Supplemental Data

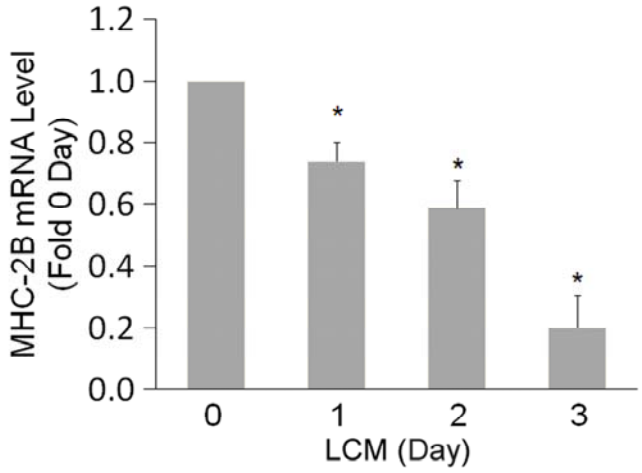

Figure S1. LCM suppresses MHC2B expression. C2C12 myotubes were treated with LCM for indicated time periods. Total RNA was collected for real time PCR analysis of MHC2B. * denotes a difference ($p < 0.05$) from Day 0 based on ANOVA.

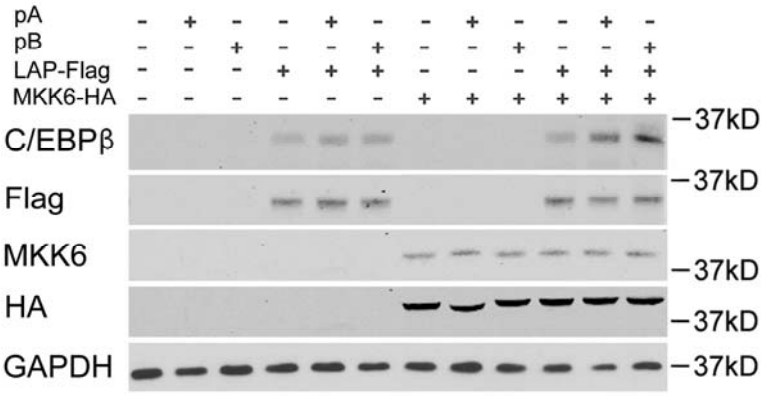

Figure S2. Expression of LAP and MKK6bE in C2C12 myoblasts co-transfected with luciferase reporters. Plasmids encoding the active form of C/EBPβ (LAP) fused to the Flag tag, MKK6bE fused to the HA tag, construct pA or pB were co-transfected into C2C12 myoblasts. In 24 h, the cells were lysed and Western blot analysis was performed to verify the expression of LAP and MKK6bE.

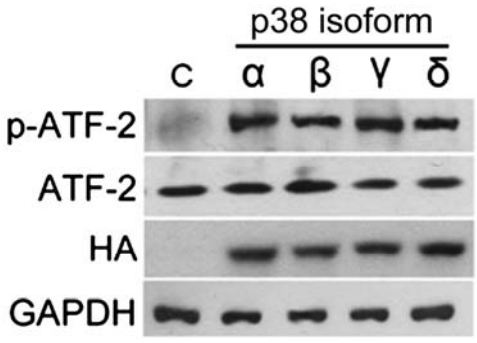

Figure S3. Expression of active mutants of p38 MAPK isoforms activates p38 substrate ATF2 in C2C12 cells. Plasmids encoding an active mutant of the α, β, γ or δ isoforms along with the HA tag were individually transfected into C2C12 myoblasts with the empty vector as control (C). In 24 h, nuclear extracts were prepared from the cells. Expression of the p38 isoforms was evaluated by Western blot analysis of p38 and HA. Activity of the p38 isoforms was evaluated by Western blot analysis of the phosphorylation state of ATF2.

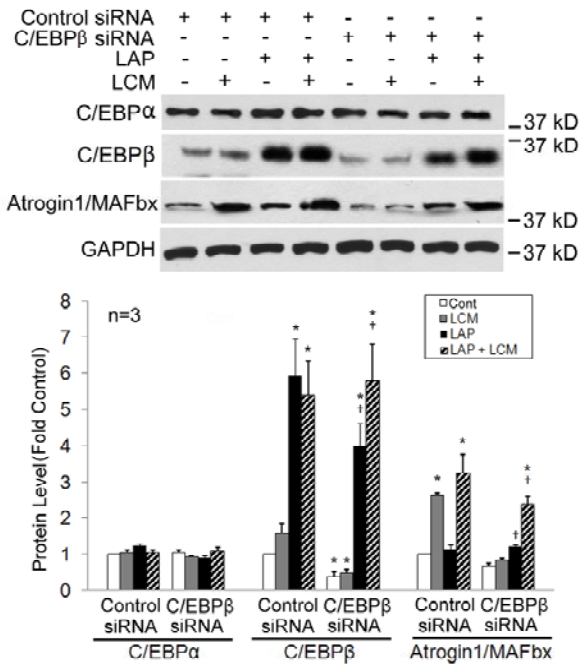

Figure S4. The C/EBPβ siRNA specifically knocks down C/EBPβ gene expression. C2C12 myoblasts were transfected with C/EBPβ or control siRNA along with a plasmid encoding LAP as indicated. After 72 h of differentiation, myotubes were treated with control medium or LCM for 8 h. Levels of C/EBPα, C/EBPβ, and atrogin1/MAFbx were evaluated by Western blot analysis (upper panel). Densitometry data of the blot was normalized to GAPDH (lower panel). * denotes a difference from the control of control siRNA ($p < 0.05$) and † denotes a difference from the control of C/EBPβ siRNA ($p < 0.05$) as indicated by ANOVA.