

## Supplementary Information

### **Chinese olive extract ameliorates hepatic lipid accumulation**

#### ***in vitro* and *in vivo* by regulating lipid metabolism**

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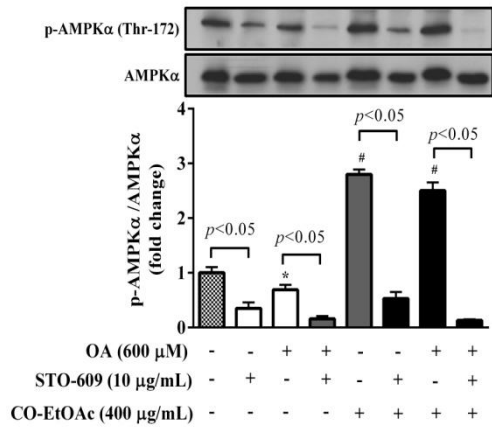
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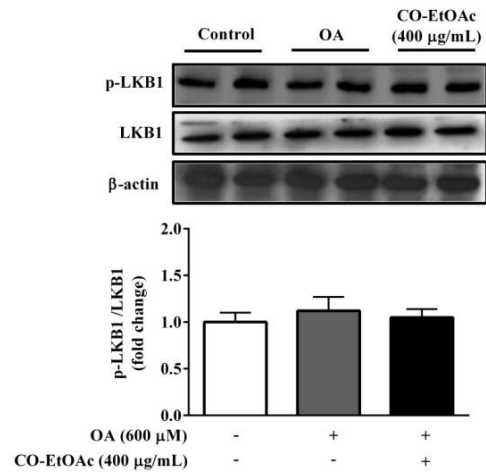
**Table S1.** Primer sequences used for qRT-PCR analysis

<b>Gene</b>	<b>Forward primer</b>	<b>Reverse primer</b>
CD36	GCTTGCAACTGTCAGCACAT	GCCTTGCTGTAGCCAAGAAC
FAS	CTGAGATCCCAGCACTTCTTGA	GCCTCCGAAGCCAAATGAGTGA
SREBP-1c	AGGTGTATTTGCTGGCTTGGT	AGAGATGACTAGGGAAGTGTGTGT
ACC1	GGCTGGGCTTGTATAAGT	GATAATATGGCAATCTCAGT
PPAR $\alpha$	CCTGCTTCCTGCCACTTG	G TTCACCCTGATTCCTGA
CPT-1	ACATCCCTAAGCAGTGCCAG	TCGTCCGGCACTTCTTGATC
FABP	CTGTGGAAAGGAAGCCTCGTT	CTCAAAGTTCTCCTGGCTCTGC
ACOX	ACCAACTGTCACACTAACATATCA	GGCATGTAACCCGTAGCACT
ATGL	CTTGAGCAGCTAGAACAATG	GGACACCTCAATAATGTTGGC
HSL	GCTGGAGGAGTGTTTTTTTGC	AGTTGAACCAAGCAGGTCACA
MGL	TCGGAACAAGTCGGAGGT	TCAGCAGCTGTATGCCAAAG
r18S	TATCCCATGACCCGCC	GTGAGGTTTCCCGTGTT

A



B



C

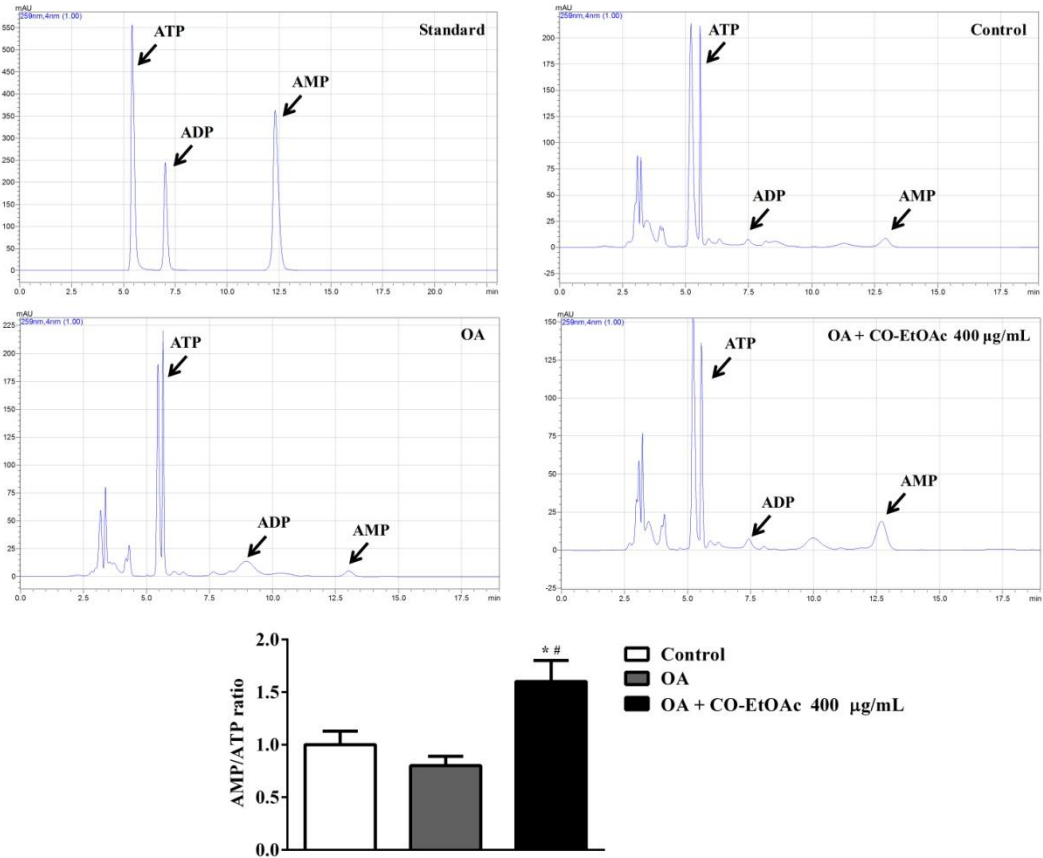


Figure S1. The involvement of CaMKK $\beta$ , LKB1, and AMP/ATP ratio in CO-EtOAc-induced AMPK activation. Cells were treatment with 600  $\mu$ M OA for 3 h and then treated with 400  $\mu$ g/mL of CO-EtOAc for another 21 h after pre-incubation. The levels of protein expression of (A) phosphorylated/total AMPK $\alpha$  in the presence or absence of 10  $\mu$ g/mL STO-609 (a CaMKK $\beta$  inhibitor) for 1 h before CO-EtOAc treatment and (B) phosphorylated/total LKB1 were measured by western blot analysis. (C) Intracellular ATP, ADP, and AMP contents were analyzed using HPLC. The position of ATP, ADP and AMP are indicated by arrows. The histogram shows the ratios of AMP:ATP for each group. All results are expressed as the mean  $\pm$  SEM of three independent experiments. \* $p$  < 0.05 versus the DMSO control group; #  $p$  < 0.05 versus the OA group.