Supplementary figure legends

Supplementary Figure S1. Silencing SRA/CD204 enhances an inflammatory response in DCs stimulated with radiation-treated tumor cells. DCs were infected with lentiviruses encoding scrambled shRNA or SRA shRNA, and co-cultured with RM1 tumor cell lysates prepared after ionizing radiation for 24 hours. Following incubation, transcriptional levels of genes \( il6 \) (A), \( ip10 \) (B) and \( ifn\beta \) (C) were analyzed by quantitative real-time PCR (qRT-PCR) using the ABI 7900HT Fast Real-time PCR System. The results were normalized to the expression level of \( \beta\)-actin gene. * \( p<0.01 \). Representative results from three experiments with similar results are shown. D. DC-scramble shRNA and DC-SRA shRNA were co-cultured with RM1 tumor lysates for 18 hours. IL-6 expression in CD11c\(^+\) cells was examined using intracellular cytokine staining assays, followed by FACS analysis.

Supplementary Figure S2. SRA/CD204-targeted radio-immunotherapy enhances cytolytic activity of effector T cells. A. Combination therapy does not cause pathological changes in the prostate. To assess the potential toxic effect of the combinatorial therapy on normal tissues, the prostates were harvested from tumor-bearing mice treated with RT plus DC-SRA-shRNA vaccination at different times indicated, and subjected to H&E staining. B. RM1-OVA tumor-bearing mice (n=5) were treated with RT alone, RT plus DC-scram or DC-SRA shRNA, or left untreated. One week after DC vaccination, splenocytes from treated mice were stimulated with OVA\(_{257-264}\) (1 \( \mu \)g/mL) \textit{in vitro} for 5 days in the presence of IL-2 (40 IU/ml). Viable splenocytes were isolated using LymphocyteTM, and then co-cultured with CFSE-labeled RM1-OVA cells at a ratio of 20:1 for 8 hours. Viability of tumor cell was examined by analyzing the percentage of 7-AAD\(^+\) CFSE\(^+\) cells. Representative histograms from three experiments with similar results are shown.

Supplementary Figure S3. The effect of combined RT and \textit{in situ} DC vaccination on tumor-infiltrating CD4\(^+\) and CD11c\(^+\) cells. RM1 tumor-bearing mice (n=5) were treated with RT alone, RT plus DC-scram or DC-SRA shRNA, or left untreated. One week after DC vaccination, cryosections of tumor tissues were stained with antibodies for CD4 (red) and CD11c (green), followed by staining with FITC-conjugated goat anti-rat secondary antibodies or Alexa 594-conjugated donkey anti-rat secondary antibodies. Quantitative analysis of infiltrating cells was
conducted by counting positive cells in at least five randomly selected fields of view (NS, not significant; * \( p < 0.05 \)). Data shown are representative of two experiments.

**Supplementary Figure S4. DC-SRA shRNA vaccination following RT upregulates Granzyme B expression in tumor-infiltrating CD8\(^+\) cells.** C57BL/6 mice (n=5) bearing RM1 tumor were locally irradiated, followed by i.t. administration with or without DC-scram or DC-SRA shRNA vaccine. One week after DC vaccination, tumors were collected, digested, and prepared into single cell suspensions. Granzyme B-expressing CD8\(^+\) cells were analyzed by intracellular staining and FACS.

**Supplementary Figure S5. SRA/CD204 silencing in DCs increases IFN-\(\gamma\) production by CD8\(^+\) cells, not CD4\(^+\) cells, after combinatorial therapy of RT and in situ DC vaccination.** Male C57BL/6 mice (n=5) bearing RM1 tumors were treated with different regimens as described. One week after DC vaccination, splenocytes were pulsed with RM1 cell lysates at a ratio of 1:3 for 48 hours. The frequency of IFN-\(\gamma\)-expressing CD8\(^+\) cells or CD4\(^+\) cells were assessed using intracellular cytokine staining assays and analyzed using flow cytometry. Data shown are representative of two experiments with similar results.