**Supplemental FIGURE LEGENDS**

**Supplemental Figure 1.** Three melanoma cell lines (CHL1, Mel888, and WM793) were treated with DMSO (0), 10uM, or 50uM of dabrafenib (BRAFi) for 3 hours. Whole cell lysates were prepared, and levels of phospho-ERK and total ERK were analyzed by immunoblotting.

**Supplemental Figure 2.** Mel888 cells were treated with vehicle (DMSO) or 50 uM of BRAFi for 3 hours. Following treatment, cells were stained with fluorescently-labeled antibodies specific for MHC-I (HLA-A,B,C), MHC-II (HLA-DR), PD-L1, or melanoma-associated chondroitin sulfate proteoglycan (MCSP), and analyzed by flow cytometry. Data shown are representative of at least three replicate experiments with similar results. \* indicates p < 0.05; ns, not significant.

**Supplemental Figure 3**. HLA-A2-transduced Mel888 melanoma cell lines were treated with vehicle DMSO (0), 10uM, or 50uM of BRAFi for 3 hours. Whole cell lysates were prepared and levels of phospho-ERK and total ERK were analyzed by immunoblotting.

**Supplemental Figure 4.** HLA-A2-transduced Mel888 or WM793 cells were treated with DMSO or MEKi for 3 hours. Following treatment, cells were stained with a fluorescently-labeled HLA-A2-specific antibody and analyzed by flow cytometry. These experiments were repeated at least four times with similar results. \*\*\* indicates p < 0.005; ns, not significant.

**Supplemental Figure 5.** T2 cells were pulsed with titrated amounts of MART-1(27-35) peptide, washed, and then used as stimulator cells for MART1-specific CD8+ TILs. Supernatants were collected after 8 hours of co-culture and analyzed by ELISA to measure interferon-gamma (IFN) release.

**Supplemental Figure 6.**  Percentage of intracellular IFN-positive TILs following 3 hours of co-culture with DMSO- or BRAFi-treated HLA-A\*0201-transduced Mel888 cells or MART-1(27-35) peptide-pulsed WM793 cells, as measured by flow cytometry. All data are representative of experiments performed at least 3 times with similar results. \*, p<0.05; ns, not significant.

**Supplemental Figure 7.** Mel888 cells were treated with 50 uM BRAFi for the indicated times, and cell lysates prepared. Cellular expression of MART-1 protein at each time point was assessed by immunoblotting and quantified using densitometry, with -actin levels measured as a loading control.

**Supplemental Figure 8.** Mel888 cells were treated with DMSO, BRAFi, or MEKi for 2 hours and then labeled with 35S-Methionine for 20 min. Cells were placed back into culture at 37 C with continued exposure to drug or vehicle. Cells lysates were prepared at each of the indicated time points and total HLA-A2 was immunoprecipitated and analyzed using polyacrylamide gel electrophoresis. Immunoprecipitated HLA-A2 was then analyzed for 35S content by Phosphor-imaging.

**Supplemental Materials and Methods**

***Antibodies and Flow cytometric analyses***

Anti-HLA-A2-APC (BB7.2), anti-HLA-A,B,C-APC (W6/32), anti-human IFN-PE (B27), anti-human CD8-Pacific Blue (SK1), and Alexa Flour 647 (IgG2b) isotype control antibodies (Biolegend) were used in these studies, in addition to streptavidin-APC (BD Biosciences) and biotin-conjugated anti-HLA-A1,A36 (US Biological). Melanoma cell lines were cultured with BRAFi, MEKi or DMSO (vehicle control) for 3 hours. Following treatment, cells were washed and stained for surface expression of HLA-A2, HLA-A,B,C, or HLA-A1 and analyzed by flow cytometry. As a functional CD8+ T-cell readout, intracellular IFN was stained with anti-human IFNγ conjugated to APC using the fixation and permeabilization protocol from the BD Cytofix/Cytoperm™ Fixation/Permeabilization Solution Kit with BD GolgiPlug™ (BD Biosciences).

***Confocal Microscopy***

Hoechst 33342 and all Alexa Fluor-conjugated secondary antibodies were acquired from Molecular Probes. Melanoma cells were seeded and cultured in Ibidi 12-well chambers on microscope glass slides one day prior to the experiments. For internalization assays, Mel888 and WM793 cells were rinsed with 2% BSA in ice-cold PBS with calcium and magnesium before being chilled on ice to stop internalization. Live cells were labeled with Alexa Fluor 488-conjugated mouse anti-human HLA-A,B,C (W6/32) for 30 minutes at 4 C. Labeled slides then were washed twice and returned to 37 C for incubation times of up to 2 hours. At each time point, slides were fixed with 4% paraformaldehyde (PFA) for 5 min, then remaining surface MHC-I was stained with Alexa Fluor 568-conjugated goat anti-mouse antibody for one hour. Cell nuclei were also labeled with Hoechst 33342 for 15 min at room temperature. Slides were washed and then mounted with fluorescence mounting medium (Dako) and imaged using a Leica SP2 confocal microscope.

To analyze the effect of BRAF inhibition on MHC-I trafficking, melanoma cells were first treated with 50nM of BRAFi or DMSO for 3 hours. Cells were then labeled with Alexa Fluor 488-conjugated W6/32 on ice 30 minutes. After washing, cells were then returned to culture at 37˚C in the presence of 50nM of BRAFi or DMSO. After 30 to 90 minutes, cells were washed, fixed and labeled as described above.

To study the distribution of MHC-I at steady state, melanoma cells were treated with 50nM BRAF inhibitor or DMSO control for 3 hours. Cells were then fixed and permeabilized with Cytofix/Cytoperm kit (BD Biosciences) according to manufacturer’s protocol. To visualize total cellular MHC-I, permeabilized cells were incubated with W6/32 antibody, washed, and then stained with Alexa Fluor 488-conjugated goat anti-mouse antibody. To visualize LAMP-1, rabbit anti-LAMP-1 mAb D2D11 (Cell Signaling) and Alexa Fluor 568-conjagated goat anti-rabbit antibody were used.

***Enzyme-linked immunosorbent assay***

T2 cells were pulsed with titrated amounts of MART-1(27-35) peptide for one hour, washed three times, and then co-cultured with MART-1-specific TILs (>98% tetramer-positive) for 8 hours. Cell supernatants were collected and analyzed for IFN secretion using the Ready Set GO! ELISA human IFNkit (eBiosciences). ELISA plates were read using a SpectraMax® M5/M5e Multimode Plate Reader and analysis program.

***Western Blotting***

Western blots were performed to determine the concentration of inhibitors required to block the MAPK pathway. CHL1 (BRAF WT), WM793 and Mel888 (BRAF V600E) melanoma cells were incubated for three hours in DMSO, BRAFi, or MEKi at different concentrations. Cell lysates were prepared and protein content was normalized using the BCA method (Thermo-Fisher). Membranes were probed with antibodies specific for Phospho(P)-ERK and total ERK2 (Santa Cruz Biotechnology).

To validate HLA-A2 lentiviral constructs and A2-transduced cell lines, BB7.2 mAb was used to immunoprecipitate HLA-A\*0201 from whole cell lysates prepared from transduced Mel888 and WM793 cells. Immunoprecipitated HLA-A2 was analyzed by Western blotting using rabbit anti-HLA-A as a secondary antibody at a 1:2000 dilution (clone EP1395Y, Abgent).

To assess changes in protein levels of MART-1 following MAPK inhibitor treatment, Western blotting was used to measure MART-1 expression in Mel888 cells. Cells were plated on 60mm dishes and treated with 50 uM BRAFi. At different time points, whole cell lysates were collected and analyzed by SDS-PAGE, and blotted onto nitrocellulose membranes. Anti-Melan-A/MART-1 (EMD Millipore) and secondary antibody anti-mouse IgG-HRP (Cat#:7076S; Cell signaling) were used to probe blots at concentrations of 1:300 and 1:25000, respectively. Western blot proteins were visualized using the Scientific Pierce Fast Western Blot Kit (Thermo-Fisher).

***35S-Methionine Pulse-Chase Experiments***

To determine the molecular half-life of MHC-I in the presence or absence of MAPK inhibitors, Mel888 melanoma cells were seeded into individual wells of a 6-well plate. All cells were pre-treated for 2 hours with BRAFi (50uM), MEKi (50 uM), or DMSO, labeled with 35S-Met for 20 minutes, and washed three times. Fresh media containing inhibitors or DMSO was added to each well and cells were placed in 37 C culture for various time points. Cell lysates were prepared and protein content was analyzed by TCA precipitation protocol. Total HLA-A,B,C was immunoprecipitated with W6/32 mAb and run on SDS-PAGE. 35S-labeled MHC-I (~43 kD) was then visualized by Phosphor-imaging analysis of dried polyacrylamide gels and quantified by densitometry.