Supplementary document

Dual concentration-dependent effect of Ascorbic acid on PAP(248-286) amyloid formation and SEVI-mediated HIV infection

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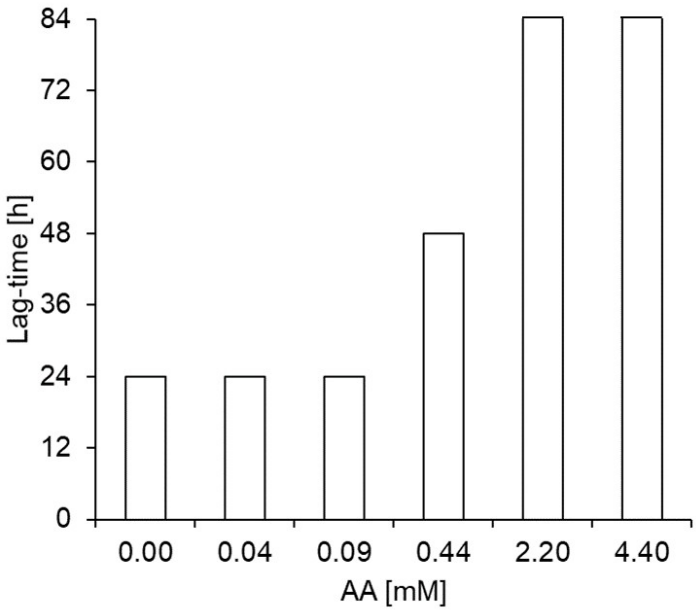
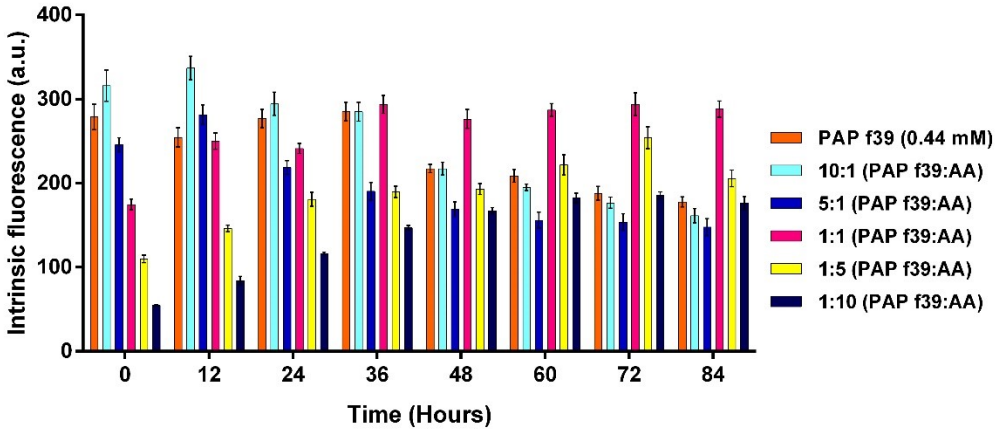
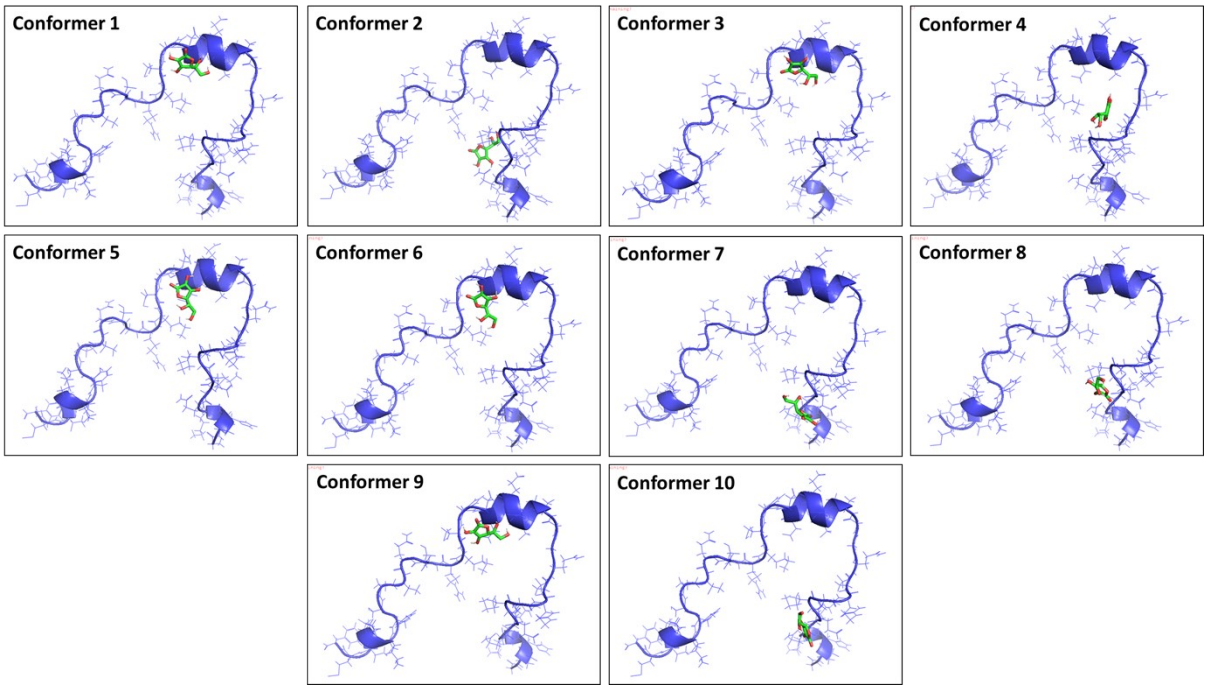


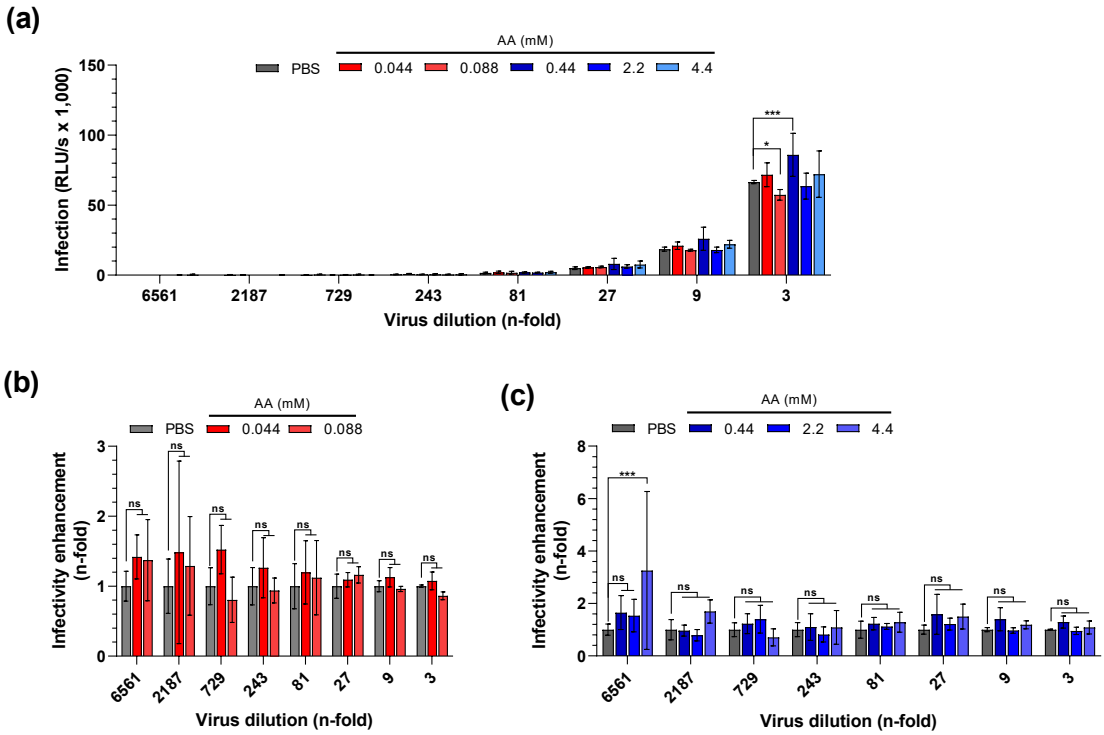
Supplementary Figure S1. Plot showing changes in lag-time of PAP(248-286) aggregation with varying concentrations of ascorbic acid.



Supplementary Figure S2. Intrinsic Tyrosine fluorescence-based aggregation assay showing the effect of various concentrations of ascorbic acid (AA) on PAP(248-286) assembly.



Supplementary Figure S3. Docked conformers of monomeric PAP(248-286) peptide with AA.



Supplementary Figure S4. Effect of ascorbic acid on serially diluted HIV-1 a) Serial dilutions of HIV-1 NL4-3 92TH014.12 were treated with AA of 0; 0.044; 0.088; 0.44; 2.2 and 4.4 mM AA (resulting in matching AA concentrations used in Figure 5) and used to infect TZM-bl cells. Infection rates were assessed by β-galactosidase assay at 3 days post-infection. b) and c) fold change in HIV-1 NL4-3 92TH014.12 infectivity treated matching AA concentrations samples relative to PBS control (calculated from a). Values shown represent mean values derived from triplicate infections in relative light units/s (RLU/s). Asterisks indicate difference of values compared to PBS control, non-significant ns: $P>0.05$, *: $P\leq0.05$, **: $P\leq0.01$, ***: $P\leq0.001$ (2-way ANOVA with Dunnett's multiple comparisons test), for clarity non-significant differences were omitted in (a).