Supporting Information

Cheent et al. 10.1073/pnas.1304366110

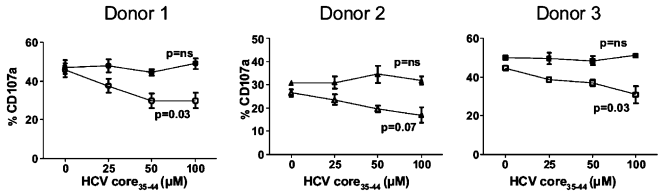

Fig. S1. Synergy of hepatitis C virus (HCV) $core_{35–44}$ peptides and HLA-A2 signal peptides ($HLA\text{-}A2_{sp}$) in inhibiting $NKG2A^+$ natural killer (NK) cells from three additional donors. Degranulation of $CD3^+$ $CD56^-$ $NKG2A^+$ $CD158b^-$ NK cells in response to .174 cells incubated with 0–100 μM HCV $core_{35–44}$ alone (closed symbols), or in the presence of 1 μM $HLA\text{-}A2_{sp}$ (open symbols). Data are shown as means ± SEM from three independent experiments. *P* values were calculated using one-way ANOVA.

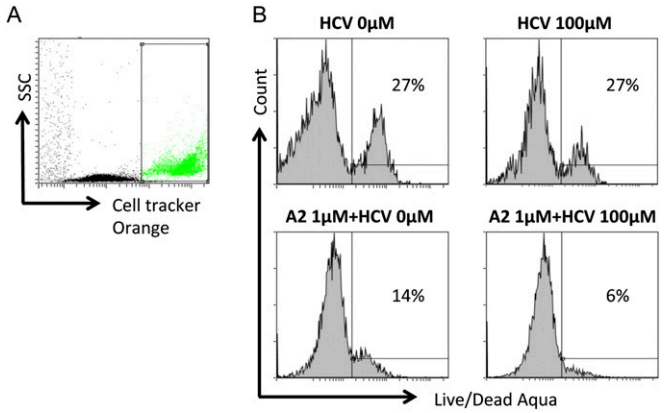

Fig. S2. Flow cytometry cytotoxicity assays using sorted $NKG2A^+$ NK cells. .174 cells incubated with 0 or 100 μM HCV $core_{35–44}$ alone or in the presence of 1 μM $HLA\text{-}A2_{sp}$ were labeled with Cell Tracker Orange and then were used as targets in cytotoxicity assays with $NKG2A^+$ NK cells. The gating strategy for the 0.174 target cells is shown in *A*, and the percentage of dead cells is indicated in *B*. SSC, side scatter.

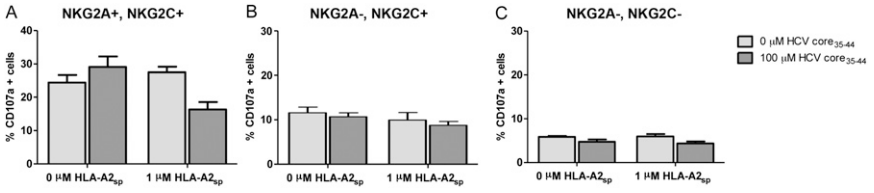

Fig. S3. Synergy of HCV $core_{35–44}$ and $HLA\text{-}A2_{sp}$ peptides in inhibiting specific subpopulations of NK cells. Degranulation of $CD158b^-$ NKG2A+ NKG2C+ (*A*), $CD158b^-$ $NKG2A^-$ NKG2C+ (*B*), and $CD158b^-$ $NKG2A^-$ $NKG2C^-$ (*C*) NK cells in response to .174 cells incubated with 0 or 100 μM HCV $core_{35–44}$ alone or in the presence of 1 μM $HLA\text{-}A2_{sp}$. Data are shown as means ± SEM from three independent experiments.

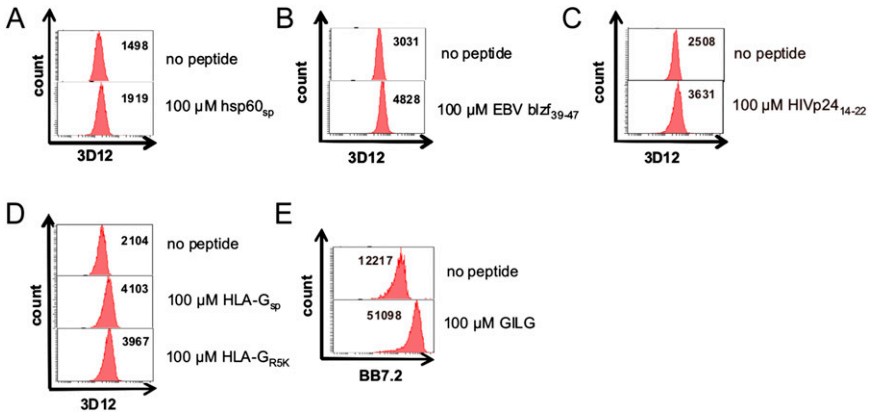

Fig. S4. Peptide stabilization assays. Stabilization of HLA-E on .174 cells in the absence of peptide or loaded with 100 μM $hsp60_{sp}$ (*A*), EBV $bzlf_{39–47}$ (*B*), HIV $p24_{14–22}$ (*C*), or HLA-G_{sp} and HLA-G_{R5K} (*D*), as determined by flow cytometry. (*E*) Stabilization of HLA-A2 by GILGFVFTL (GILG) as measured by the HLA-A2–specific antibody BB7.2. For all histograms the mean fluorescence intensity of staining is shown within the relevant histogram plot.