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Supplementary Materials for

A β-Arrestin–Biased Agonist of the Parathyroid Hormone Receptor (PTH1R) Promotes Bone Formation Independent of G Protein Activation

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This PDF file includes:

Supplementary Data
Figs S1-S3

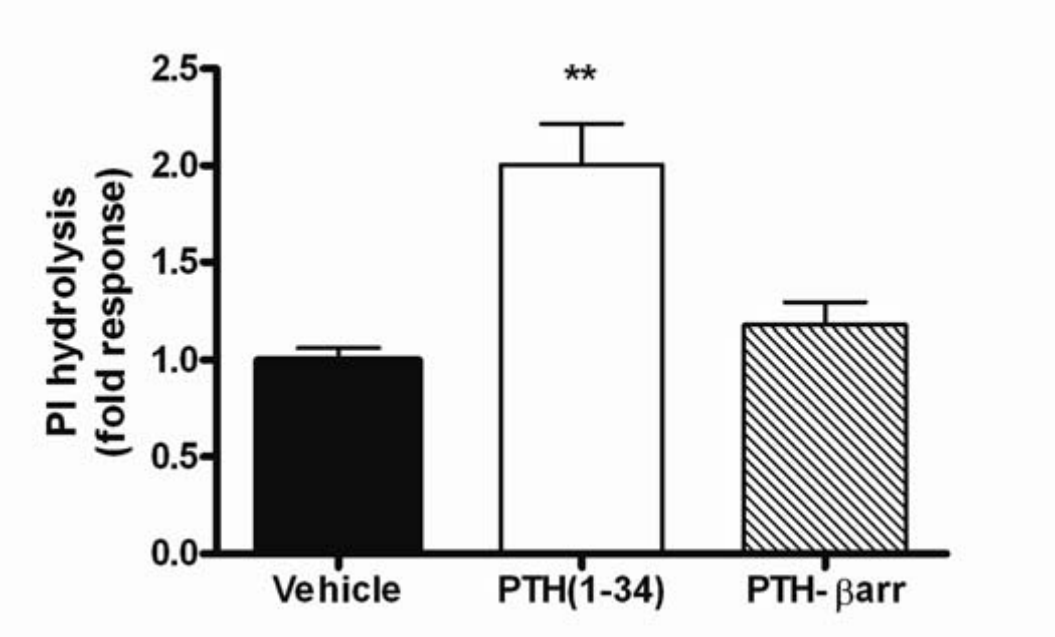

Fig. S1. PTH-βarr does not activate $G_{q/11}$ PI hydrolysis. PI hydrolysis in response to PTH(1-34) and PTH-βarr stimulation of endogenously expressed PTH1R in WT POBs. Data correspond to the mean ± SEM of four independent experiments. (**, $P<0.01$ compared with the non-stimulated WT POB using two-tailed unpaired t-test).

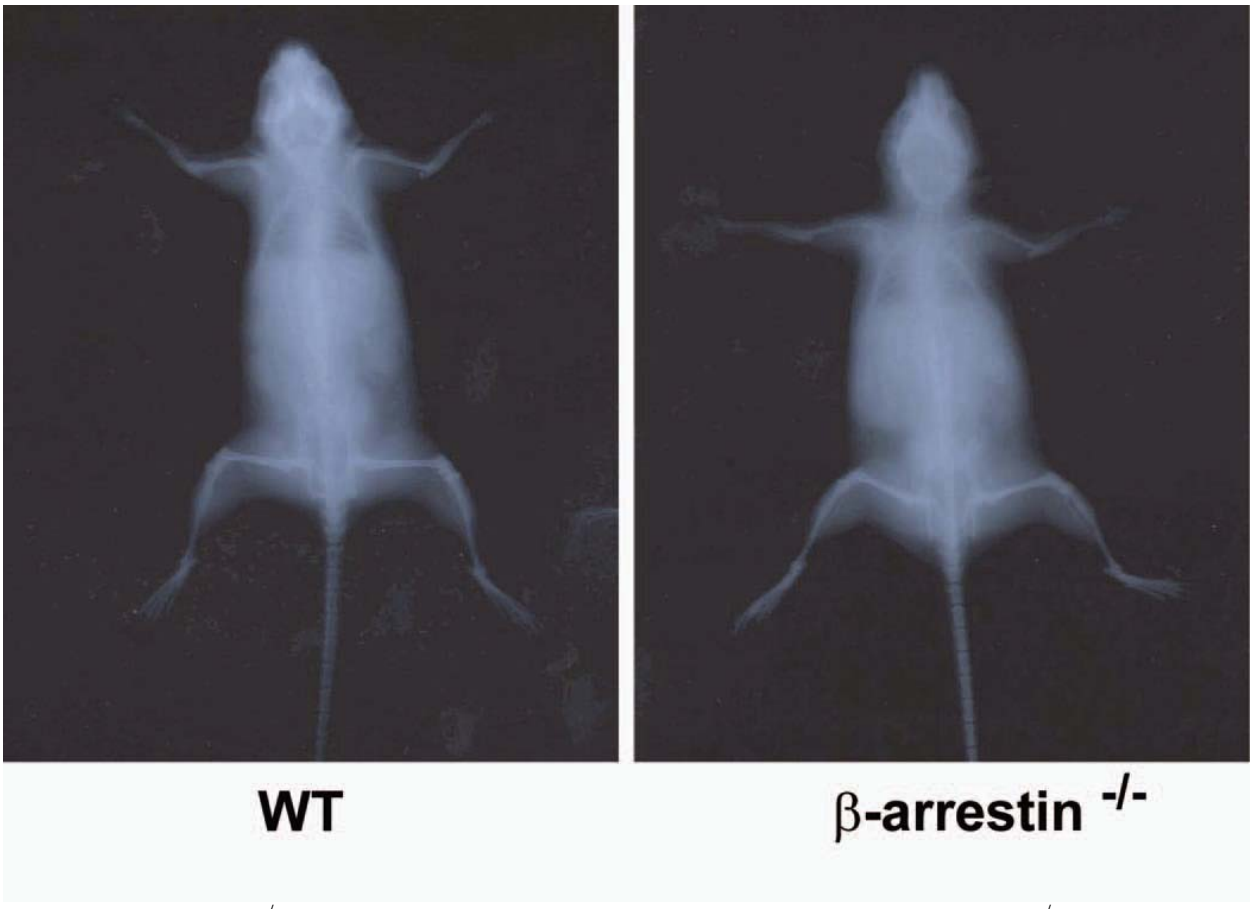

Fig. S2. β-arrestin2$^{-/-}$ mice show no skeletal or size abnormalities. β-arrestin2$^{-/-}$ and wild type mice were assessed by x-ray and for gross morphological features.

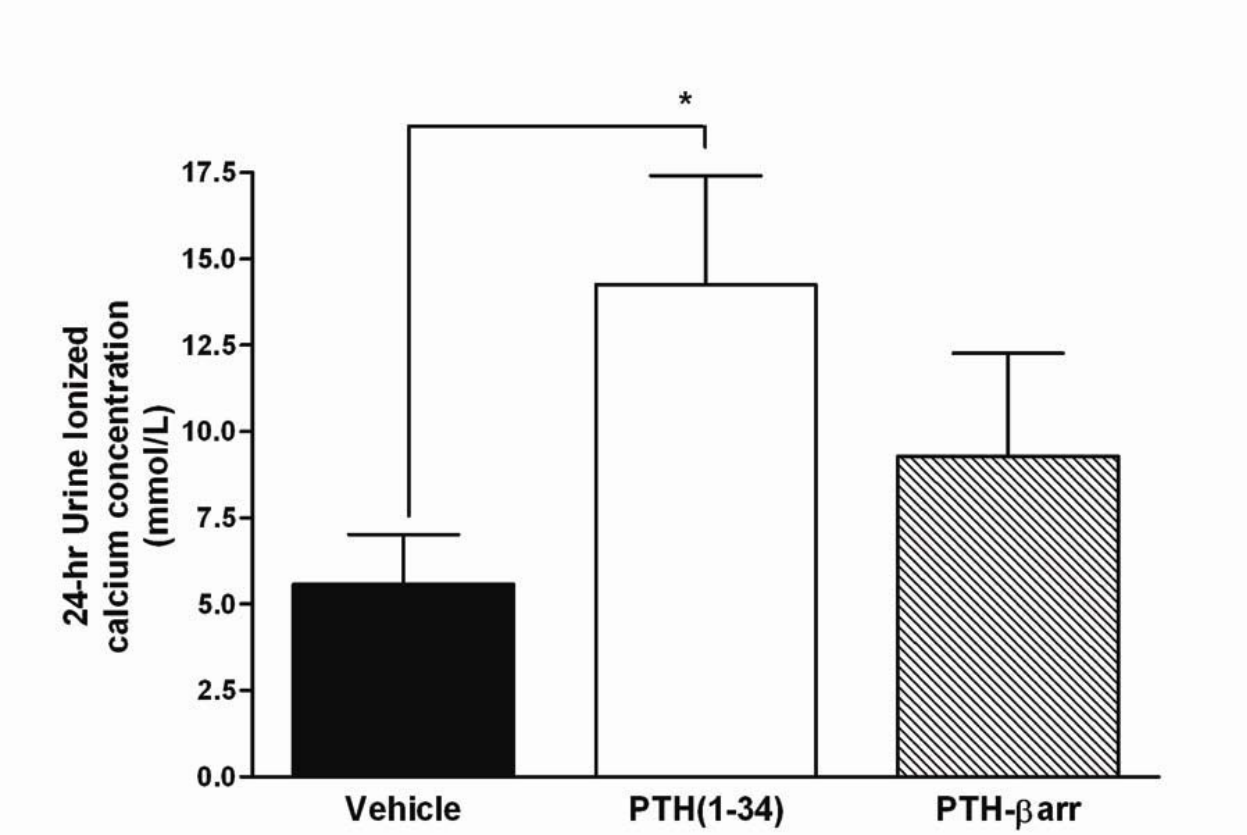

Fig. S3. PTH(1-34), but not PTH-βarr, increases urinary calcium. 24 hour urine calcium concentration was measured in male WT mice after 8 weeks of treatment with vehicle, PTH(1-34) or PTH-βarr. Mice were treated starting at nine weeks of age. Data represent the mean ± SEM of measurements taken from at least 7 male mice. (*, $P<0.05$ compared with vehicle treated WT mice; using two-tailed unpaired t-test).