Supplemental Figure 11: Hepatic p38 MAPK activity associated with HCM is not due to inflammatory, oxidative or hypoxic stress. (A) Fatty acid composition of whole plasma from fasted 12 month old males. Mean±SEM; t-test; n = 5. (B) PGC1 immunoblot of FaO cell lysates (and liver control) following (24 or 64 hour) infection by GFP or PGC1-expressing adenovirus. (C) ELISA determination of circulating inflammatory cytokines; interleukin 1β (IL-1β), interleukin 6 (IL-6) and tumor necrosis factor α (TNFα). Mean±SEM; t-test; n=4. (D) Immunoblot analysis of phosphorylated and total apoptosis signaling kinase-1 (ASK-1) in the 12 month liver. Mean±SEM; t-test; n=5-7. (E) Measurement of hepatic reactive oxygen species (ROS) in 12 month old males. Normalized to tissue protein content. Mean±SEM; t-test; n=3-4. (F) Measurement of plasma lipid peroxides (TBARS, thiobarbituric acid reactive substances) in 12 month wildtype and HCM mice. Mean±SEM; t-test; n = 4. (G) Immunoblot analysis of steady-state hypoxia inducible factor 1α (HIF1α) levels in the 12 month livers of wildtype and HCM males. Loading was normalized to β-tubulin. Mean±SEM; t-test; n = 5. (H) qPCR of HIF1α target vascular endothelial growth factor (Vegf) in the 12 month livers of wildtype and HCM males. Mean±SEM; t-test; n = 5. (I) Diagram of possible routes to PKC and p38 MAPK activation; Blue represents observed increases, Grey represents unaltered hepatic components. * p < 0.05, significantly different from wildtype.