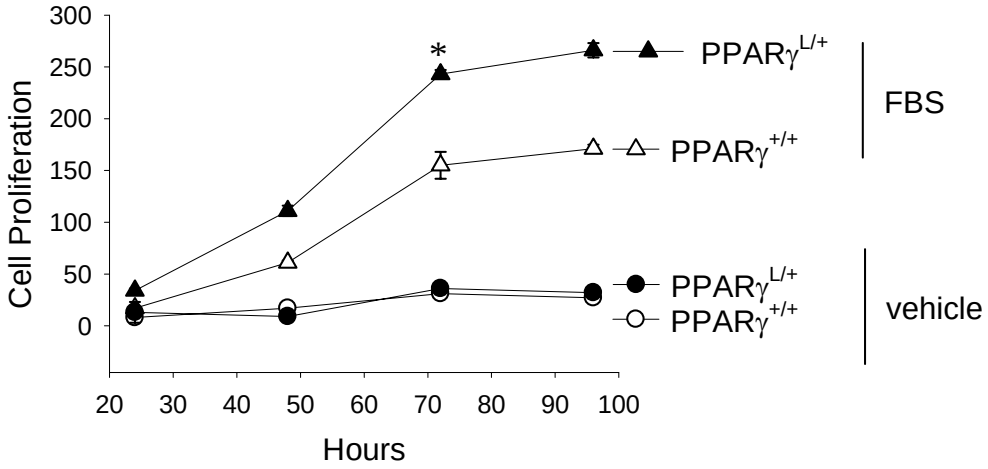

Supplemental Figure 1. Enhanced serum-stimulated proliferation of PPARγ$^{L/+}$ aortic SMC. Serum starved PPARγ$^{+/+}$ or PPARγ $^{L/+}$ SMCs were plated onto a 96-well dishes with FBS (10%) or vehicle for 24, 48, 72 and 96 hours. At each given time point, WST-1 was added and incubated in each well for 2 hours. The absorbance of the resulting solution was then measured at 450 nm. Values are presented as mean ± se. * P = 0.003 versus at PPARγ$^{+/+}$ 72 hours.

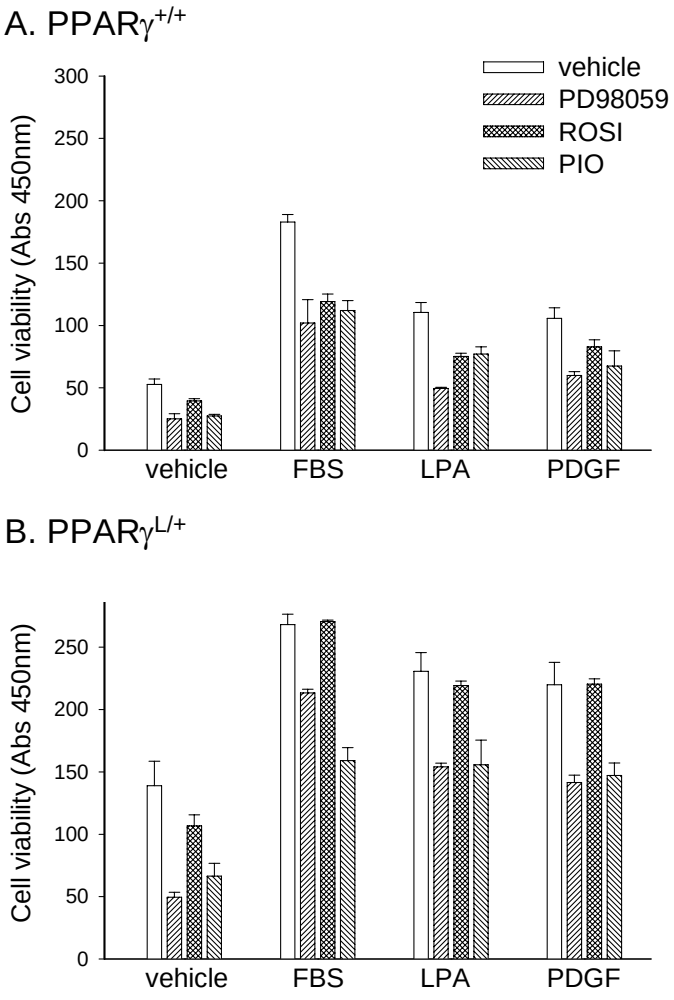

Figure 2. Effects of ERK inhibitors and TZDs on PPARγ$^{L/+}$ SMC proliferation. Serum-starved PPARγ $^{+/+}$ or PPARγ $^{L/+}$ SMCs were plated onto a 96-well dishes with vehicle, FBS (10%), LPA (1 μM), or PDGF (20 ng/ml) for 72 hours in the presence of either the MEK inhibitor PD98059 (10 μM) or the TZDs rosiglitazone (ROSI;10 μM) or pioglitazone (PIO; 10 μM). WST-1 was added and incubated in each well for 2 hours. The absorbance of the resulting solution was then measured at 450 nm. Values are presented as mean ± sd.

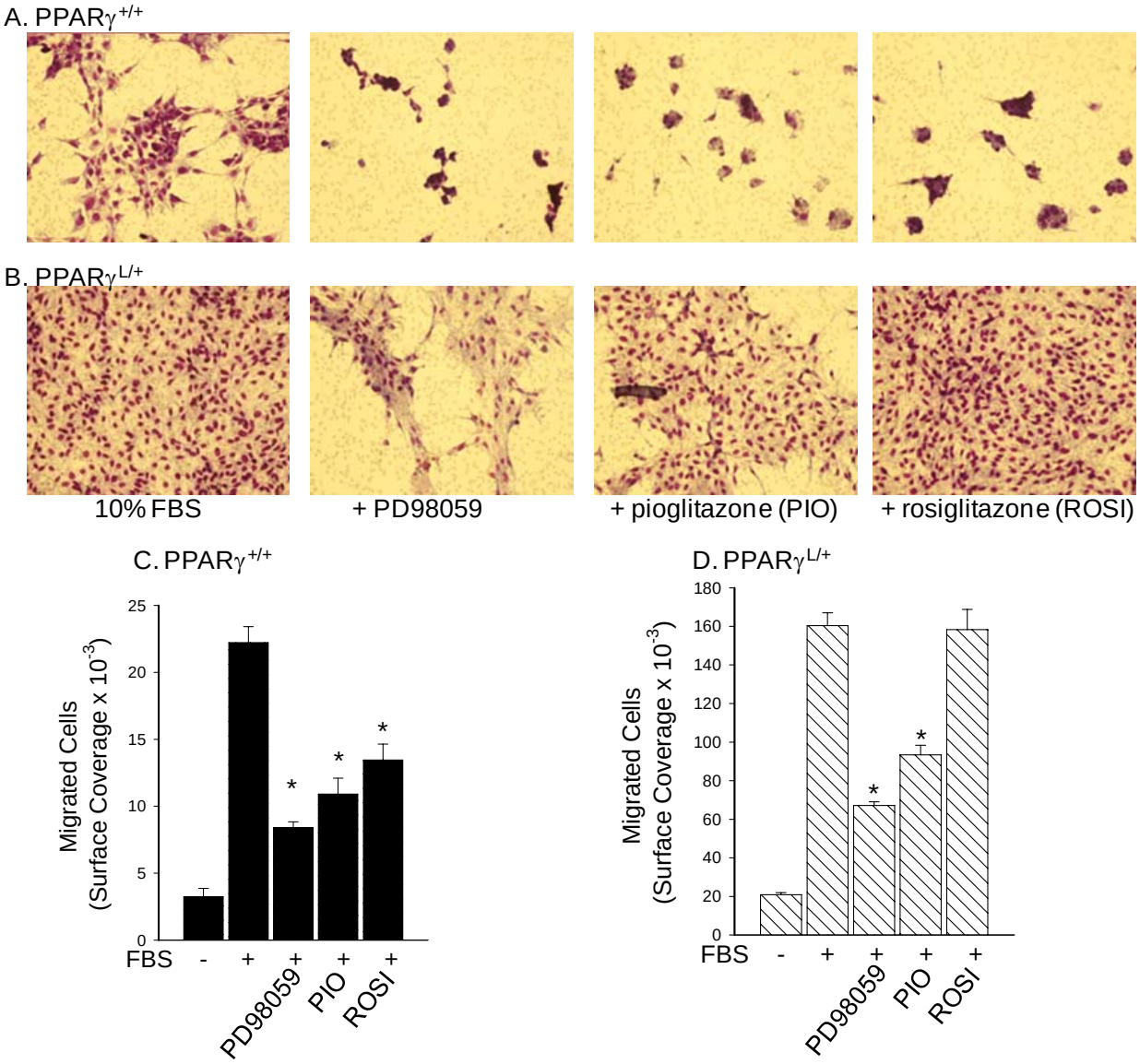

Figure 3. Effects of ERK inhibitors and TZDs on PPARγ$^{L/+}$ SMC migration. (A) Representative images of migrated PPARγ $^{+/+}$ SMCs and (B) PPARγ $^{L/+}$ SMCs stained with Diff-Quik®. The migration assay was performed in the presence of 10% FBS and either the MEK inhibitor PD98059 (10 μM) or the TZDs pioglitazone (PIO; 10 μM) or rosiglitazone (ROSI;10 μM). The surface area occupied by migrated cells is graphed as mean ± se. To demonstrate the effects of inhibitors, the results in PPARγ $^{+/+}$ and PPARγ $^{L/+}$ are graphed on different scales. *P<0.05 versus untreated by ANOVA.