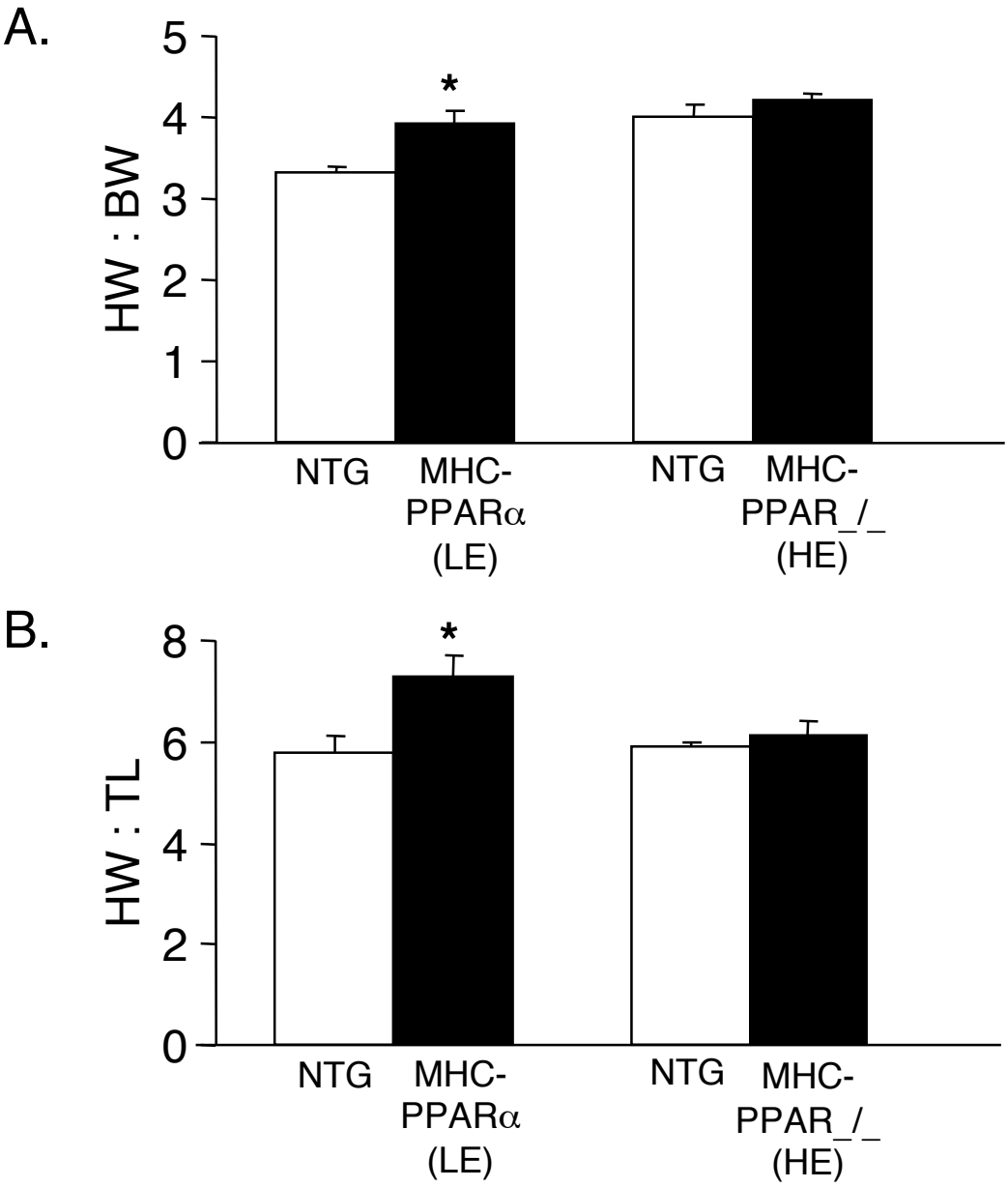
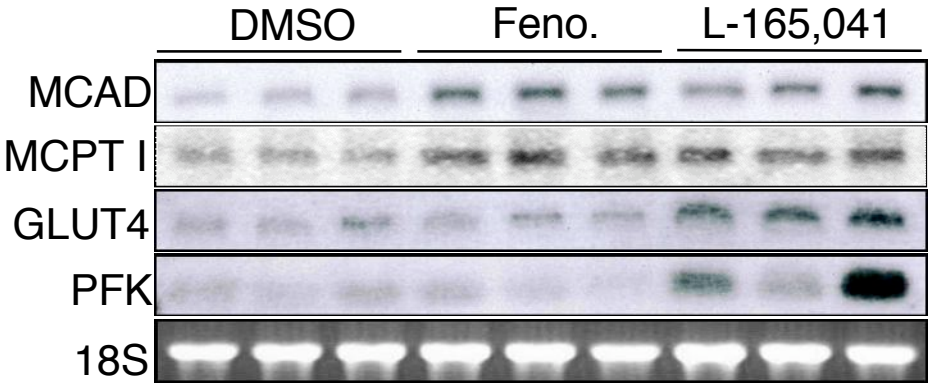
Supplemental Figure 1 (Burkart et al)



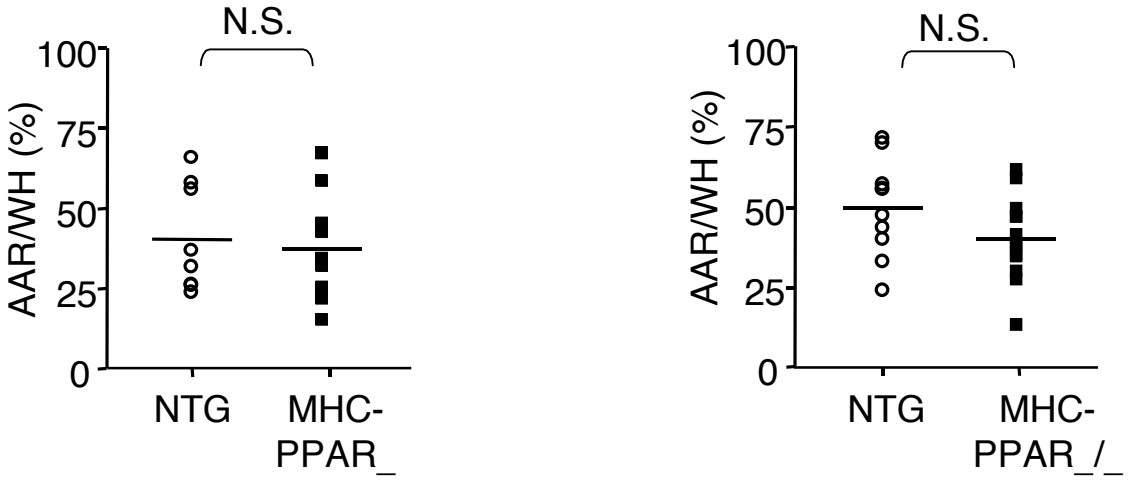
Supplemental Figure 1. Cardiomyopathy develops in MHC-PPARα (LE) mice, but not MHC-PPAR_/_ (HE) mice. Bars represent (**A**) mean biventricular-to-body-weight (HW:BW) ratios and (**B**) mean biventricular-to-tibial-length (HW:TL) ratios for 8-week old male and female MHC-PPAR_/_ (HE) and MHC-PPARα (LE) mice, and respective NTG littermates ($n \geq 15$ per group). *, $p < 0.05$ vs. NTG littermates.

Supplemental Figure 2 (Burkart et al)



Supplemental Figure 2. Representative Northern blot analyses performed with RNA isolated from rat ventricular cardiomyocytes exposed to fenofibrate (Feno), L-165,041, or vehicle (DMSO) for 48 hours. Gene abbreviations are defined in the text. 18s rRNA is shown as a loading control.

Supplemental Figure 3 (Burkart et al)



Supplemental Figure 3. Graphs represent percent of area-at-risk (AAR) normalized to the whole heart (WH) for MHC-PPARα or MHC-PPARβ/δ mice, or non-transgenic littermates, subjected to myocardial I/R injury. As shown, the mean percent AAR/WH (horizontal bar) was not different between NTG and MHC-PPARα mice (42.4 ± 5.6% vs. 34.9 ± 3.7%) or between NTG and MHC-PPARβ/δ mice (49.6 ± 4.8% vs. 40.0 ± 3.6%).