SUPPPLEMENTARY INFORMATION

Perilipin 5 fine-tunes lipid oxidation to metabolic demand and protects against lipotoxicity in skeletal muscle

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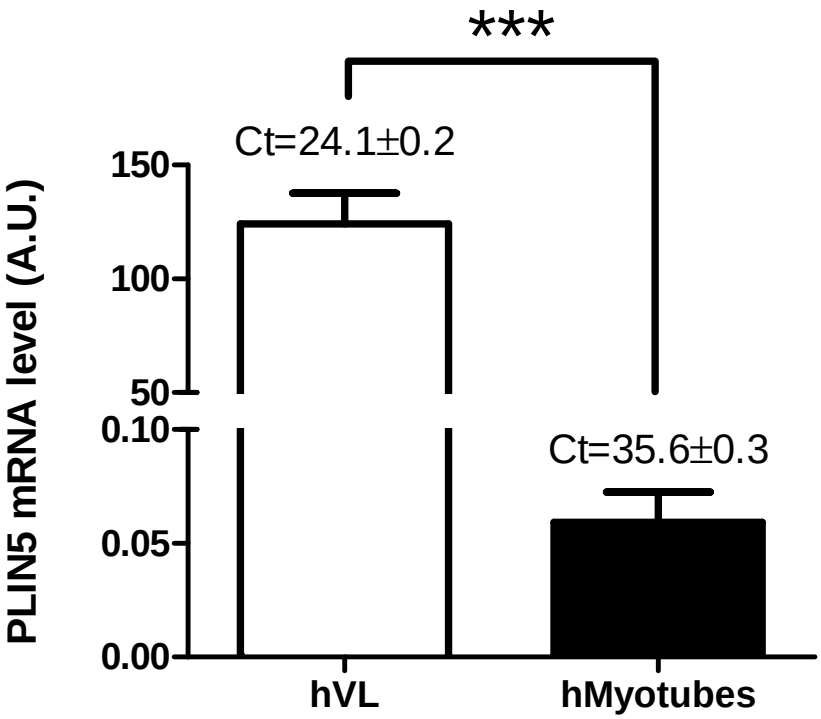
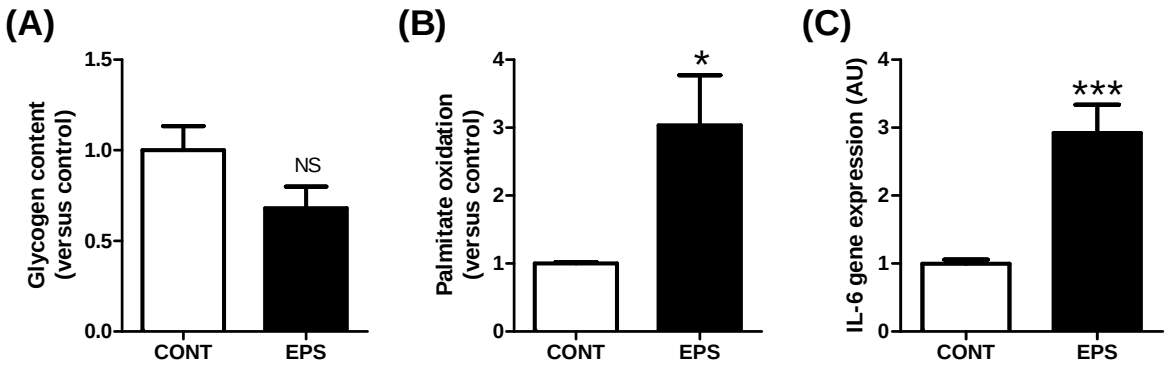
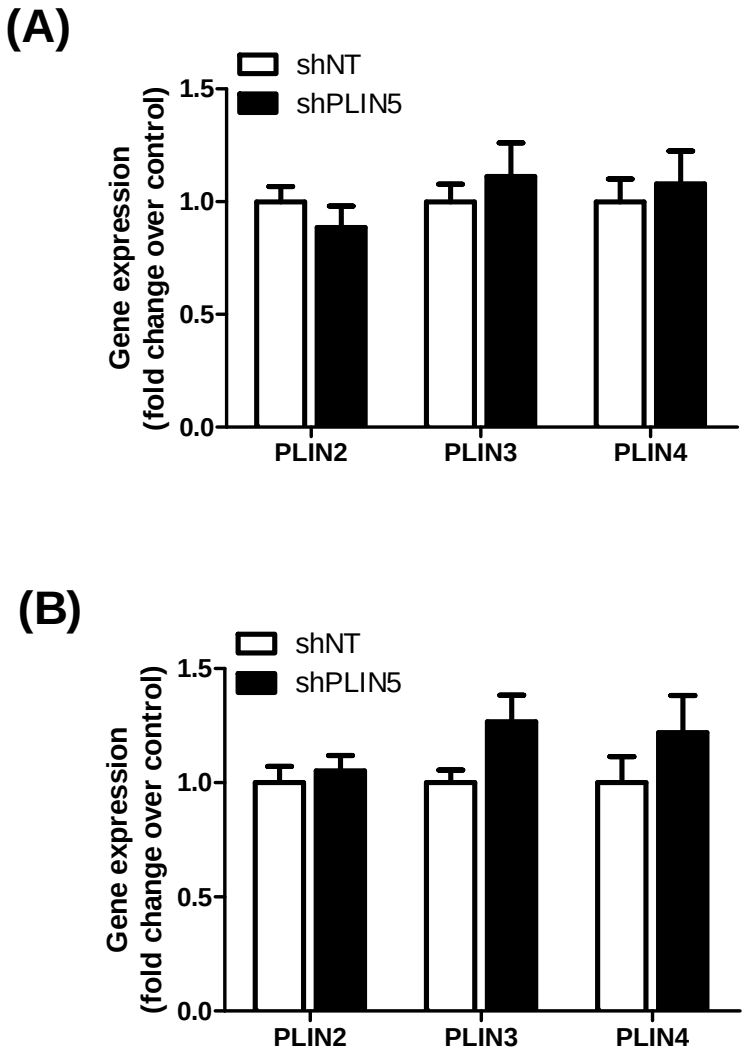


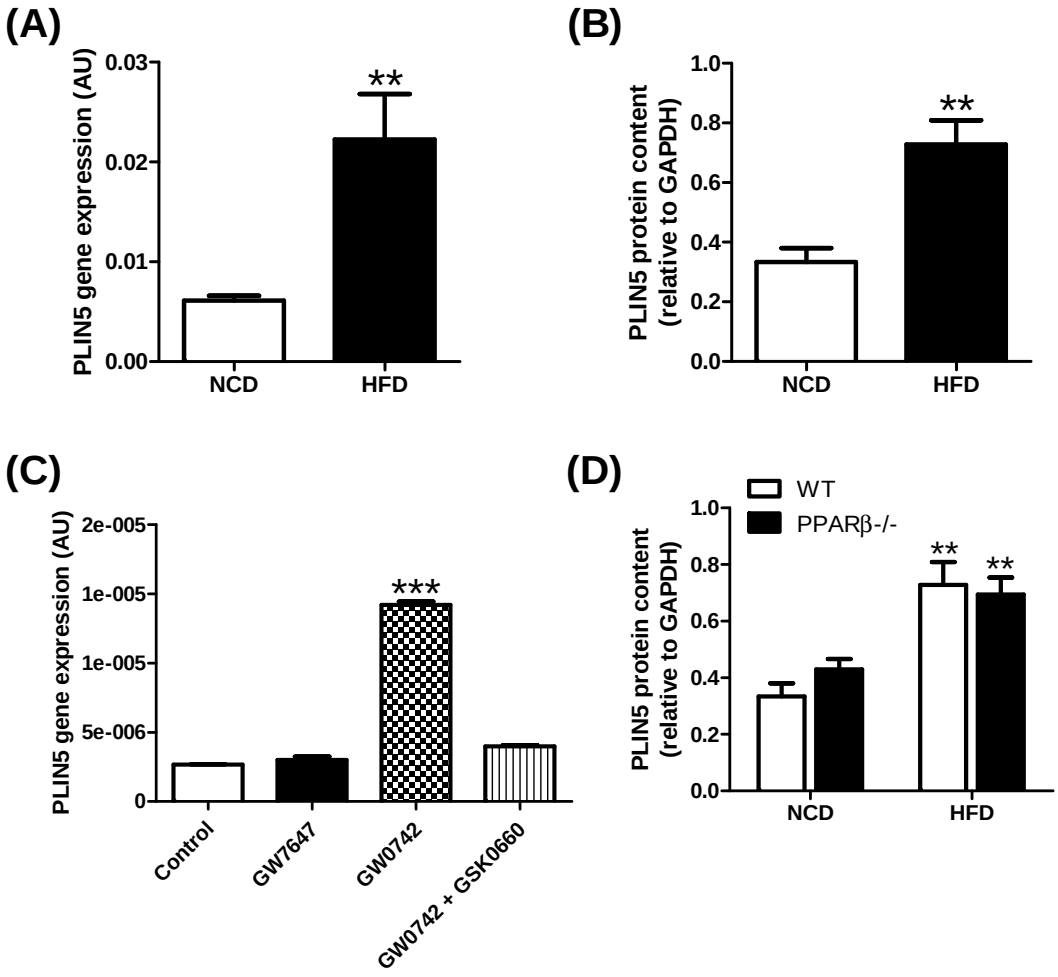
Supplemental Figure S1. PLIN5 gene expression in human native skeletal muscle and cultured myotubes. PLIN5 mRNA levels in human *vastus lateralis* muscle biopsy samples and human primary myotubes (n=9). Average Ct ± SEM are shown on the graph. ***p<0.001 versus hVL.



Supplemental Figure S2. Validation of the electrical pulse stimulation model in human myotubes. (**A**) Total glycogen content (n=4), (**B**) palmitate oxidation (n=4) and (**C**) interleukin-6 (IL-6) gene expression (n=4) were measured in control (CONT) and electrically stimulated (EPS) myotubes for 24 hours. NS : non-significant, *p<0.05, ***p<0.001.



Supplemental Figure S3. PLIN5 knockdown does not induce any compensatory changes in other PLIN isoforms. PLIN2, PLIN3 and PLIN4 gene expression in control (shNT) and PLIN5 knocked down (shPLIN5) *tiabialis anterior* muscles, measured in mice fed either (**A**) normal chow (NCD) or (**B**) high-fat diets for 12 weeks (n=6).



Supplemental Figure S4. PLIN5 is induced by high-fat feeding in mouse skeletal muscle independently of PPARβ activation. PLIN5 (**A**) gene expression and (**B**) protein content were measured in skeletal muscle of mice fed either normal chow (NCD) or high-fat (HFD) diet for 12 weeks (n=7). (**C**) PLIN5 gene expression was measured in myotubes treated for 24 h in absence (control) or presence of selective PPARα agonist GW7647 1 nM, PPARβ agonist GW0742 1nM and PPARβ antagonist GSK0660 500 nM (n=3). (**D**) PLIN5 protein content was measured in skeletal muscle from wild-type (WT) and PPARβ knockout (PPARβ-/-) mice fed either chow (NCD) or high-fat (HFD) diet (n=6). **p<0.01, ***p<0.001 versus control.