Figure S1. CIH enhances O-GlcNAc levels, OGT and p-p38 MAPK and p-ERK1/2, but decreases OGA expression in mesenteric arteries. Representative data of western blotting conducted in different rat samples. The groups were as follows: i) CON, normoxia (21% $O_2$); and ii) CIH, intermittent hypoxia cycles (6-8% $O_2$ for 2 min and 21% $O_2$ for another 2 min). CIH, chronic intermittent hypoxia; O-GlcNAc, O-linked-β-N-acetylglucosamine; OGT, O-GlcNAc transferase; OGA, O-GlcNAcase; p-, phosphorylated; CaMKII, $Ca^{2+}$/calmodulin-dependent kinase II; t-, total protein.

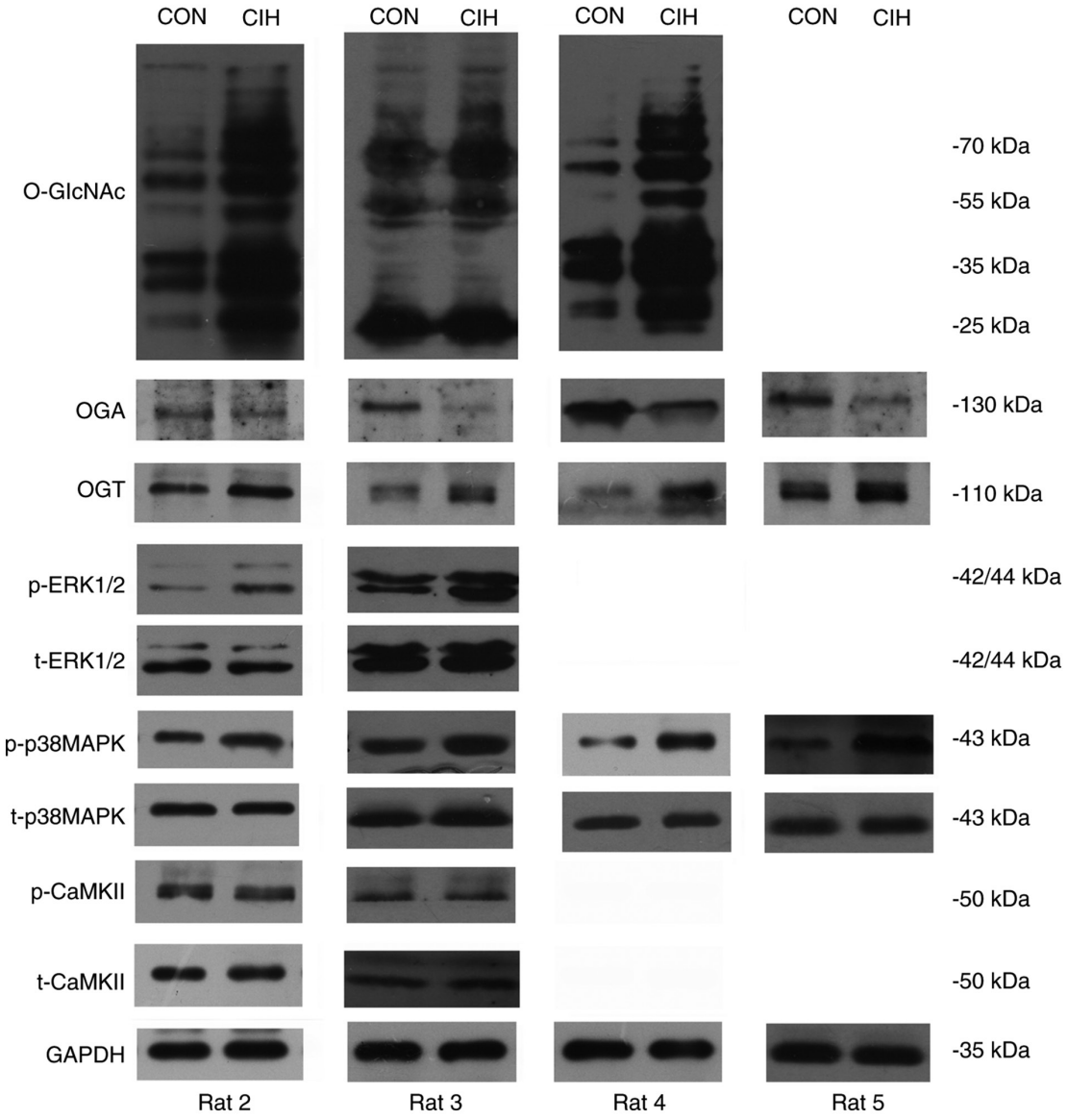

Figure S2. Protein O-GlcNAc levels interfere with the phosphorylation of p38 MAPK, ERK1/2 and CaMKII. (A) Protein levels of O-GlcNAc, OGA, OGT, p-p38 MAPK, p-ERK1/2 and p-CaMKII in cultured aortas that received either AON or AIH treatment in the presence of DMSO, PugNAc or ST045849. (B) Protein levels of O-GlcNAc, OGA, OGT, p-p38 MAPK, p-ERK1/2 and p-CaMKII in cultured aortas from the CON or CIH rats in the presence of DMSO, PugNAc or ST045849. Representative data of western blotting conducted in different rat samples. i) AON, 3-h normoxia; ii) AIH, 3-h intermittent hypxia treatment; iii) CON, normoxia (21% $O_2$); and iv) CIH, intermittent hypoxia cycles (6-8% $O_2$ for 2 min and 21% $O_2$ for another 2 min). O-GlcNAc, O-linked-β-N-acetylglucosamine; CaMKII, $Ca^{2+}$/calmodulin-dependent kinase II; OGA, O-GlcNAcase; OGT, O-GlcNAc transferase; AIH, acute intermittent hypoxia; CON, control; CIH, chronic intermittent hypoxia; p-, phosphorylated; t-, total protein.