Undercarboxylated osteocalcin reverts insulin resistance induced by endoplasmic reticulum stress in human umbilical vein endothelial cells

Running title: Effects of ucOCN on insulin resistance

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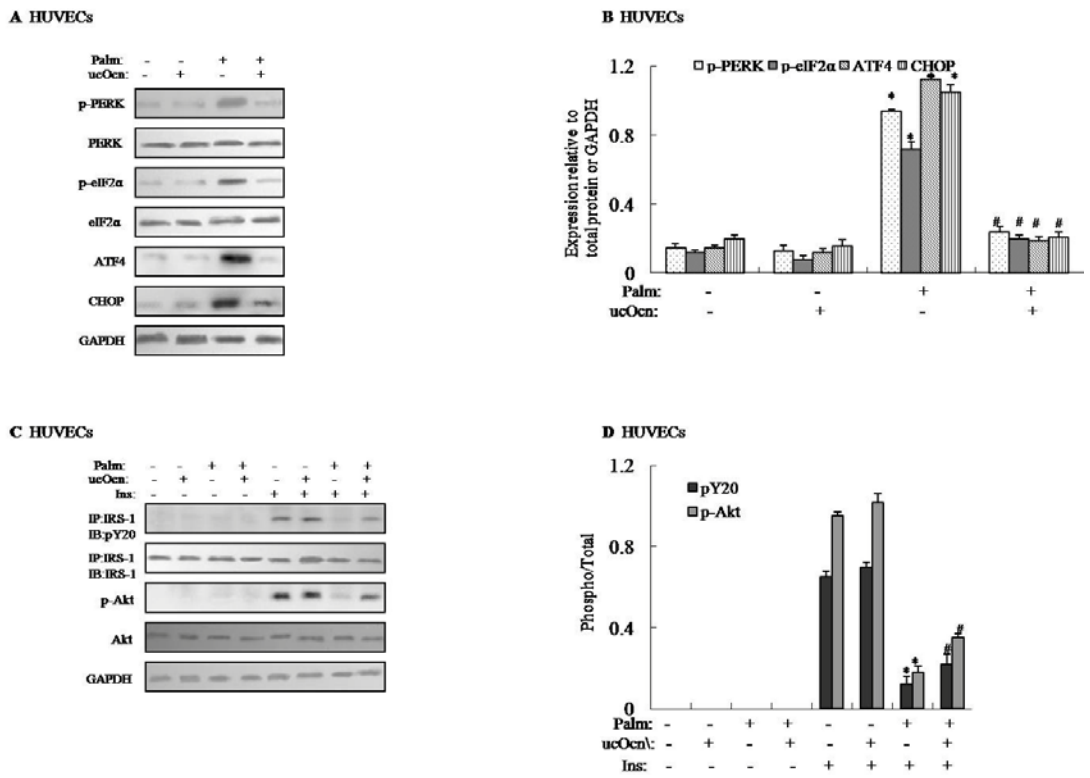
Supplemental Figure 1. Effects of ucOCN on palmitate-induced ER stress and impaired insulin signaling in HUVECs. Palmitate (Palm) was used to induce insulin resistance in HUVECs. The relative quantity of proteins was analyzed using Quantity One software. (A) Protein expression of ATF4, CHOP and phosphorylation of PERK and eIF2α in HUVECs. (B) Densitometric analyses of ATF4, CHOP and phosphorylation of PERK and eIF2α in HUVECs. (C) IRS-1 tyrosine phosphorylation and Akt Ser-473 phosphorylation in HUVECs. (D) Densitometric analyses of IRS-1 tyrosine phosphorylation and Akt Ser-473 phosphorylation in HUVECs. A representative blot from three independent experiments is shown and the data expressed as mean ± SEM in each bar graph represent the average of three independent experiments. *$P < 0.05$ (Palm vs. control). $^{\#}P < 0.05$ (Palm/ucOcn vs. Palm). IB, immunoblot; IP, immunoprecipitation.

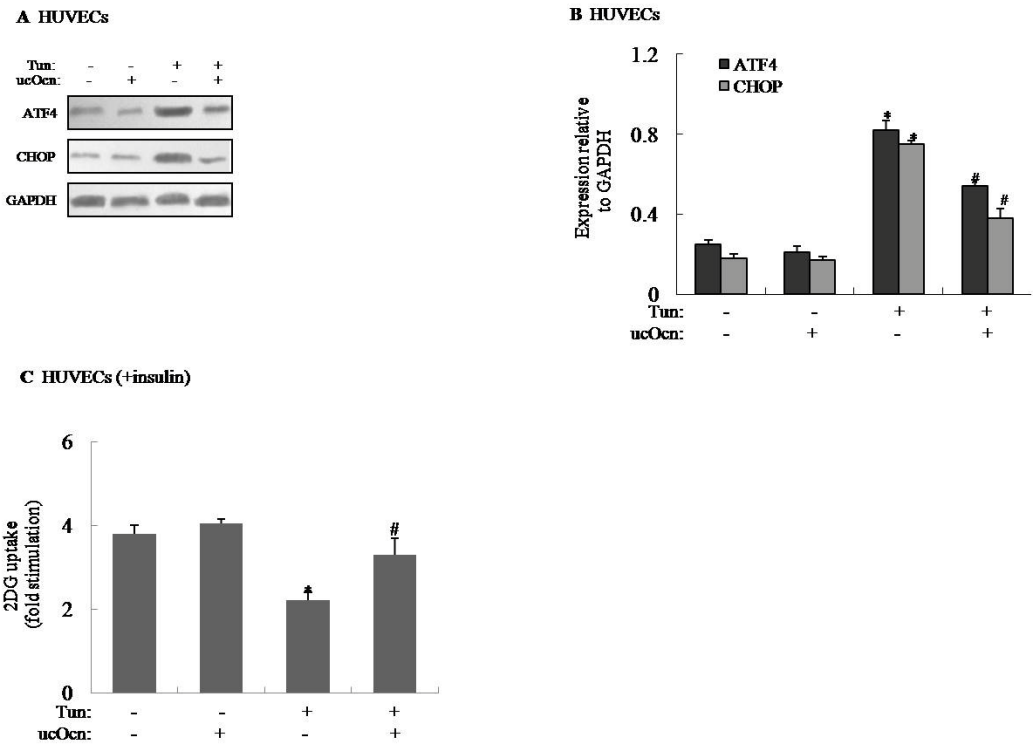
Supplemental Figure 2. (A) Protein expression of ATF4 and CHOP in HUVECs. (B) Densitometric analyses of ATF4 and CHOP in HUVECs. (C) Glucose uptake in HUVECs. *$P < 0.05$ (Tun vs. control). $^{\#}P < 0.05$ (Tun/ucOcn vs. Tun).

Supplemental Figure 3. (A) Phosphorylation of PERK and IRS-1 in HUVECs with or without Akti-1/2 in the presence or not of tunicamycin. (B) Densitometric analyses of PERK and IRS-1 in HUVECs with or without Akti-1/2 in the presence or not of tunicamycin. (C) Phosphorylation of PERK and IRS-1 in HUVECs with or without wortmannin in the presence or not of tunicamycin. (D) Densitometric analyses of PERK and IRS-1 in HUVECs with or without wortmannin in the presence or not of tunicamycin. *$P < 0.05$ (Tun/inhibitor vs. control).

Supplemental Figure 1



Supplemental Figure 2



Supplemental Figure 3