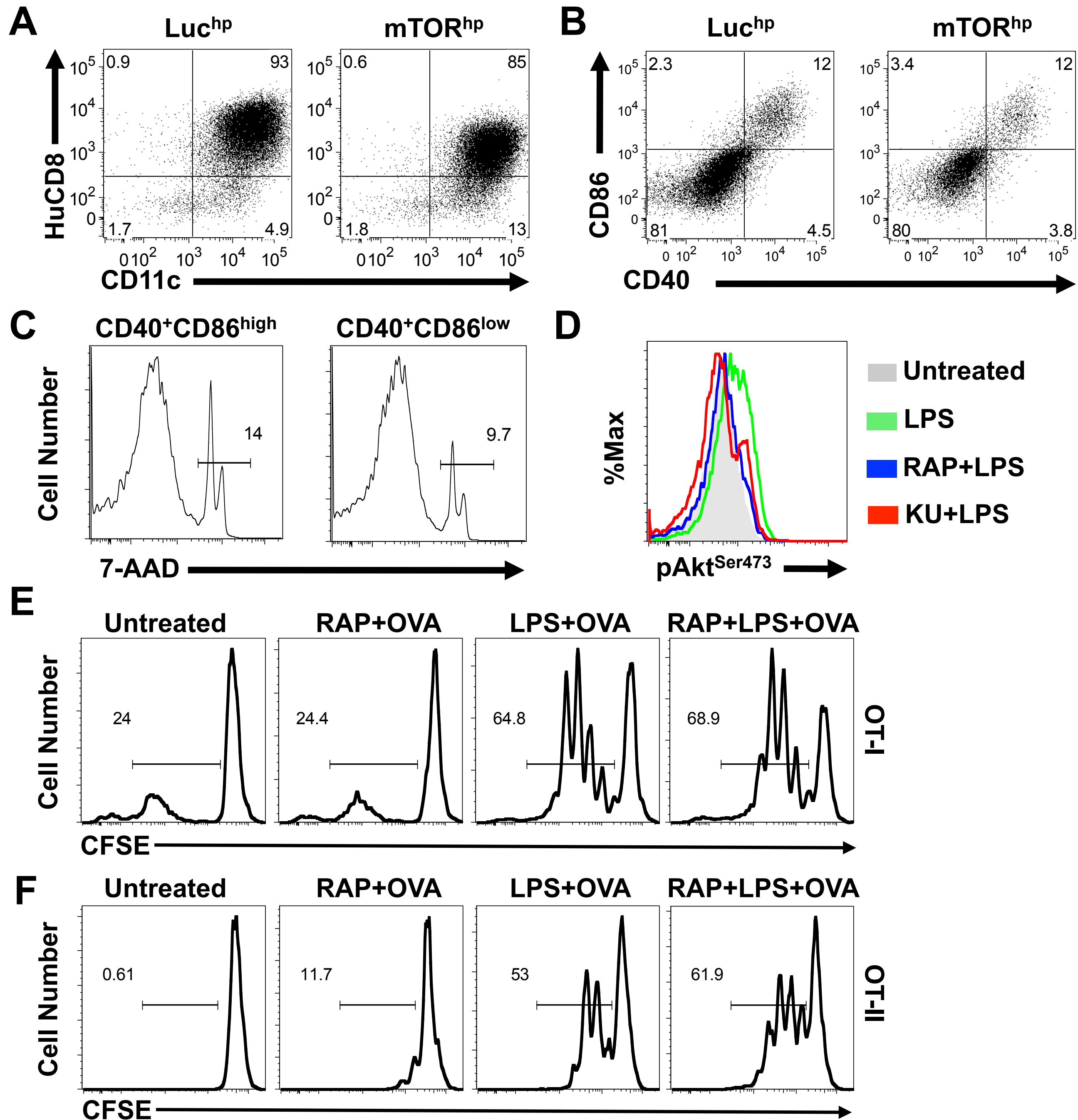
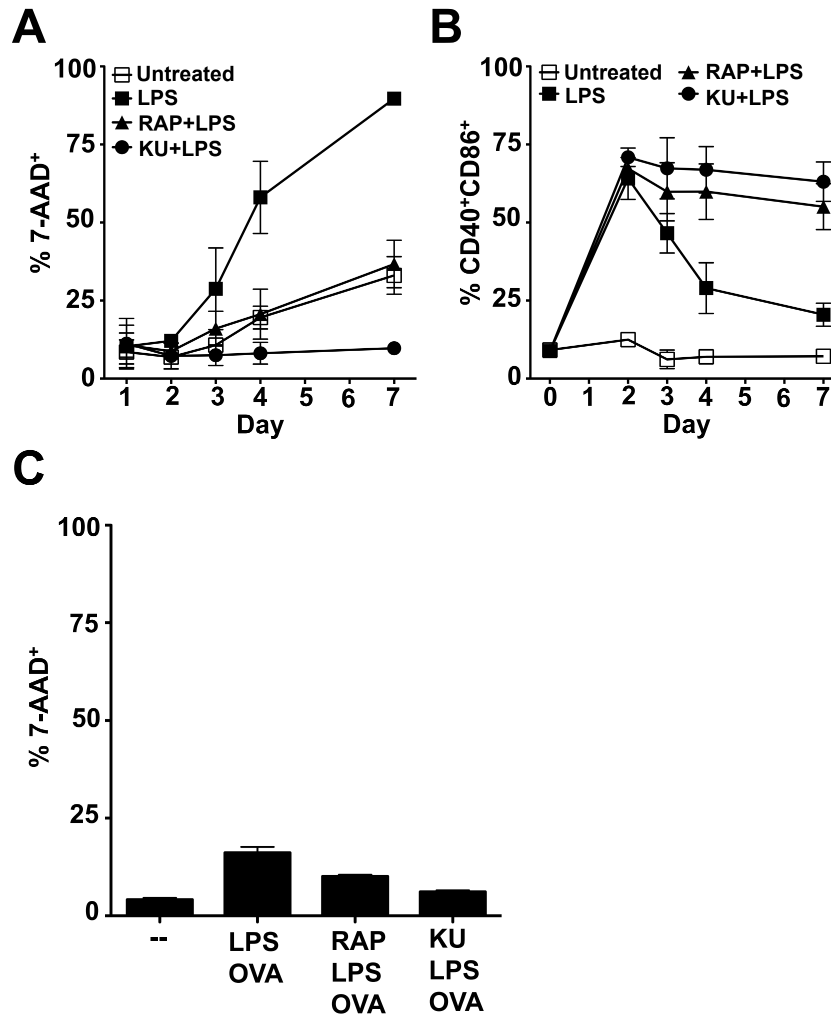
Supplemental Figure 1



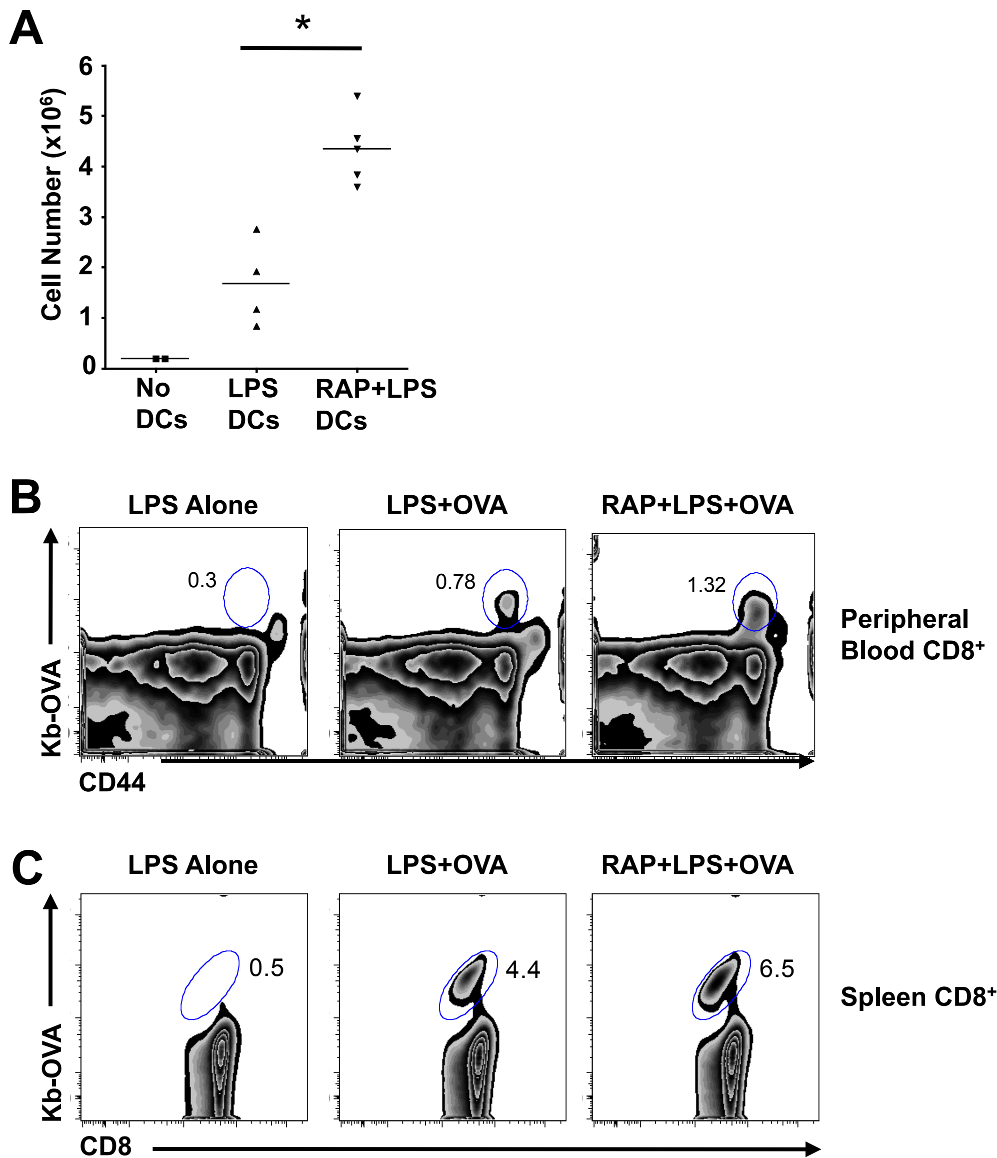
Supplemental Figure 1. (A) DCs were transduced with either Luc^{hp} or mTOR^{hp} –expressing retrovirus and purity of vector^{+} (human CD8) CD11c+ cells were assessed by flow cytometry. (B) DCs were transduced with either Luc^{hp} or mTOR^{hp} –expressing retrovirus. Unstimulated cells were analyzed by FACS on Day 6 of differentiation for CD40 and CD86 expression. (C) CD40^{+}CD86^{high} and CD40^{+}CD86^{low} cells from Luc^{hp} DCs from Figure 1E were gated on and histogram of 7-AAD staining is displayed. CD40/CD86 gates were determined from total CD11c^{+} cells. (D) DCs were treated as indicated and stained for phospho-Akt (Ser473) 24 hours later. Histograms are gated on CD11c^{+} cells. (E and F) DCs were pulsed for 6 hours with media alone, or ovalbumin (OVA) with RAP, LPS, or RAP + LPS and then washed and incubated at a 1:5 ratio with either CFSE-labeled OT-I splenocytes (E) or CSFE-labeled MHCII-depleted OT-II splenocytes (F) for 4 days. T-cell proliferation was determined by CFSE dilution within CD8^{+} or CD4^{+} cells by FACS analysis.

Supplemental Figure 2



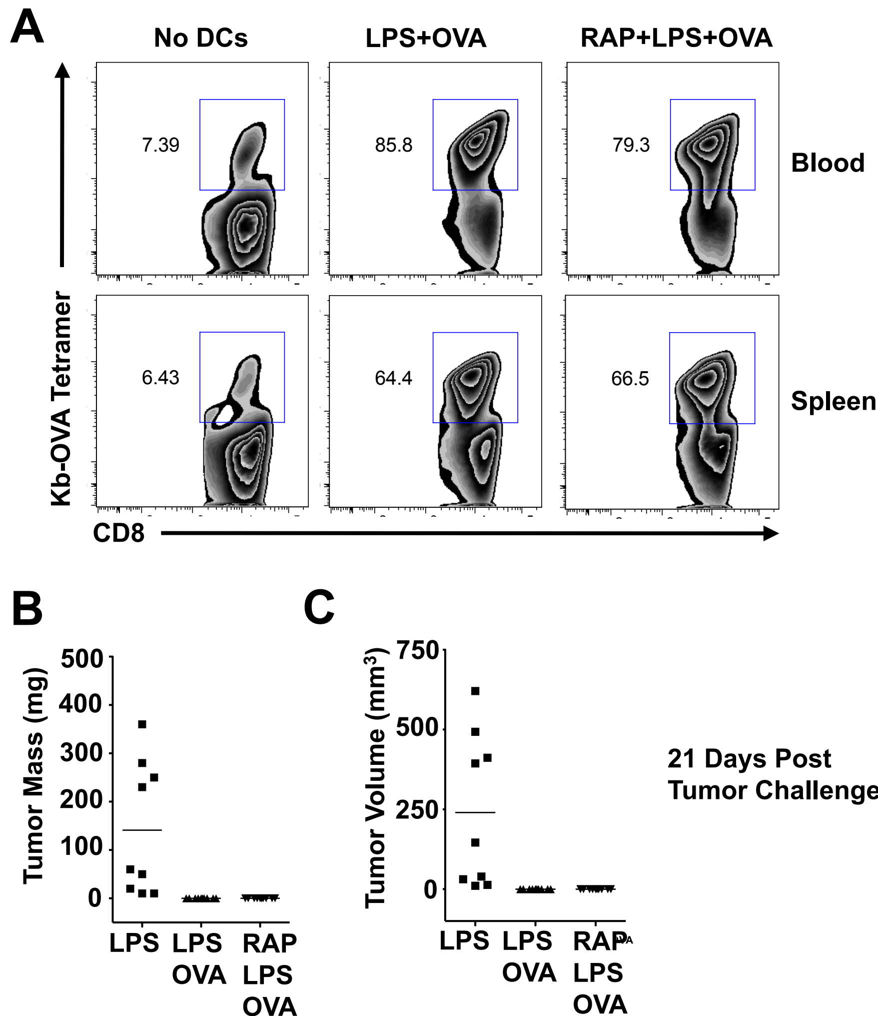
Supplemental Figure 2. The effects of mTOR inhibition on DC lifespan and costimulatory molecule expression in the presence of blocking anti-IL-10R. DCs were cultured in the presence of anti-IL-10R under the conditions indicated . (A) Changes in cell viability over time post activation were measured by FACS analysis of 7-AAD staining. (B) DCs were additionally analyzed daily by FACS for CD40 and CD86expression. For all graphs, data are presented as mean +/- SD of 2 independent experiments. (C) DCs were pulsed for 24 hours with media alone, or ovalbumin (OVA) with LPS, RAP + LPS, or KU+LPS and then washed and cultured in complete media for 4 days with daily media changes. DC viability for each treatment group was assessed at day 4 after stimulation by 7-AAD staining using FACS analysis.

Supplemental Figure 3



Supplemental Figure 3. DCs activated in the presence of rapamycin induce larger immune responses *in vivo* following autologous DC transfer. (A) $5x10^5$ DCs pulsed with either LPS+OVA or RAP+LPS+OVA for 6 hours were transferred into the footpads of mice. 7 days later, popliteal LNs were harvested and the total number of LN cells was quantified as compared to naïve popliteal LNs. Asterisk (*) indicates statistically significant difference ($p < 0.05$) in total cell numbers in the draining LNs between LPS DCs and RAP+LPS DCs groups. (B and C) Mice were treated as in (A) and the frequency of $CD8^+$Kb-OVA $Tetramer^+$ cells were quantified in the peripheral blood (B) and the spleen (C). FACS plots represented concatenated data from 5-10 individual mice.

Supplemental Figure 4



Supplemental Figure 4. Rapamycin-treated DCs are competent at generating $CD8^+$ T-cell memory responses *in vivo*. (A) Cohorts of mice received either no DCs, or $5x10^5$ DCs pulsed with LPS+OVA or RAP+LPS+OVA for 6 hours in the left footpads. 35 days later, mice were challenged with $1x10^6$ CFU Ova-expressing *L. monocytogenes* intravenously. Five days after bacterial challenge, mice were sacrificed and the frequency of Kb-OVA $tetramer^+$ T-cells ($CD8^+$ gate) were determined. Data represent concatenated data from 4 individual mice per group. (B and C) $5x10^5$ DCs were pulsed with LPS alone, LPS+OVA, or RAP +LPS+OVA for 6 hours and then injected subcutaneously into cohorts of mice (n = 9). 28 days later, mice were challenged with $1x10^5$ Ova-expressing B16 melanoma cells intradermally and monitored for tumor growth. 21 days following tumor challenge mice were sacrificed and tumor mass (B) and volume (C) were recorded.