Supplement

Supplemental Figure 1

Splenic leukocytes after treatment with RGR-conjugated or unconjugated compounds. (A) Spleen cellularity (B) splenic $CD8^+$ T cells and (C) splenic $CD11c^+$ $MHCclassII^+$ dendritic cell counts were analyzed in control and treatment groups (n=3 per group). Mice were analyzed after 2 weeks of treatment (see Figure 2A). Controls are untreated 29-week-old RIP1-Tag5 mice and mice injected with rat IgG plus RGR peptide. Treatment with RGR-conjugated compounds results in significantly decreased splenic leukocytes as compared to unconjugated anti-CD40/IL-2. Error bars represent s.d.

Supplemental Figure 2

Pancreas histology of long term survivors after triple treatment therapy. (A) Untreated 14 week-old RIP1-Tag5 mouse, anti-$CD8^+$ staining of an islet of Langerhans embedded in exocrine pancreatic tissue. (B) Anti-$CD8^+$ staining of an islet of similar size in long term survivors (45 weeks) following IL-2-RGR/anti-CD40-RGR/transfer treatment with enhanced infiltration. Note, tumors are not detectable after therapy. Dotted lines delineate islets of Langerhans, 20x objective, bar 50 μm. (C) Representative staining for Tag in an untreated RIP1-Tag5 islet at 14 weeks of age. (D) “Patchy” Tag expression pattern after triple treatment in mice at the age of 45 weeks, 60x objective, bar 20 μm. New tumors arise from these islets after a lengthy period without treatment (age >90 weeks) due to continuous Tag expression in some β cells.

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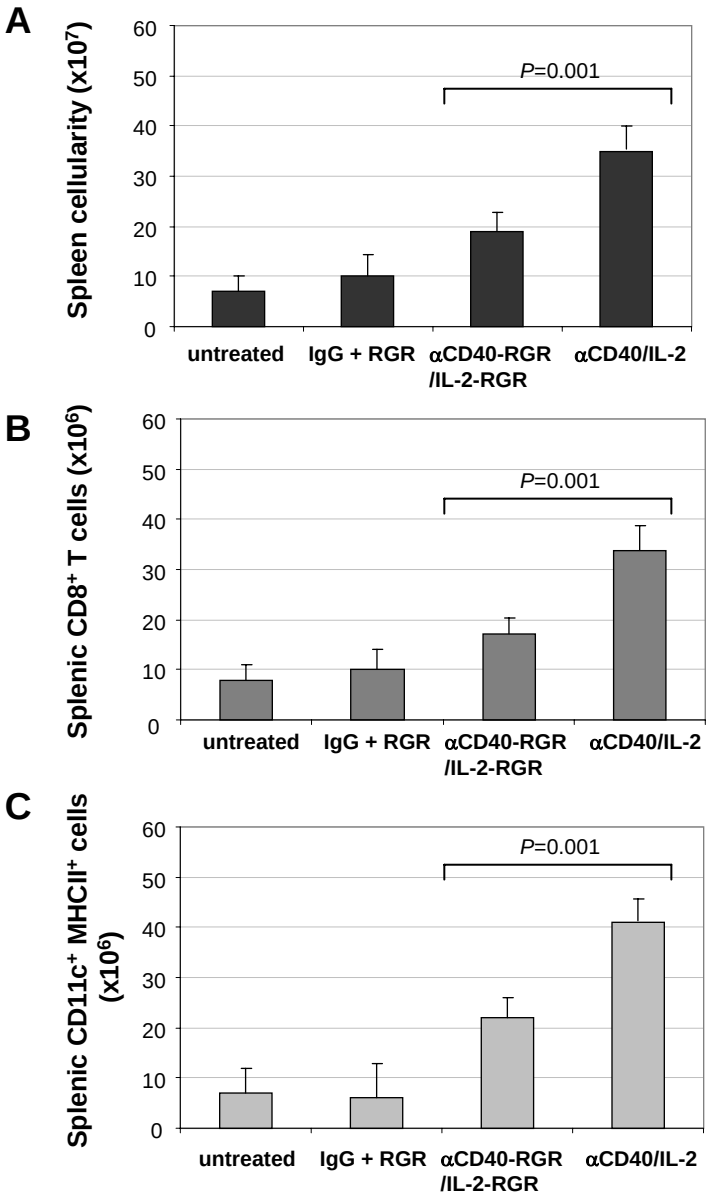
Hamzah et al.



Supplemental Figure 2
Hamzah et al.