



Supp Figure 1: Targeting *Tgfβ* signaling in *BRAF^{V600E}*-induced PTCs. **A) Western blots of protein lysates from a cell line derived from a *Braf^{V600E}* PTC incubated in serum free medium for 24h and then treated with TGFβ1 for 1 h in the absence or presence of the TGFβ1 blocking antibody 1D11 or the TGFβR1 kinase inhibitor SD208 (1 μM). **B, C**) Effect of TGFβ1 blockade on SMAD activation and ¹²⁴I incorporation *in vivo*. *Braf* mice were treated with isotype control IgG or 1D11 antibody once every 2d for 14d. **B**) Western blots of PTC lysates collected at day 14 probed with the indicated antibodies. **C**) Thyroid ¹²⁴I uptake (%ID/g ±SEM) in *Braf* mice before and after treatment with 1D11 or isotype control. ¹²⁴I uptake was measured by micro-PET 24 h post ¹²⁴I administration before and after 14d of treatment. Each cohort contained 4 mice. **D,E**) Knockdown efficiency screen shRNAs to *TgfβR1* (**D**) and *TgfβR2* (**E**) in mouse *Braf*-PTC cells. *Top*: Quantitative RT-PCR performed in triplicate. *Bottom*: Western blots for pSMAD of TGFβ1 treated cells. **F**) Comparison of *TgfβR1* and *TgfβR2* shRNAs on TGFβ1-induced SMAD activation in *Braf*-PTC cells. **G**) Quantitative RT-PCR of thyroid differentiation markers in PTCs from *Braf*, *Braf/TβR1* and *Braf/shTβR1* mice. Four mouse thyroid tissues from each genotype were analyzed in triplicate. Unpaired t test with Welch's correction: *p<0.05, ** p<0.01 compared to *Braf* mice.**