Supplemental Figure 1. MICA antibody masking prevents NKG2D-initiated CD3ζ downmodulation in NKG2D-licensed T cell lines. Proportions (in percent) of CD3ζ$^{low/-}$ cells among NKG2D-licensed T cells (line 4) exposed for 72 h to LTk–MICA-transfectants are reduced by treatment with anti-MICA/B (mAb 6D4) but not control Ig. Data shown are representative of duplicate experiments with three T cell lines. Error bars indicate SD.

Supplemental Figure 2. Caspase inhibition attenuates NKG2D-induced CD3ζ loss. Averaged percentages of caspase-3/7-specific FLICA (FAM-DEVD-FMK)-binding CD3ζ$^{low/-}$ cells among total live CD8 T cells recorded by flow cytometry at the indicated time points in separate experiments with the NKG2D-licensed T cell lines 1-3 exposed to solid-phase anti-NKG2D (mAb 1D11) with or without addition of caspase inhibitor Q-VD-OPh or DMSO solvent control following the 48 h time point. Error bars indicate SD.

Supplemental Figure 3. NKG2D ligand-free *in vitro* culture reduces proportions of CD3ζ$^{low/-}$ T cells and NK cells among SLE PBMC. Flow cytometry dot plots comparing levels of CD3ζ and NKG2D expression on freshly *ex vivo* isolated SLE T cells and NK cells with those recorded following 24 h *in vitro* culture in the absence of NKG2D ligands (recovery). Dot plots were derived from a gating tree based on exclusion of LIVE/DEAD fixable violet/Annexin V staining and inclusion of surface CD3 (T cells) or CD16 (NK cells). Numbers in plots specify proportions of CD3ζ$^{low/-}$ cells among total live/non-apoptotic T cells and NK cells in percent. Data shown are representative of three juvenile-onset SLE PBMC samples.