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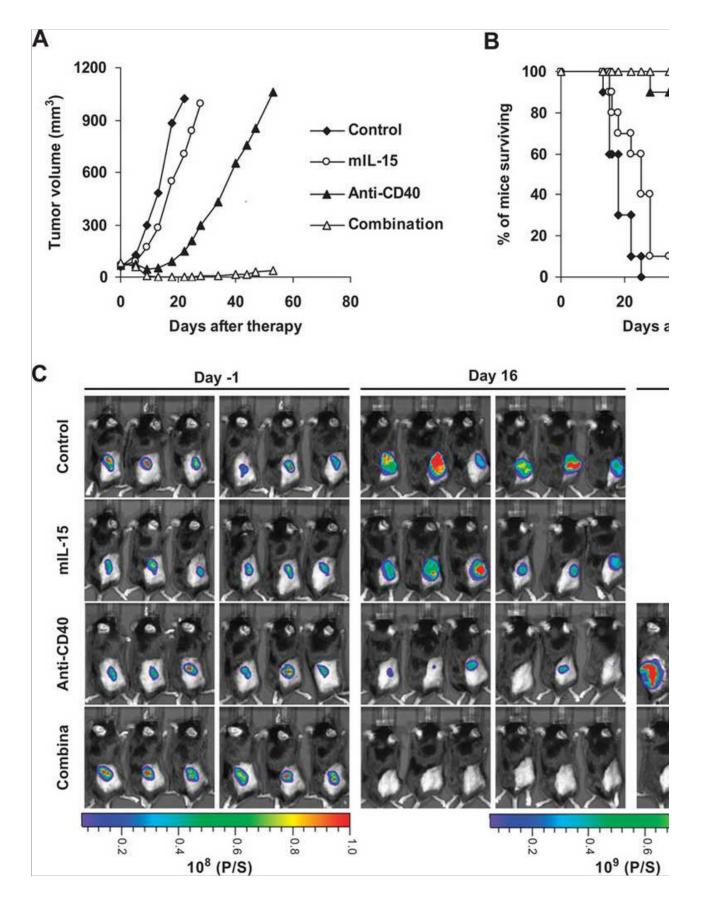
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Figure 1

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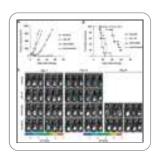
The combination therapy that involved mIL-15 and an agonistic a to the regression of established TRAMP-C2 tumors in wild type C

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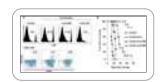
The therapy was started when the tumors were well established with an ave (A) Changes of tumor volumes during the therapeutic course and (B) Kapla mice in one of the two therapeutic studies (n=9-10). Treatment with mIL-1 2.5µg/mouse, 5 days a week for 2 weeks inhibited the tumor growth slightly the TRAMP-C2 tumor-bearing mice when compared with the mice in the P. (p<0.05) while treatment with the anti-CD40 antibody (▲) at a dose of 20 100 μg/mouse on days 3, 7 and 10 provided greater inhibition of the tumor survival of the TRAMP-C2 tumor-bearing mice when compared with the mi mIL-15 alone group (p<0.001). Furthermore, combination therapy (\triangle) with anti-CD40 antibody provided a greater therapeutic efficacy as demonstrated mice in the combination group were alive at day 60 with 80% becoming tur the mice in the anti-CD40 antibody alone group and none of the mice in th alone groups were alive at that time. (C). Bioluminescence imaging of the T bearing mice confirmed efficacy of combination treatment. Groups of 6 mice C2/luc-GFP tumors were treated using the same therapeutic protocol. The l were taken at different time points. Treatment with anti-CD4o antibody sig tumor growth. However, at day 40 after initiation of the therapy, only one c anti-CD40 group was tumor free. In contrast, the combination treatment le tumors with all of the 6 mice in the group becoming and remaining tumor f

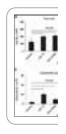
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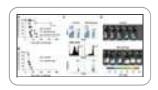
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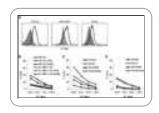












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