7.26, C(ε)H=7.15
Glu[2]	8.28	4.48	1.89, 1.80	C(γ)H=2.13
Tys[3]	8.40	4.54	2.80, 2.18	C(δ)H=6.97, C(ε)H=7.05
Gly[4]	8.70	4.11, 3.43	-	-
Thr[5]	8.04	4.21	4.36	C(γ)H=1.07
Phe[6]	7.67	4.16	3.12, 3.03	C(δ)H=7.09, C(ε)H=6.99, C(ξ)H=7.09
Glu[7]	8.35	4.89	1.94, 1.87	C(γ)H=2.21
Ile[8]	8.56	4.53	1.94	C(γMe)H=0.82, C(γ)H=1.45, 1.05, C(δ)H=0.73
D-Pro[9]	-	4.52	2.16, 1.74	C(γ)H=2.04, 1.91, C(δ)H=3.50, 3.75
L-Pro[10]	-	4.30	1.83, 1.55	C(γ)H=1.60, 0.76, C(δ)H=3.51, 3.40

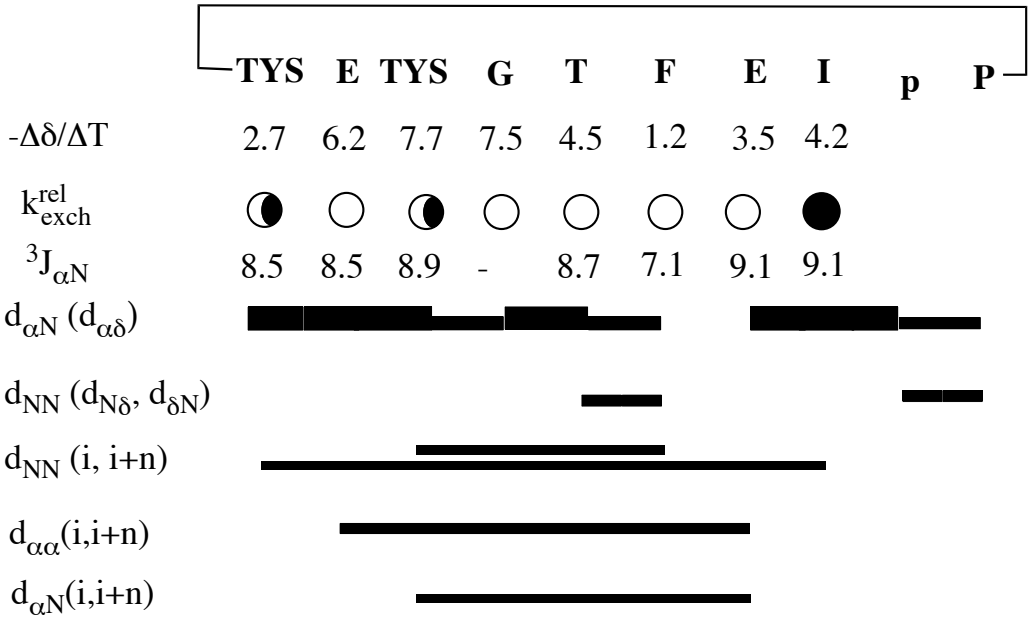
^{3}J(Cα,N) Coupling constants show predominantly values > 8 Hz within the ß-strands (Figure S1). Temperature coefficients with values lower than 5 ppb/K, and slower H-D exchange rates of the amide protons, indicating shielding of these protons from the bulk solvent, were observed at residues occupying hydrogen-bonding positions in the ß-hairpin structure, especially, those close to the D-Pro-L-Pro template (Figure S1).

The structure calculations were performed by restrained molecular dynamics in torsion angle space by applying the simulated annealing protocol implemented in the program DYANA[2]. Starting from 100 randomized conformations a bundle of 20 conformations will be selected, which have the lowest DYANA target energy function. The program MOLMOL[3] was used for structure analysis and visualization of the calculated averaged structure models. Statistics derived from the results are given in Table-S2. The structures were determined using 87 NOE upper distance limits (see below). The final ensemble of 20 structures with a backbone rmsd of 0.54 Å reveals a highly populated ß-hairpin conformation in solution in which the two strands of antiparallel ß-sheet are connected by a type II turn between Gly4 and Thr5 (Figure 1). The close contact of the two strands is reflected by characteristic backbone cross strand NOEs, for example between HN-HN, Hα-Hα and Hα-HN, respectively, shown in Figure S1.

Table S2. Experimental distance restraints and statistics for the final 20 NMR structures calculated for **1**.

	Peptide 1
NOE upper-distance limits	87
• Intraresidue	25
• Sequential	28
• Medium- and long-range	34
Residual target function value (Å^2)	0.45 ± 0.01
Mean rmsd values (Å)	
• All backbone atoms	0.54 ± 0.22
• All heavy atoms	1.62 ± 0.24
Residual NOE violation	
• Number > 0.2 Å	0
• Maximum (Å)	0.11

Figure S1. Amide HN temperature coefficients (in ppb/K), relative H-D exchange rates of peptide amide protons (black circles: slow; half-white circles: medium; and white circles: fast), backbone $^3J(C\alpha,N)$ values and characteristic backbone NOE connectivies (thick line: strong, medium line: medium, and thin line: weak) measured for peptide **1**.



3. List of ROE connectivities observed for peptide 1

INTRA-RESIDUE NOE UPPER-DISTANCE LIMITS

1	TYS	HN	1	TYS	QB	3.54
1	TYS	HA	1	TYS	QB	2.72
2	GLU-	HN	2	GLU-	HB2	3.27
2	GLU-	HN	2	GLU-	HB3	3.27
2	GLU-	HN	2	GLU-	QG	6.10
3	TYS	HN	3	TYS	HB2	3.95
3	TYS	HN	3	TYS	HB3	3.95
3	TYS	HN	3	TYS	QB	3.55
4	GLY	HN	4	GLY	HA1	2.71
5	THR	HN	5	THR	QG2	4.85
6	PHE	HN	6	PHE	HB2	3.73
6	PHE	HN	6	PHE	HB3	3.73
6	PHE	HN	6	PHE	QB	3.43
7	GLU-	HN	7	GLU-	HB2	3.58
7	GLU-	HN	7	GLU-	HB3	3.58
7	GLU-	HN	7	GLU-	QB	3.33
7	GLU-	HN	7	GLU-	QG	6.13
7	GLU-	HA	7	GLU-	HB2	3.02
7	GLU-	HA	7	GLU-	HB3	3.02
8	ILE	HN	8	ILE	HB	3.02
8	ILE	HN	8	ILE	QG1	5.88
8	ILE	HA	8	ILE	HB	2.70
8	ILE	HA	8	ILE	HG12	4.17
8	ILE	HA	8	ILE	HG13	4.17
8	ILE	HA	8	ILE	QG1	3.81

SEQUENTIAL NOE UPPER-DISTANCE LIMITS

1	TYS	HN	10	PRO	HA	3.55
1	TYS	HN	10	PRO	QD	4.84
1	TYS	HA	2	GLU-	HN	2.99
1	TYS	QB	2	GLU-	HN	4.42
1	TYS	QD	10	PRO	QD	7.44
1	TYS	QE	10	PRO	QD	7.67
2	GLU-	HA	3	TYS	HN	2.80
2	GLU-	QG	3	TYS	HN	6.38
3	TYS	HN	4	GLY	HN	3.86
3	TYS	HA	4	GLY	HN	3.36
4	GLY	HA2	5	THR	HN	2.68
5	THR	HN	6	PHE	HN	3.11
5	THR	HN	6	PHE	QD	7.62
5	THR	HA	6	PHE	HN	3.61
5	THR	HB	6	PHE	HN	4.23
6	PHE	HB2	7	GLU-	HN	4.14
6	PHE	HB3	7	GLU-	HN	4.14
6	PHE	QB	7	GLU-	HN	3.65
6	PHE	QD	7	GLU-	HN	7.62
7	GLU-	HA	8	ILE	HN	2.93
7	GLU-	QG	8	ILE	HN	6.38
8	ILE	HA	9	DPR	HD3	2.99
8	ILE	HA	9	DPR	HD2	2.99
8	ILE	HA	9	DPR	QD	2.75
8	ILE	QG2	9	DPR	QD	6.16
9	DPR	HA	10	PRO	HD2	3.39
9	DPR	HA	10	PRO	HD3	3.39
9	DPR	HA	10	PRO	QD	3.11

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MEDIUM and LONG RANGE NOE UPPER-DISTANCE LIMITS
 1 TYS  HN      8 ILE  HN      4.14
 1 TYS  HN      8 ILE  QD1     5.72
 1 TYS  HB2     3 TYS  QE      6.59
 1 TYS  HB2     8 ILE  QD1     6.22
 1 TYS  HB3     3 TYS  QE      6.59
 1 TYS  HB3     8 ILE  QD1     6.22
 1 TYS  QB      3 TYS  QE      6.44
 1 TYS  QB      8 ILE  HN      5.91
 1 TYS  QB      8 ILE  HB      4.42
 1 TYS  QB      8 ILE  QG2     6.87
 1 TYS  QB      8 ILE  QD1     6.06
 1 TYS  QD      8 ILE  HB      7.29
 1 TYS  QD      8 ILE  QG2     8.20
 1 TYS  QD      8 ILE  QD1     8.32
 1 TYS  QE      8 ILE  QG2     8.70
 1 TYS  QE      8 ILE  QD1     8.61
 2 GLU- HA      7 GLU- HA      3.24
 2 GLU- QG      4 GLY  HN      6.38
 3 TYS  HN      6 PHE  HN      4.42
 3 TYS  HN      7 GLU- HA      4.20
 3 TYS  QB      6 PHE  HN      4.91
 3 TYS  QD      7 GLU- HA      7.67
 3 TYS  QD      8 ILE  HN      7.67
 3 TYS  QD      8 ILE  QD1     8.69
 3 TYS  QE      8 ILE  HN      7.68
 3 TYS  QE      8 ILE  HB      6.19
 3 TYS  QE      8 ILE  QG2     8.70
 3 TYS  QE      8 ILE  QD1     8.27
 6 PHE  QD      8 ILE  QG2     8.65
 6 PHE  QD      8 ILE  HG12    6.94
 6 PHE  QD      8 ILE  HG13    6.94
 6 PHE  QD      8 ILE  QG1     6.58
 6 PHE  QD      8 ILE  QD1     7.25
 6 PHE  QE      8 ILE  QD1     8.65
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4. BIAcore sensorgrams

Surface plasmon resonance experiments were performed on a *BIAcore3000* (*Biacore, Uppsala*). All measurements were carried out at 25 °C using HBS buffer (10 mM HEPES, 150 mM NaCl, 3.4 mM EDTA, 0.005 % P20, pH 7.4) as running buffer. All solutions were filtered using a 0.2 μm filter.

Assays with immobilized mAb 17b

400 RU of mAb 17b were linked covalently to a CM5 chip by random amine coupling as described in the manufacturer's protocol. Then various concentrations of gp120 (JR-FL) ranging from 0-100 nM were pre-incubated with a 5-fold molar excess of CD4-IgG_2 in running buffer (10 mM HEPES, 150 mM NaCl, 3.4 mM EDTA, 0.005 % P20, pH 7.4) at room temperature for at least 30 min. After 5 minutes stabilization time the samples were injected at a flow rate of 25 μl/min for 10 min. After 10 min dissociation the surface was regenerated by two 60 s pulses of 10 mM NaOH solution in water at a flow rate of 10 μl/min. Regeneration was followed by a 5 min regeneration time before start of the next cycle.

Recorded data were fitted to a 1:1 Langmuir binding model (kinetic fit) using the BIAevaluation software, which gave the on and off rates, k_{on} = 2.29*10^4 M^{-1}s^{-1}, k_{off} = 5.75*10^{-5} s^{-1} and a dissociation constant K_D = 2.51 nM with a χ^2 of 4.15 at a maximum response of 200 RU. BIAcore responses are shown in Figure S2.

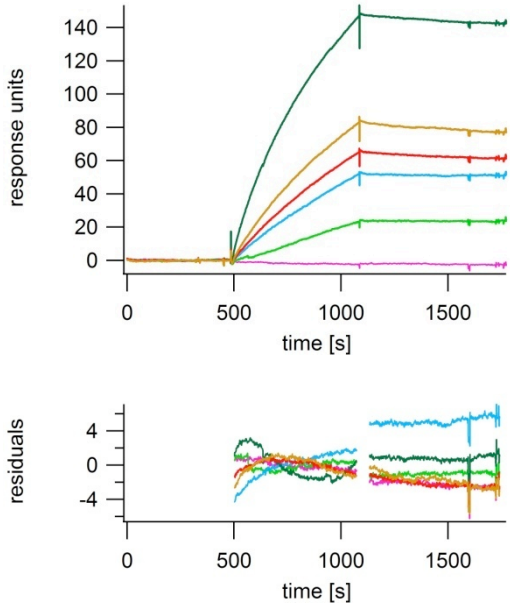

Figure S2. BIAcore response upon direct binding of gp120/CD4-IgG$_2$ complex to immobilized mAb 17b. Gp120 concentration from top to bottom: 100 nM, 40 nM, 30 nM 20 nM, 10 nM, 0 nM.

For IC_{50} determinations, gp120 (JR-FL) (50 nM) was pre-incubated with CD4-IgG$_2$ (250 nM) and various concentration (0-1000 nM) of either peptide **1** (Figure S3) or **2** (Figure S4) in 10 mM HEPES, 150 mM NaCl, 3.4 mM EDTA, 0.005 % P20, pH 7.4. After 5 min stabilization time the samples were injected at a flow rate of 25 μl/min for 10 min. After a 10 min dissociation period, the surface was regenerated by two 60 s pulses of 10 mM NaOH solution in water at a flow rate of 10 μl/min. Regeneration was followed by a 5 min regeneration time before starting the next cycle. For analysis, the maximum RUs observed at the end of the injection period were plotted against the logarithm of the peptide concentration in μM. Curve fitting to a sigmoid binding model was performed by using *IGORpro* software (*WaveMetrics, Lake Oswego, OR, USA*). The resulting IC_{50} values were 25 μM for peptide **1**, and 400 μM for peptide **2**.

Assays with immobilized gp120

400 RU gp120 (JR-FL) were linked covalently to a CM5 chip by random amine coupling as described in the manufacturer's protocol. Then various concentrations of CD4-IgG$_2$ ranging from 0-125 nM in 10 mM HEPES, 150 mM NaCl, 3.4 mM EDTA, 0.005 % P20, pH 7.4 were injected at room temperature. After 5 min stabilization time, the samples were injected at a flow rate of 25 μl/min for 10 min. After 10 min dissociation period, the surface was regenerated by a 5 s pulses of 100 mM aqueous H_3PO_4 at a flow rate of 25 μl/min. Regeneration was followed by a 5 min regeneration time before starting the next cycle. Recorded data were fitted to a bivalent binding model (standard model for IgG type antibodies) using BIAevaluation software, which gave k_{a1}= 1.1*10^5 M^{-1}s^{-1}, k_{d1} = 5.76*10^{-5} s^{-1}, k_{a2}= 1.13*10^{-3} M^{-1}s^{-1}, k_{d2} = 4.03*10^{-3} s^{-1}, and a K_D = 2.51 nM with a Chi^2 of 0.886 at a maximum response of 46.3 RU. Resulting curves are shown in Figure S5.

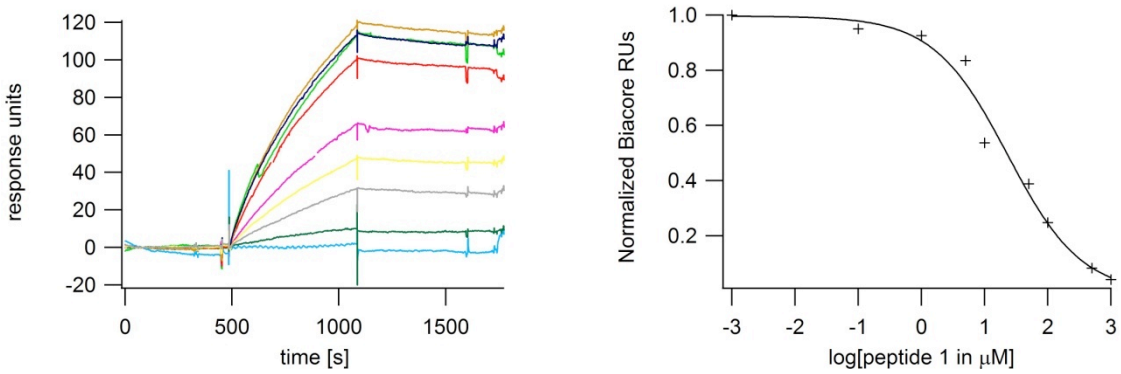

Figure S3. Inhibition of gp120/CD4-IgG$_2$ binding to mAb 17B by peptide 1. The peptide 1 concentrations from top to bottom: 0 nM, 0.1 nM, 1 nM, 5 nM, 10 nM, 50 nM, 100 nM, 500 nM, 1000 nM.

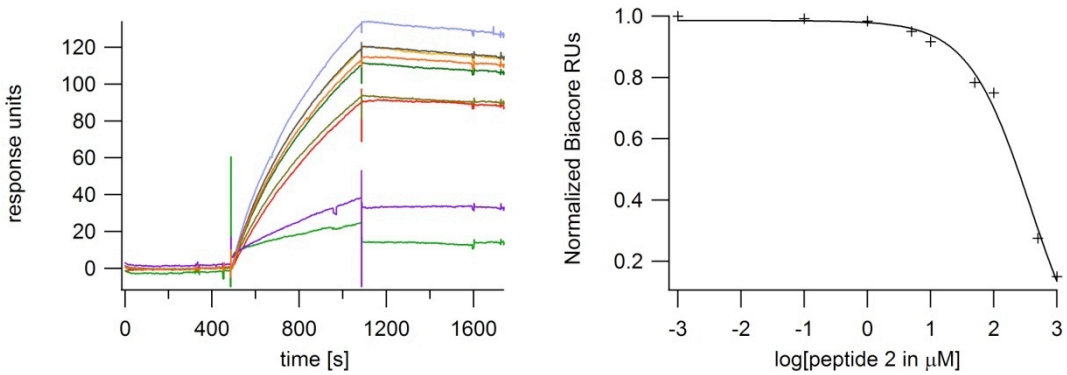

Figure S4. Inhibition of gp120/CD4-IgG$_2$ binding to 17B by peptide 2 (both Tys residues replaced by Tyr). Peptide 2 concentrations from top to bottom: 0 nM, 0.1 nM, 1 nM, 5 nM, 10 nM, 50 nM, 100 nM, 500 nM, 1000 nM.

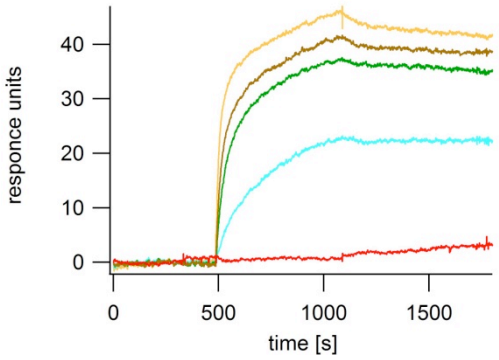

Figure S5. Direct binding of CD4-IgG$_2$ to gp120. CD4-IgG$_2$ concentrations from top to bottom: 125 nM, 62.5 nM, 31.25 nM 7.8 nM, 0 nM.

5. HIV-1 inhibition assay

Cell lines
The following cell lines were used: For transfection: 293-T (ATCC); TZM-bl cells[5-7] (obtained through the NIH AIDS repository).

Reagents: sCD4 (Deen et al, 1988, Nature) and CD4IgG2 (Allaway et al. 1995, AIDS Res Hum Retroviruses) were provided by W. Olson (Progenics) and the monoclonal antibody 17b was obtained through the NIH AIDS Research and Reference Reagent Program, from Dr. James E. Robinson.

Generation of env-pseudotyped HIV particles
293-T cells were transfected with plasmids carrying the reporter gene expressing the virus backbone, pNLluc-AM[8] (a kind gift from A. Marozsan and J. P. Moore), and the functional envelope clone at a ratio of 3:1 polyethylenimine (PEI, linear 25kDa, Polysciences)[9] as described[10]. Viral supernatants were harvested 2 days post transfection and the TCID50 determined on TZM-bl cells by end point dilution. To this end, TZM-bl cells were infected with viral supernatants in DMEM, 10% FCS, and 1% penicillin-streptomycin (BioWhittaker) containing 10 µg/ml DEAE-Dextran (Amersham Biosciences).

Inhibition assays with env-pseudotyped reporter gene viruses.
Inhibition activities against pseudotyped virus on TZM-bl cells were evaluated as described[5]: Briefly, viruses were pre-incubated with the mimetic 1 inhibitor for 1h at 37 ^{o}C. TZM-bl cells were seeed at $5*10^4$/ml (1*104/well)and CD8 depleted PBMC $1*10^6$/ml. The virus input was adjusted to a TCID50 of 200/well for TZM-bl cells and to 1000/well for PBMCs. Infection of TZM-bl cells was performed in DMEM medium, containing 10% FCS, antibiotics and 10µg/ml DEAE-Dextran (Amersham). PBMCs were infected in RPMI1640 medium containing 10% FCS, antibiotics and 2.5 µg/ml polybrene (Sigma). Then, 150 µl supernatant were removed 72 h post infection and 50 µl lysis buffer (Promega) added to the cells. After 5 minutes luciferase activity was measured in 50 µl of the cell lysate, upon transfer to a white opaque plate (Costar) and addition of 50 µl firefly luciferase substrate (Promega). The RLUs were measured on a Dynex MLX luminometer. To control for unspecific inhibitory effects of the inhibitors control experiments using pseudotyped viruses caring the MuLV envelope (murine leucemia virus) were performed.

Generation of env-pseudotyped HIV particles
293-T cells were transfected with plasmids carrying the reporter gene expressing the virus backbone, pNLluc-AM[8] (a kind gift from A. Marozsan and J. P. Moore), and the functional envelope clone at a ratio of 3:1 polyethylenimine (PEI, linear 25kDa, *Polysciences*)[9] as described elsewhere[10].

Analyses or dose response curves

Sigmoid dose respond curves with variable slope were fitted by analyzing the percentage neutralization (y-axes) and the corresponding logarithmic value of the inhibitor concentration (x-axes) by GraphPad Prism 5.01 setting constrains for upper values to 100% and lower values to 0%. The inhibitior concentration resulting in 50% inhibiton (IC50) and its 95% confidence intervals based on the fitted curves were calculates by the same application.

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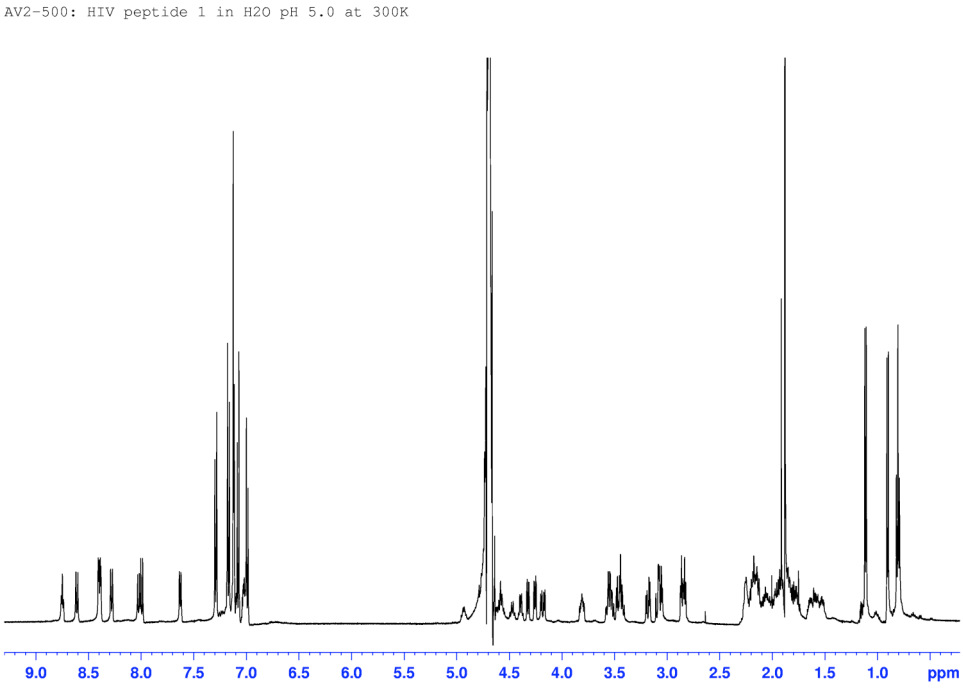
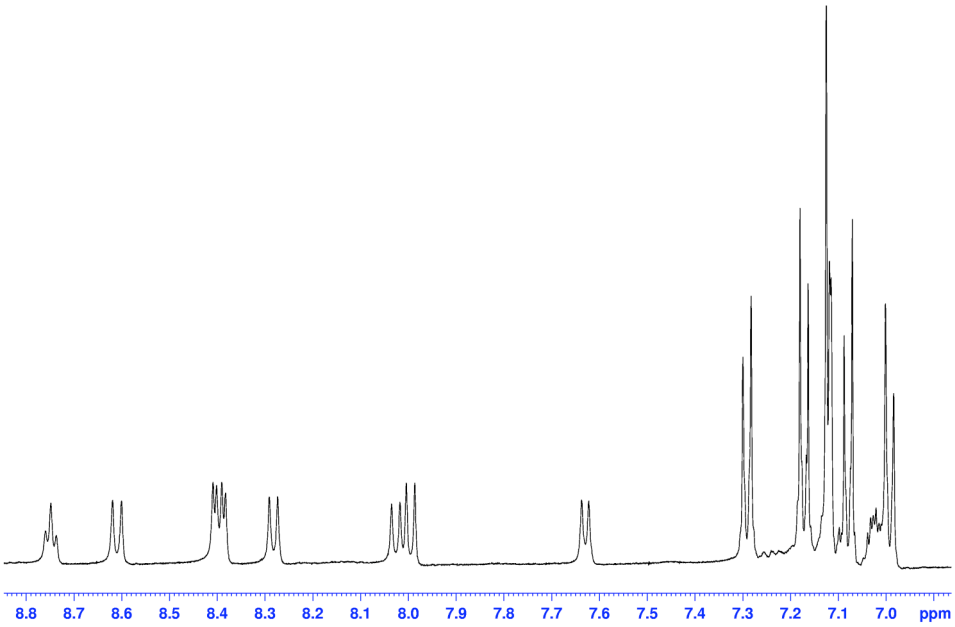
Spectra for peptides 1 and 2.

Spectra are shown below in the following order:

- ^{1}H-NMR (500 MHz) spectra of peptide 1 (full range)
- ^{1}H-NMR (500 MHz) spectra of peptide 1 (amide region)
- Neg. mode ESI MS spectra of peptide 1
- ^{1}H-NMR (500 MHz) spectra of peptide 2 (full range)
- ^{1}H-NMR (500 MHz) spectra of peptide 2 (amide region)
- Neg. mode ESI MS spectra of peptide 2

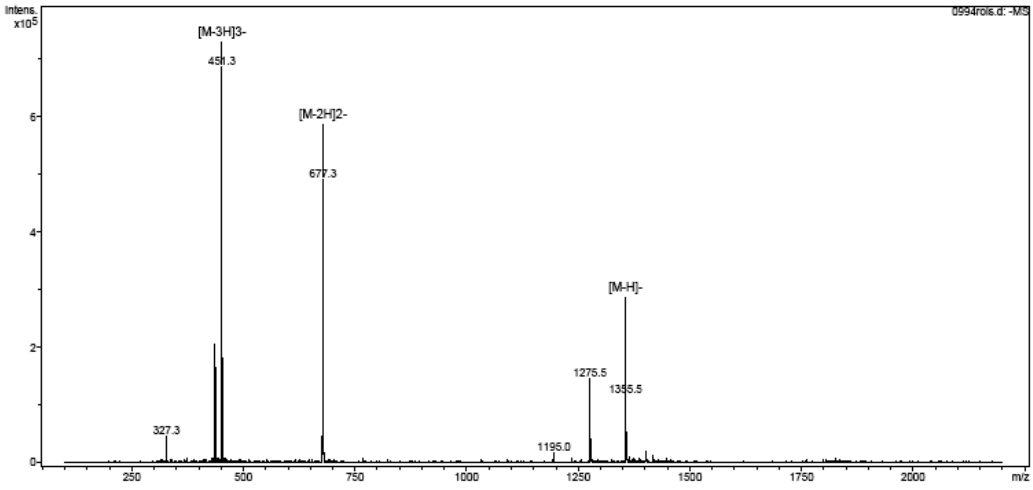


AV2-500: HIV peptide 1 in H2O pH 5.0 at 300K



Display Report

Analysis Name	0994rois.d	Acquisition Date	08/04/08 15:51:56	Operator	Administrator
Sample Name	cont. inj.	Method	STD_NR.MS	Instrument	Esquire-LC_00028
Comment	sample : HIV1 solvent : H2O/MeCN 1:1 + 10mM NH4OAc client Seits				



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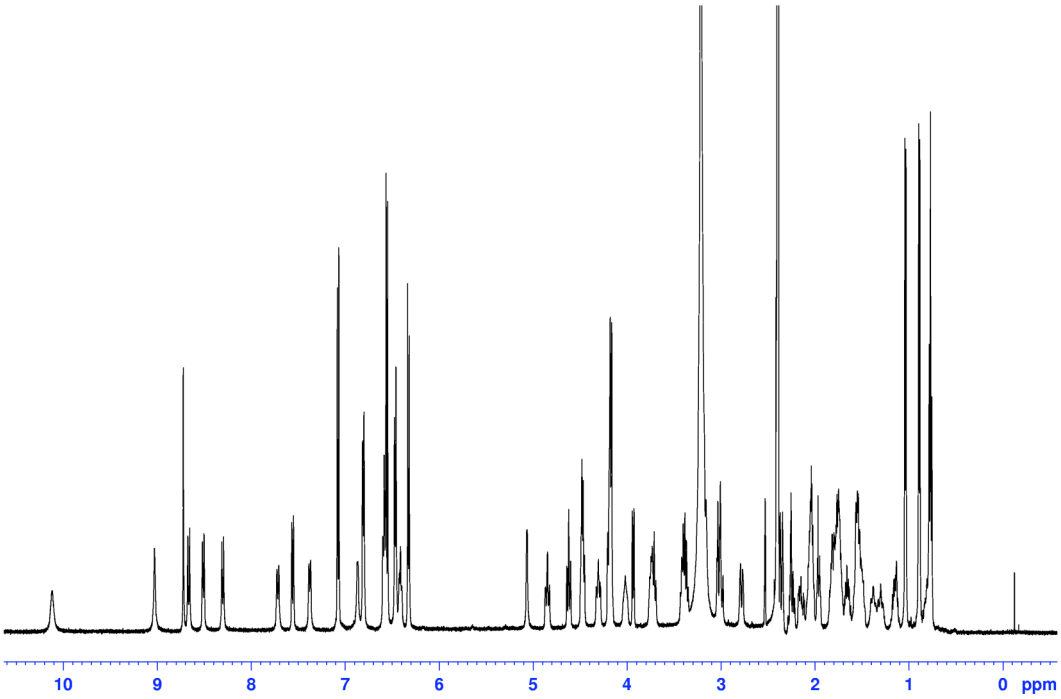
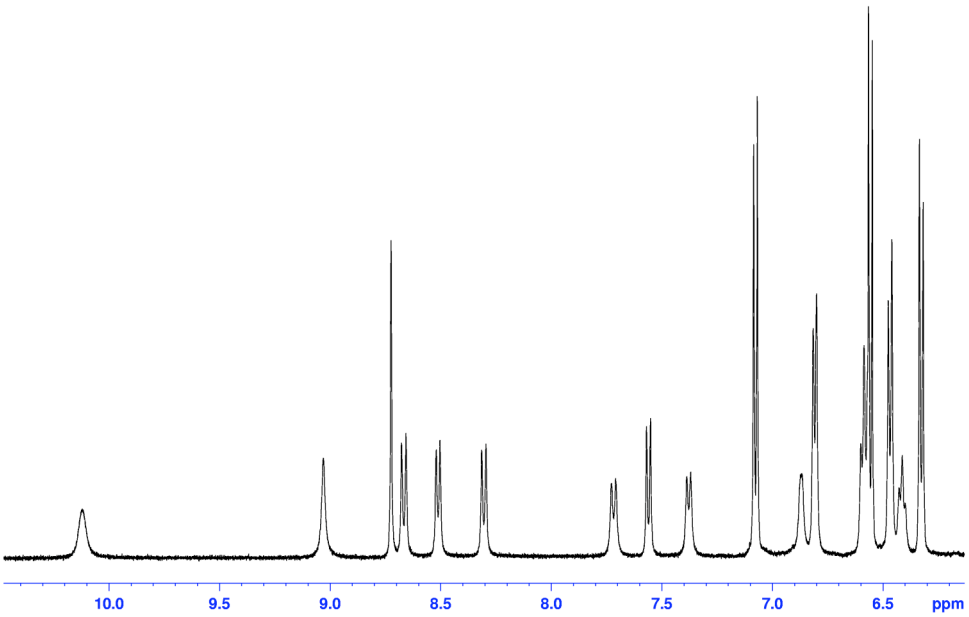
AV2-500: 1H of HIV peptide 2 in DMSO at 300K

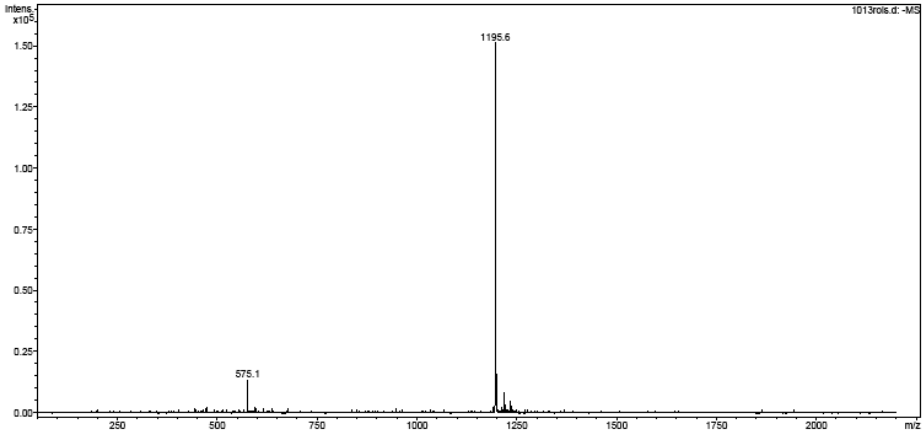


AV2-500: 1H of HIV peptide 2 in DMSO at 300K



Display Report

Analysis Name	1013rois.d	Acquisition Date	08/12/08 16:58:04	Operator	Administrator
Sample Name	Default	Method	STD_NR.MS	Instrument	Esquire-LC_00028
Comment	cont. inj. sample: HIV2a solvent: H2O/MeOH 1:1 + 10 mM NH4OAc client: Seitz				



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