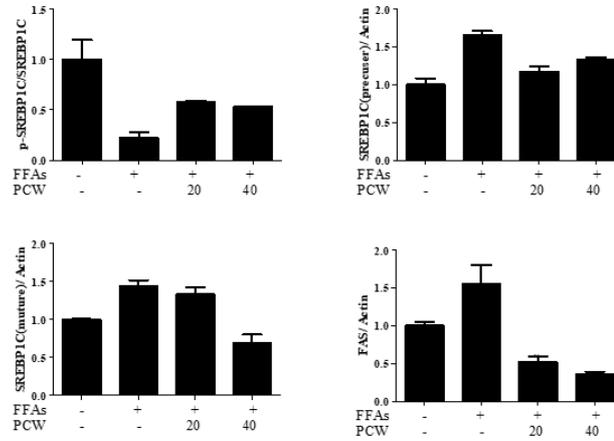
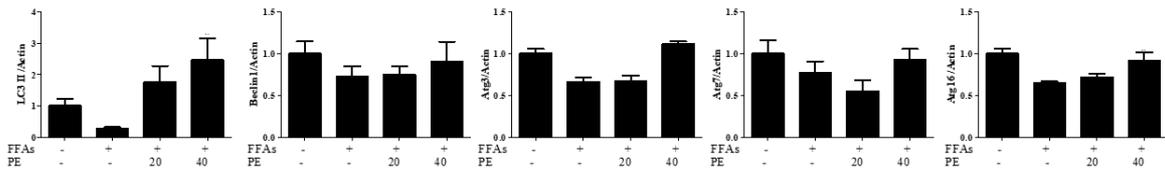


Supplementary Materials

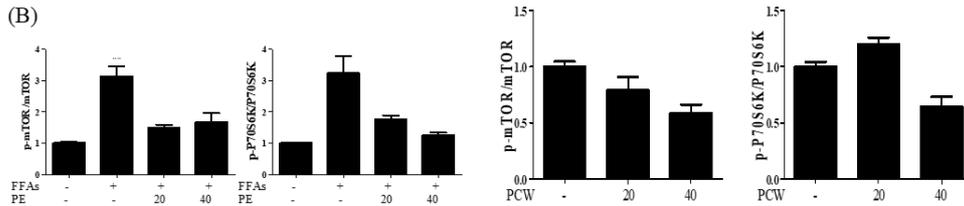


Supplementary Figure S1. PCW inhibited lipogenesis in HepG2 cells treated with FFA. HepG2 cells were treated with FFA (1 mM) and/or PCW (20 or 40 μ g/ml) for 24 h. Protein levels of p-SREBP1C, SREBP1C and FAS were analyzed by western blot. Bar graphs represent densitometric analysis of band intensity ratio for p-AMPK/AMPK, p-ACC/ACC.

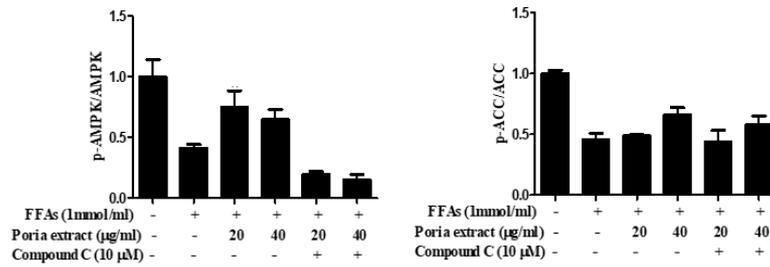
(A)



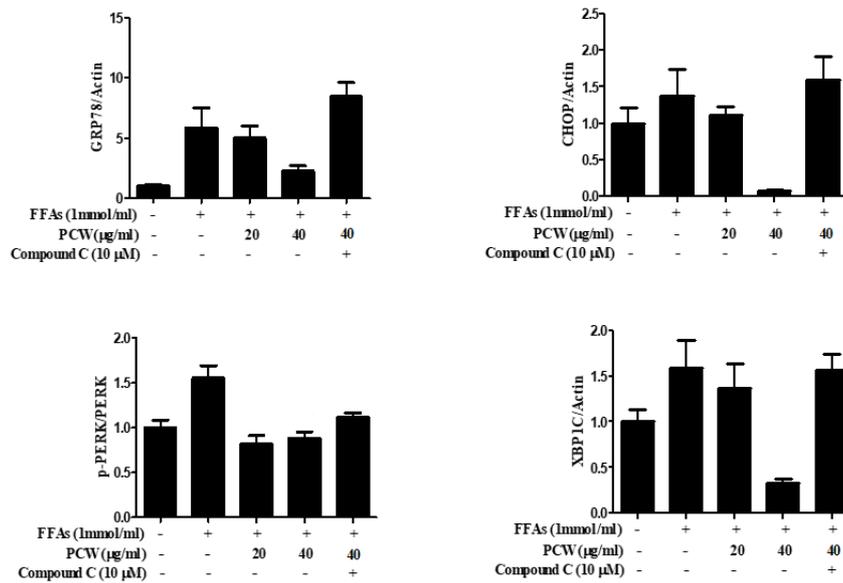
(B)



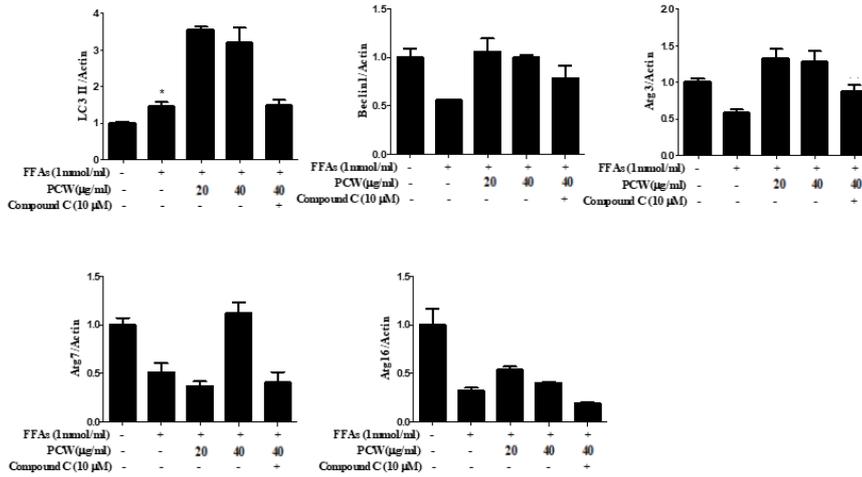
Supplementary Figure S2. PCW activated autophagy in HepG2 cells treated with FFA. HepG2 cells were treated with FFA (1 mM) and/or PCW (20 or 40 μ g/ml) for 24 h. (A) Protein levels of autophagy markers were analyzed by western blot. (B) The phosphorylation of p-mTOR/p-P70S6K was determined by western blot. Bar graphs represent densitometric analysis of band intensity.



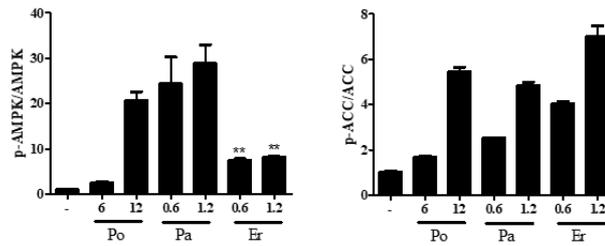
Supplementary Figure S3. Pretreatment with compound C inhibited PCW-mediated activation of AMPK in HepG2 cells treated with FFA. HepG2 Cells were pre-treated with compound C (Comp C, 10 µM) for 3 h and then treated with FFA (1 mM) and PCW (20 or 40 µg/ml) for 24 h. The phosphorylation of AMPK/ACC was determined by western blot. Bar graphs represent densitometric analysis of band intensity ratio for p-AMPK/AMPK and p-ACC/ACC.



Supplementary Figure S4. Inhibition of AMPK using compound C reversed PCW-mediated induction on ER stress in HepG2 cells treated with FFA. HepG2 Cells were pre-treated with compound C (Comp C, 10 µM) for 3 h and then treated with FFA (1 mM) and PCW (20 or 40 µg/ml) for 24 h. Bar graphs represent densitometric analysis of band intensity.



Supplementary Figure S5. Inhibition of AMPK using compound C reversed PCW-mediated reduction of autophagy in HepG2 cells treated with FFA. HepG2 Cells were pre-treated with compound C (Comp C, 10 µM) for 3 h and then treated with FFA (1 mM) and PCW (20 or 40 µg/ml) for 24 h. Bar graphs represent densitometric analysis of band intensity.



Supplementary Figure S6. Poricoic acid, pachymic acid and ergosterol activated AMPK in HepG2 cells. HepG2 cells were treated with Po (6 or 12 µM), Pa (0.6 or 1.25 µM) or Er (0.6 or 1.25 µM) for 24 h. The phosphorylation of AMPK/ACC was determined by western blot. Bar graph represents densitometric analysis of band intensity for p-AMPK/AMPK and p-ACC/ACC.