Figure S1. PLX4720 enhances MART-1-specific T-cell recognition of BRAF mutant melanomas (**A**, **B** and **C**) BRAF mutated and BRAF wild-type (WT) melanoma cells were pre-treated with PLX4720 for 48hrs, then co-incubated with DMF5 T cells in 96-well plate for 24 hrs, with or without adding back PLX4720. IFN-γ release was measured by ELISA. (**D**) [3H]Thymidine incorporation was quantified in a liquid scintillation counter, as described in Methods. Data shown are representative of 3-5 independent experiments with similar results.

Figure S2. Chromium release assay demonstrating the cytolytic activity of pmel-1 T-cells against A375/H-2D^b/gp100 tumor cells pre-treated with the indicated concentrations of PLX4720. Data are expressed as mean ± SD and are representative of 2 experiments with similar results.

Figure S3. Administration of PLX4720 in vivo increases tumor infiltration of adoptively transferred T cells. B6 nude mice (5-7 mice/group) bearing A375/H-2D^b/gp100 tumors were treated with OFL-expressing pmel-1 T cells, along with gp100 peptide-pulsed DCs, by i.v. injection on day 7 after tumor inoculation. 6 doses of IL-2 were administered intraperitoneally on days 7, 8 and 9. A diet containing PLX4720 or vehicle alone was fed from day 9 to 11. 5 days after T cell adoptive transfer, tumors were harvested for IHC or flow cytometric analysis. (**A**) CD3+ T cell infiltration in two representative tumor biopsies from vehicle or PLX4720-treated mice, as determined by IHC analysis. Original magnification, ×100. (**B**) Summary of quantitative analysis of pmel-1 T cells infiltration in tumors. CD3+ T cell count was performed on slides in 10 adjacent HPF (400 ×) based on lymphocyte morphology. (**C**) Pmel-1 T cell infiltration was determined by Thy1.1 and CD45 staining, Data shown are expressed as mean ±

SEM. (n=7, * $P < 0.05$) (**D**) Quantitation of T cell tumor infiltration 5 days following pmel-1 T cell transfer. Data are expressed as mean ± SEM and are representative of 2 experiments with similar results.

Figure S4. Pmel-1 T cells in the peripheral blood of tumor-bearing mice, treated as described in Fig. 2A. Data are representative of 3 experiments with similar results.

Figure S5. B6 nude mice (5 mice/group) were subcutaneously implanted with A375/H-2D^b tumor cells on day 0 and treated as described in Fig. S3. (**A**) *In vivo* trafficking of OFL-expressing pmel-1 T cells into A375/H-2D^b tumors on day 5 after T cell transfer. (**B**) Summary of quantitative imaging analysis of transferred T cells into tumor sites.

Figure S6. Administration of PLX4720 in vivo increases tumor infiltration of adoptively transferred T cells and enhances antitumor responses. B6 nude mice (5-8 mice/group) bearing WM35/H-2D^b/gp100 tumors were treated with OFL-expressing pmel-1 T cells, along with gp100 peptide-pulsed DCs, by i.v. injection on day 24 after tumor inoculation. 2 days after T cell transfer, mice were fed with a diet containing PLX4720 or vehicle for 3 days. (**A**) Luciferase imaging showing *in vivo* trafficking of OFL-expressing pmel-1 T cells on day 5 after T cell transfer. (**B**) Summary of quantitative imaging analysis of transferred T cells at the tumor site. Quantification is expressed as the average of photon flux within ROI. (**C**) Tumor growth was monitored over time. Data shown are expressed as mean ± SEM.

Figure S7. PLX4720 treatment does not increase infiltration of adoptively transferred T cells into tumors containing WT BRAF. C57BL/6J-Tyr-2J/J albino mice (5 mice/group) bearing MC38/gp100 tumors were irradiated on day 6 after tumor inoculation and treated with OFL-expressing pmel-1 T cells, along with gp100 peptide-

pulsed DCs, by i.v. injection on day 7. 2 days after T cell transfer, PLX4720 or vehicle alone was administered by oral gavage for 3 days. (**A**) *In vivo* trafficking of OFL-expressing pmel-1 T cells into MC38/gp100 tumors on day 5 after T cell transfer. (**B**) Summary of quantitative imaging analysis of transferred T cells into tumor sites. (**C**) Tumor growth curves. Data are expressed as mean ± SEM and are representative of 2 experiments with similar results.

Figure S8. VEGF production by A375/H-2D^b/gp100 melanoma cells treated with PLX4720 or vehicle alone for 24 hrs in 96-well plates, as determined by ELISA. Data are expressed as mean ± SD and are representative of 3 experiments with similar results.

Figure S9. PLX4720 exposure reduces binding of c-myc to the VEGF promoter in BRAF mutant melanoma cells. (**A**) A375/H-2D^b/gp100 melanoma cells were treated with PLX4720 or vehicle, and protein extracts were subject to immunoblotting for p-ERK and total ERK, which served as loading control. (**B**) Potential consensus binding sites for transcription factor c-myc, identified by examination of the proximal promoter region of human VEGF with TFSEARCH. (**C**) A375/H-2D^b/gp100 cells were treated with PLX4720 or DMSO for 2 hours. ChIP assay was then performed, as described in Fig. 4D. Data are expressed as mean ± SD and are representative of 2 experiments with similar results.

Figure S10. Growth inhibition of human melanoma cell lines expressing WT BRAF (MEWO) or mutant BRAF (A375/H-2D^b/gp100 and A375/ H-2D^b/gp100/VEGF), treated with PLX4720 or vehicle control for 72 hrs. Cell viability was determined using the CellTiter-Blue Cell Viability assay. Data are expressed as mean ± SD and are representative of 3 experiments with similar results.

Figure S11. Tumors were harvested and mRNA levels of VEGF in patients with metastatic melanoma undergoing treatment with a selective inhibitor of BRAF(V600E) were assayed by qPCR. (**A**) Expression levels of VEGF were normalized to actin. (**B**) Fold decreases over pre-treatment value were plotted on a whiskers plot. The bottom and top of the 25th and 75th percentile respectively for all patients, with the bar indicating the median value. The whiskers indicate the extremes with open circles represent data points greater than 1.5 times the interquartile range.