**Supplemental Figure Legends**

***Supplemental Figure 1. Gating Strategies used for DC identification and in-vitro T cell assays.*** Gating strategy used to assess checkpoint and costimulatory molecule surface expression on HD and patient DC (A). Monocytes were gated based on FSC vs SSC gating (B), followed by single-cell/viability gating (C). HLA-DR+CD86+ double-positive DC were then analyzed for co-expression of the indicated DC markers, immune checkpoint molecules and costimulatory molecules by flow cytometry (D). Gating for positive frequencies of ICOSL are shown for a representative patient and HD. Quantified frequencies are also graphed for each DC subtype (E). Error bars represent standard deviation. Isotype controls were used to account for background staining. Molecules of interest were considered to be expressed on the surface of DC if positive staining was greater than 2%, compared to background (isotype) levels. CD8dim+ and CD4dim+ T cells were selected to assess functional T cell interactors in flow cytometry analyses. The following gating strategy was used to analyze DC-activated T cells from in vitro co-cultures: Lymphocyte cells were selected based on an FSC vs SSC gate, with CD3+ single-cells selected for consequent analysis (F). CD8+ and CD4+ T cells were then separated into high (resting, non-activated) and dim (recently activated) expressor populations (G-H). The CD4/CD8 dim populations were then assessed for expression of CD107a as a surrogate marker of their cytotoxic potential. DC viability was monitored by flow cytometry using zombie aqua dye (to validate data obtained by visual inspection based on trypan blue exclusion) (I).

***Supplemental Figure 2. Confirmation of AdVTMM2 transduction of patient DC ; melanoma patient mDC and DC express concentrations of immunoregulatory molecules.*** Differential expression analysis of patient DC (pre/post AdVTMM2 infection) was performed (n=33) using microarrays focusing on vector inclusive transcripts from Tyrosinase, MART-1/MLANA, MAGE-A6 and AdV Hexon-1 (A). A volcano plot is depicted with vector-associated melanoma antigen transcripts indicated. QPCR analysis was performed in a subset of patients (n=6) to verify the microarray data (B). The Y axis is expanded on the left to show differences in encoded tumor antigen expression. Each end of the box and whisker plot represents the first (25%) and third (75%) quartile, while the middle line represents the mean. Shown isa heatmap for the top 62 differentially expressed genes (FC > 2 and type 1 error rate of 0.05) in iDC, mDC and AdV/DC of melanoma patients (n=32) (C). Secreted IL-10 levels in melanoma patient mDC (n=22) compared to HD (n=4) (D). Error bars represent standard deviation. Patients were segregated based on clinical outcomes and unpaired student T tests were used to determine the significance of inter-group differences. \*: p value ≤ 0.05.

***Supplemental Figure 3. Consensus DC phenotypic and potency biomarkers do not correlate with clinical outcome.*** Consensus markers associated with immunostimulatory DC were assessed by flow cytometry after patient DC adenoviral transduction (AdV/DC) (A). DC were gated based on FSC vs SSC profiles, with IgG isotype-matched antibodies used to define negative control staining patterns. Spontaneous (B) and CD40L-induced (C) IL12p70 and IL-10 secretion from DC were assessed in culture supernatants by Luminex. Patients (n=35) were segregated by clinical outcome, and data comparisons were performed using unpaired student t-tests or Wilcoxon rank sum tests (CD80, CD40, CD11C, and IL12p70/IL10 comparisons) to determine significance. ns = not significant. Microarray analysis of critical DC genes (D), spontaneous or induced *IL12A*, *IL12B,* and *IL10* transcriptional levels (E) in DC subtypes from melanoma patients(n = 33). Each end of the box and whisker plots represent the first (25%) and third (75%) quartile, while the middle line represents the mean. Patients are segregated based on clinical outcome, with the significance of inter-group differences for *IL10* and *ITAX* determined using a one-way ANOVA analysis with Tukey’s multiple comparison test. All other inter-group differences were assessed using the Kruskal-Wallis one-way ANOVA with Dunnett’s multiple comparison test. \*\*: p value ≤ 0.01, \*\*\*\*: p value≤ 0.0001, ns= not significant.

***Supplemental Figure 4. HD and patient DC express surface expression of PD-L1, PD-L2 and CTLA-4 post-maturation and viral transduction.*** DC surface expression of immune checkpoint molecules PD-L1 (A, left), PD-L2 (A, right) an CTLA-4 (A, second row) on DC subtypes (Day 5 iDC, Day 6 mDC, and Day 7 AdV/DC) by flow cytometry in HD (n=4) and melanoma patients (n = 32). Error bars represent standard deviation. Patients were segregated based on clinical outcomes. Significance of inter-group differences for PD-L1 & CTLA-4 expression on patient cells and CTLA-4 expression on HD were determined using the Kruskal-Wallis one-way ANOVA with Dunnett’s multiple comparison test. PD-L2 expression ion patient & HD DC and PD-L1 expression. Patient DC were determined using one-way ANOVA analysis with Tukey’s multiple comparison test. \*: p value ≤ 0.05, \*\*: p value ≤ 0.01, \*\*\*: p value ≤ 0.001, \*\*\*\*: p value≤ 0.0001. Correlation of AdV/DC surface protein expression (flow cytometry; MFI) of PD-L1 and CTLA-4 (n=32) (B). Correlations of *CTLA4* with *IL12A*, *CD86,* and *IL10*  transcriptional levels in patient AdV/DC (n=33) (C). The grey shaded areas represent the 95% Confidence Interval bands. Patients are segregated based on clinical outcomes. Spearman correlations were used to assess the relationship between PDL1 and CTLA4 surface protein on AdV/DC, as well as to assess the relationship between *CTLA4* and *CD80*. Pearson’s R was used to determine the correlation between *CTLA4* and *IL12A* and the correlation between *CTLA4* and *IL10.*

***Supplemental Figure 5. ICOSL surface protein is significantly reduced in melanoma patient DC post-maturation*.** ICOSL protein surface expression (flow cytometry, MIF) was analyzed in HD (n=4) and melanoma patient (n=30) DC subtypes (A). Error bars represent standard deviation. Patients were segregated based on clinical outcomes. HD significance was determined using one-way ANOVA analysis with Tukey’s multiple comparison test. Patient significance was determined using Kruskal-Wallis one-way ANOVA with Dunnett’s multiple comparison test. sICOSL levels were determined in patient (n = 7) or HD (n=4) matched pairs mDC & AdV/DC culture supernatants using a specific ELISA. Due to a washout step following viral transduction, the levels of sICOSL detected in AdVTMM2/DC do not represent “net total”. We calculated cumulative totals (mDC + AdV/DC sICOSL levels) to estimate the total of sICOSL produced from day 7 AdV/DC for each patient/HD (B). Multivariable Cox proportional hazard model for CD8+ T cell infiltration and ICOSLG expression in SKCM tumors (C). KM curves for the corresponding immune infiltrates in SKCM (both primary tumor and metastasis). The CD8+ T cell infiltration and *ICOSLG* gene expression are divided into low and high levels. The hazard ratio and p value for Cox model and the log-rank p value indicated on the KM plot (D). \*: p value ≤ 0.05, \*\*\*\*: p value≤ 0.0001.

***Supplemental Figure 6. Primer design and DNA input used in Chromatin Immunoprecipitation (ChIP) assays.*** The predictedNF-κB binding site in the ICOSLG promoter is highlighted in red. Purple regions denote primer sequences used to amplify N-κB enrichment in ChIP assays. Additional primer pairs (see Methods section) targeting downstream EXON sequence was used for negative/background binding control (A). Shown is the distribution of micrococcal nuclease-digested DNA fragments from one representative donor used in the ChIP assay (B).