SUPPLEMENTAL DATA

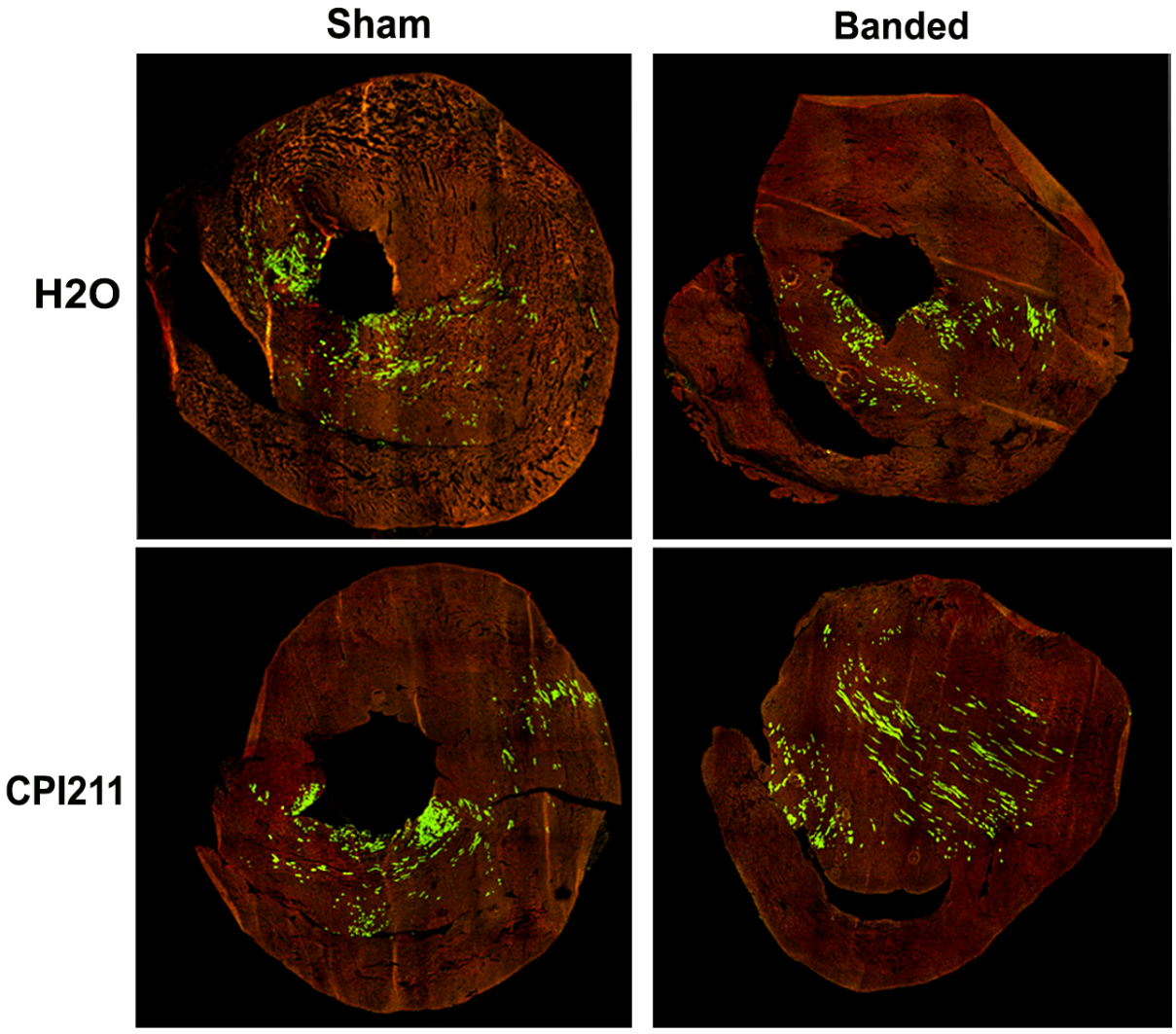
Luciferase assay

H9C2 rat cardiomyocytes were differentiated and transfected with a TGF-ß1-responsive PAI-1 promoter luciferase plasmid [1] and pRL-thymidine kinase transfection control plasmid before treating for 24h with 10 ng/mL TGFß1, in the presence of 500 nM CPI211 or vehicle. A Dual-Luciferase assay was performed per manufacturer's protocol (Promega, Madison, WI).

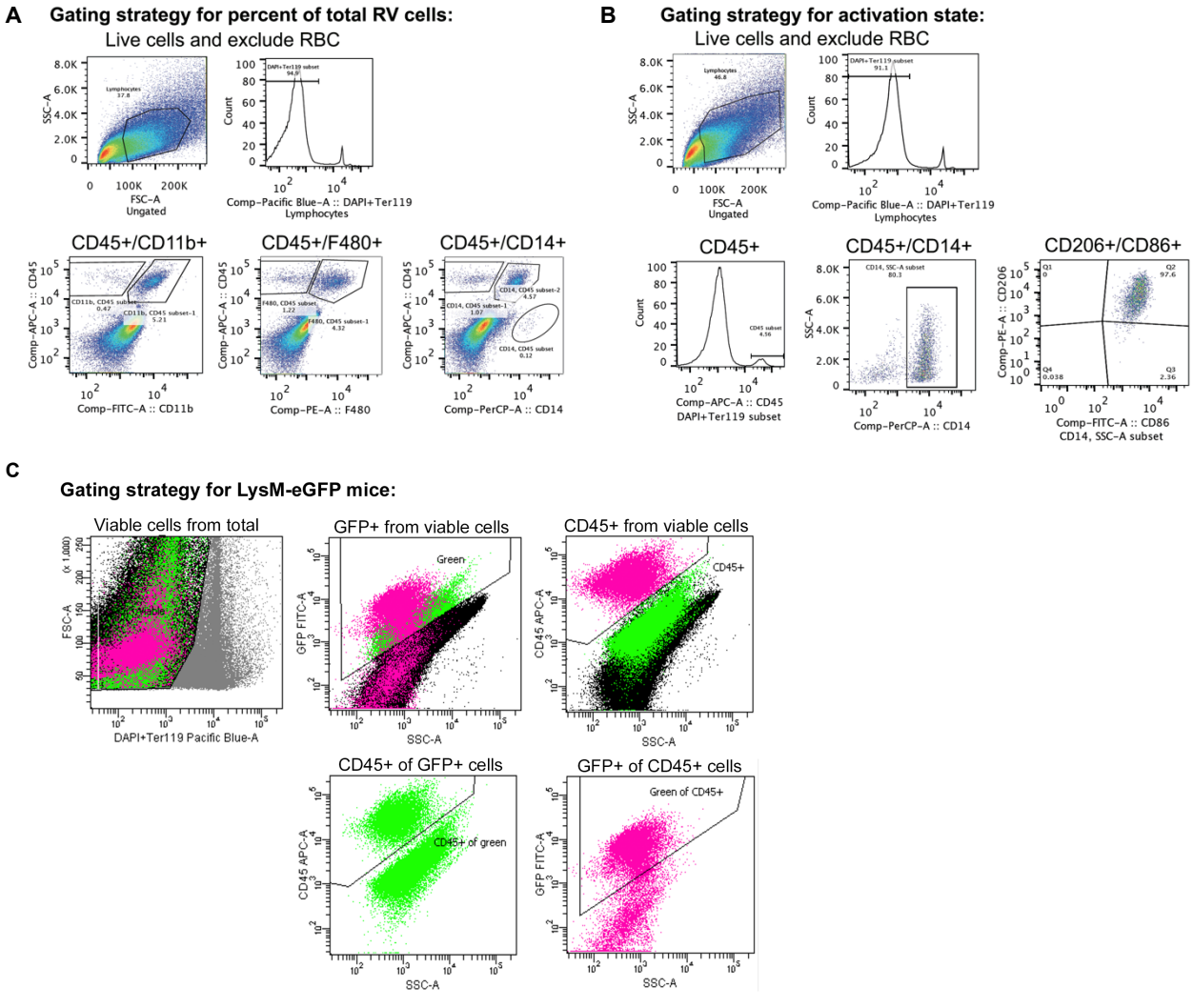
References

1. Hutcheson JD, Ryzhova LM, Setola V and Merryman WD. 5-HT(2B) antagonism arrests non-canonical TGF-beta1-induced valvular myofibroblast differentiation. *J Mol Cell Cardiol*. 2012;53:707-14.

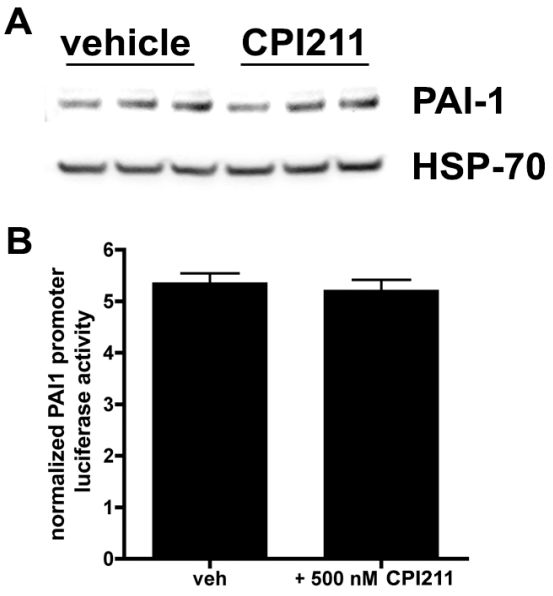
Supplemental Figure 1: Visualization of LymM-GFP cells. Whole hearts from LysM-Cre mice were snap-frozen, sectioned, and confocally visualized at 10x. Sections were stitched together with 15% overlap using NIS Elements software to get an overview of monocyte-lineage cells in the heart.



Supplemental Figure 2: (**A-B**) Gating strategies for FACS analysis of monocytes from C57Bl/6 mice, with representative images. (**C**) Gating strategy for LysM-eGFP RV (green = GFP+; fuchsia = CD45+). The GFP gate was derived using a GFP-negative littermate heart as FMO control.



Supplemental Figure 3: In cultured H9C2 cardiomyocytes, CPI211 does not block protein expression of canonical TGF-β target PAI1 (**A**), nor does it block induction of a luciferase activity driven by a PAI1 promoter (**B**).



Supplemental Figure 4: Immunoblot of TP receptor in RV from sham-operated and PAB mice. When normalized to HSP-70, mean band densities were 0.561 (sham) and 0.542 (PAB; SEM for each was 0.026). Intervening lanes were removed from blot where indicated.