



Figure 4 a) Broad perturbation of C-terminal anchor preference is observed in the presence abacavir but not analogues 15, D, H or M; however, potential neo-epitopes show a range of overlap with abacavir induced ligands. a) Length (i) and primary anchor characteristics (ii. Position 2, iii. C-terminal) of HLA-B*57:01 ligands isolated from CIR.B*57:01 grown in the absence of drug treatment, or in the presence of 35μM abacavir (black) or analogues 15 (red), D (green), H (orange) or M (blue). Analyses are based on non-redundant peptide identifications (by sequence, modifications not considered) per data set made at a confidence greater than that for a 5% false discovery rate (FDR) and filtered for ligands of endogenous HLA molecules of parental CIR cells. Anchor residue preferences are shown for 9mers and are depicted as the proportion of peptides that possess specific amino acids at position 2 (ii) and the C-terminus (iii). Data shown is the mean ± SD of triplicate experiments for untreated and abacavir treated cells, duplicate experiments for analogue 15, and single experiments for the remaining analogues (due to availability of compounds). b) iceLogos for P1 to P3 and PΩ-2 to PΩ of 9-12mer peptides in the constitutive repertoire of HLA-B*57:01. c) iceLogos for 9-12mer HLA-B*57:01 ligands detected in the presence of abacavir in this study either i) unfiltered or ii) filtered for constitutive ligands to identify potential neo-epitopes. d), e), f) and g) show i) icelogos for 9-12mer HLA-B*57:01 ligands detected in the presence of analogues 15, D, H and M, ii) Venn diagrams showing the numbers of potential neo-epitopes identified in both abacavir and analogue treatments and iii. icelogos for 9-12mer neo-epitopes identified. iceLogos were generated using icelogo software utilising the human swiss-prot proteome as the reference set. Letter height corresponds to % difference in frequency of the amino acid compared to presence in the human proteome.