Supplemental data

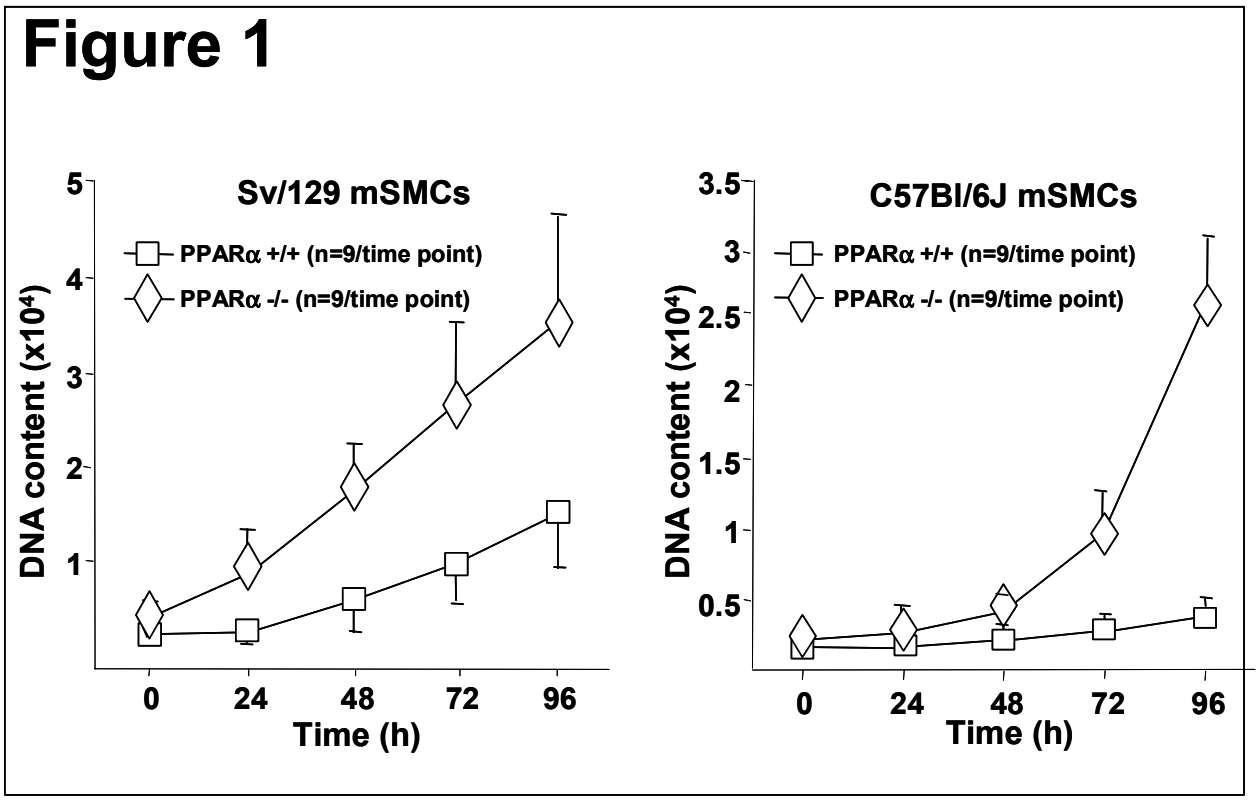
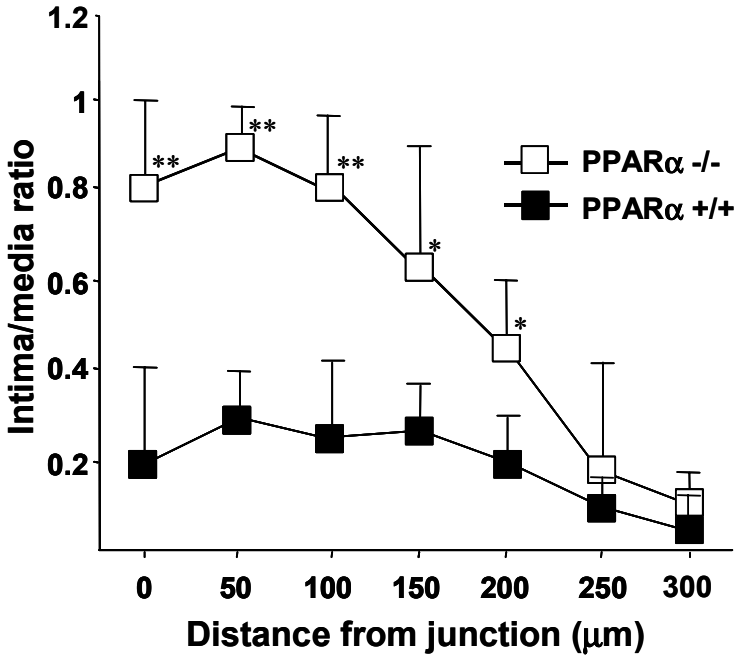


Figure 1. PPARα controls SMC proliferation. G_1-arrested PPARα-null (-/-) or wild-type (+/+) Sv/129 or C57Bl/6J mSMCs resumed growth at T0 in normal growth medium (GM). Values are the mean ± SEM of triplicate points from a single experiment performed with preparations from three different animals (n = 9/time point) .

Figure 2



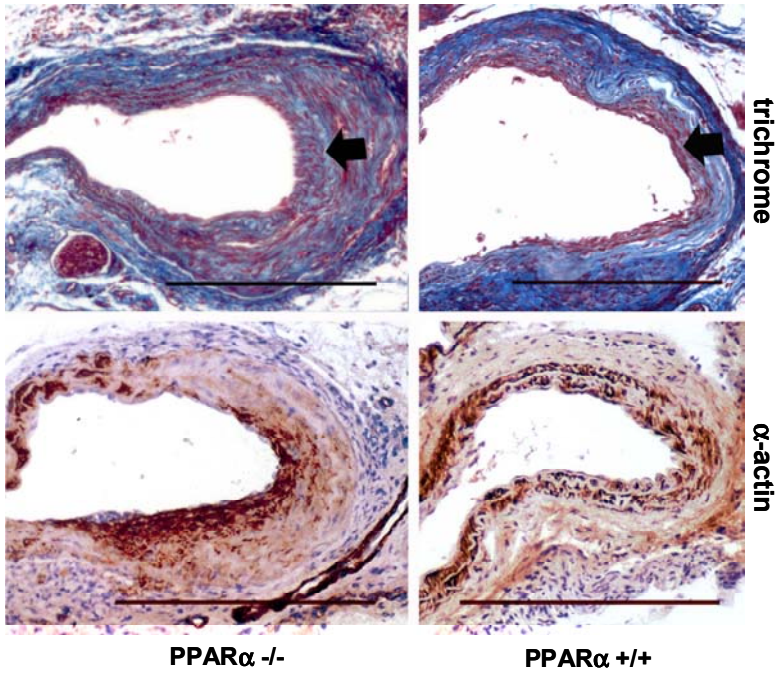


Figure 2. PPARα-deficiency abolishes hyperplasia-resistance in C57Bl/6J mice. Left carotid arteries from PPARα-null (n=7) or wild-type (n=5) C57Bl/6J mice were submitted to

a carotid mechanical injury and analyzed as described in "*Experimental Procedures*". ***A.*** **Topographic pattern of intima/media area ratio after injury.** Values are mean ± SEM of the intima/media ratios measured at the indicated distances from the junction on Masson's trichrome stained sections. *p≤0.05, **p≤0.01 *vs* wild-type sections ***B.*** **Representative sections of mouse carotid stained for morphometry or SMC identification.** Carotid section were stained with Masson's trichrome or an α-actin antibody, at respectively 100 and 50 μM from the junction. Bar = 300 μM. The internal elastic lamina delineating the neointima is indicated by a black arrow.

Figure 3

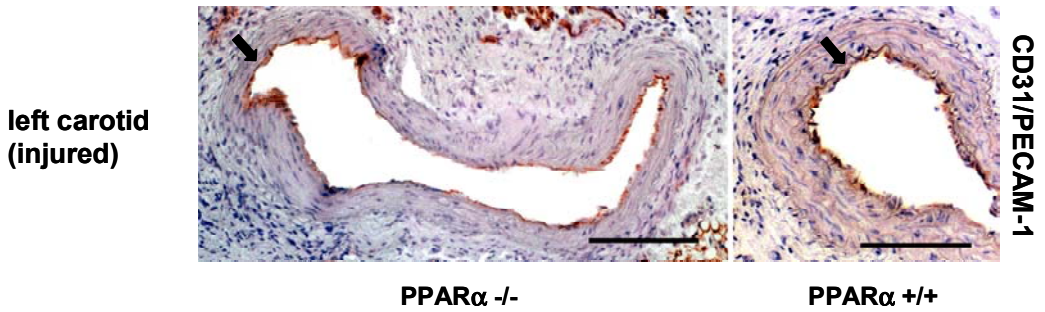


Figure 3. Reendothelialization in PPARα-null and wild-type mice after carotid injury. Representative PPARα-null (*left panel*) or wild-type (*right panel*) left carotid artery sections 3 weeks after injury at 200 μM from the junction stained with a biotin-conjugated anti-mouse CD31 (PECAM-1) monoclonal rat antibody (BD Biosciences; 1:100 dilution) for endothelial cell labelling. Immunostains were visualized using the Vector NovaRed substrate kit (Vector Laboratories) (red). Bar = 100 μM. The internal elastic laminæ delineating the neointima are indicated by a black arrow.