Cyclooxygenase inhibition improves endothelial vasomotor dysfunction of visceral adipose arterioles in human obesity

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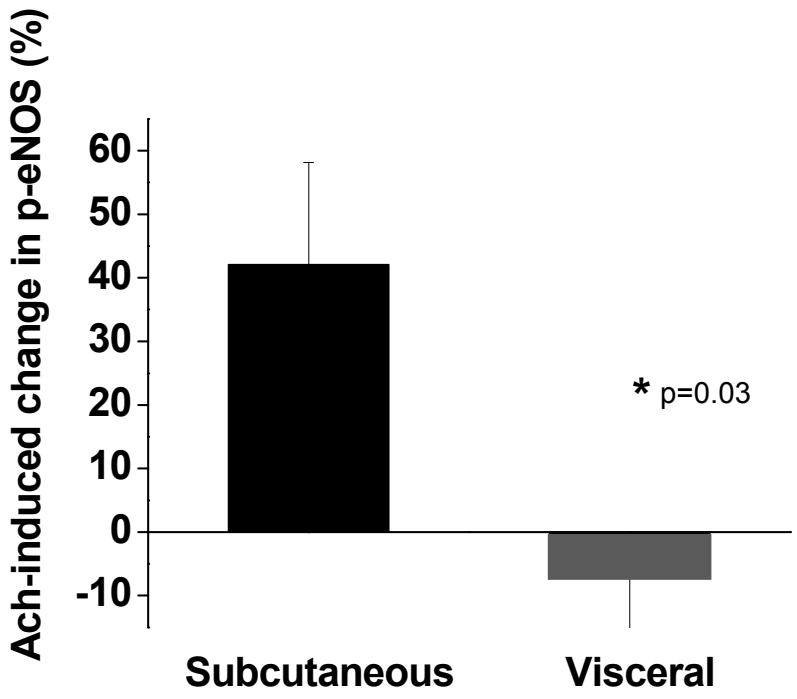
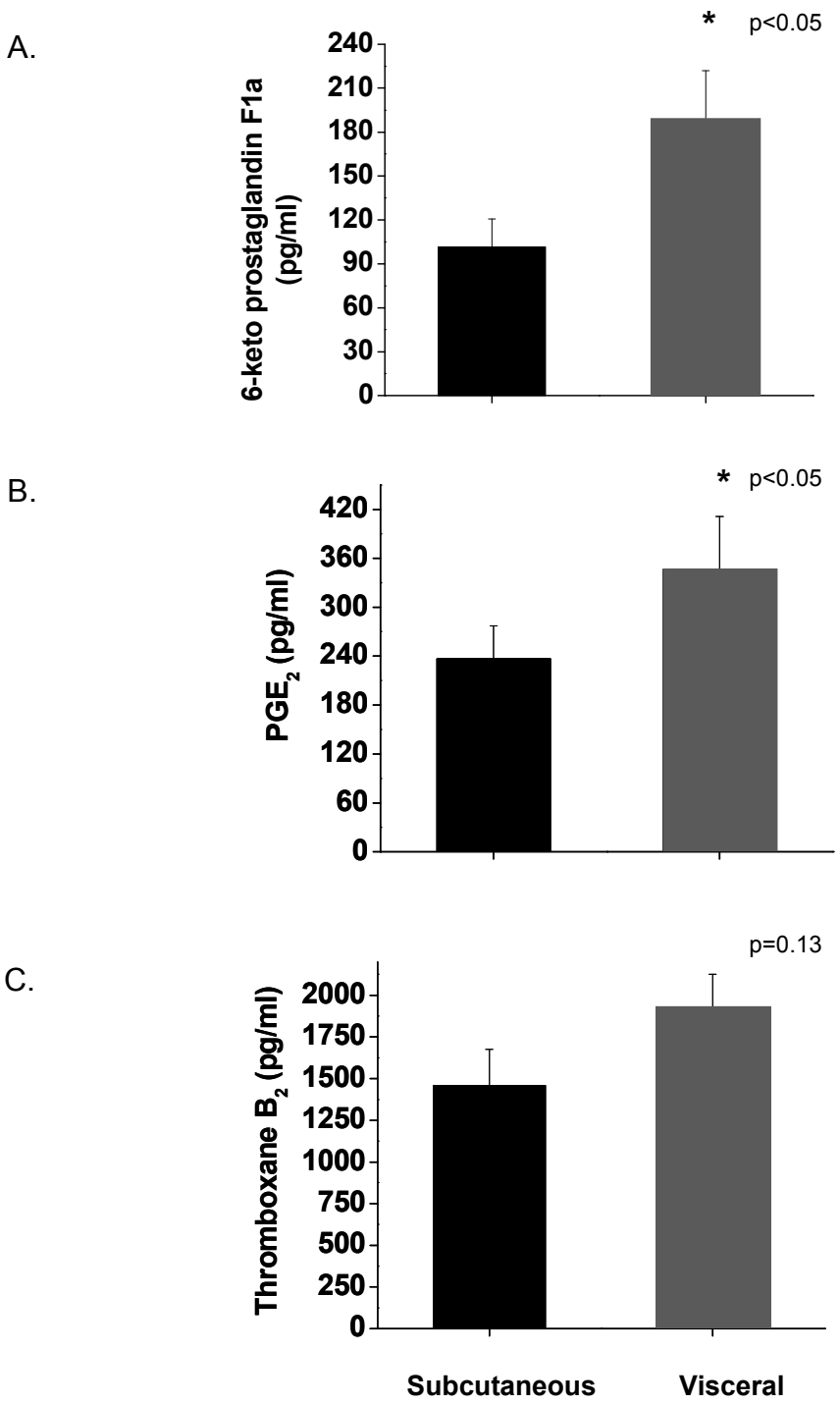
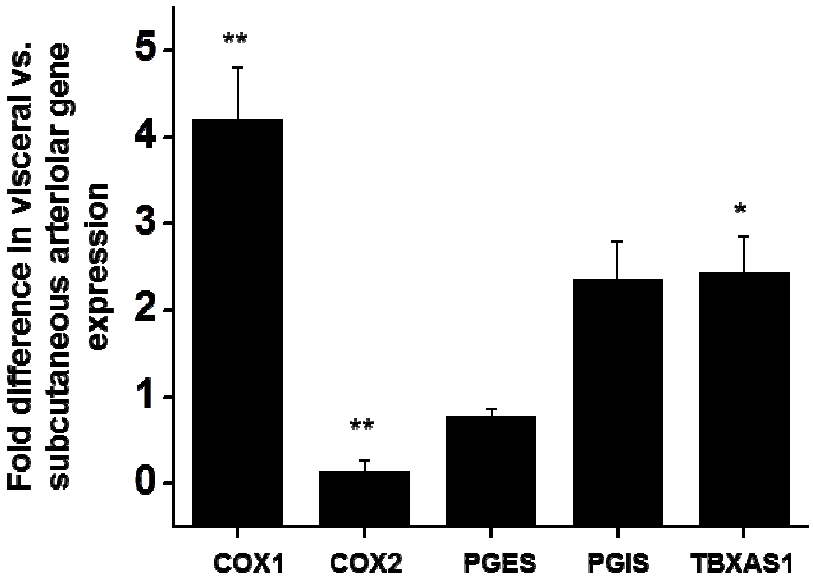
Supplemental Materials



Supplemental figure I: Ach-induced phosphorylation of eNOS at serine 1177 is impaired in endothelial cells isolated from visceral compared to subcutaneous depots. * $p<0.05$, n=7.



Supplemental figure II: 6-keto F alpha (A) production (metabolite of PGI_2) and PGE_2 (B) were significantly higher in the supernatant of cultured visceral compared to subcutaneous fat tissue (*$p<0.05$), and TBX_2 (C) also trended higher in visceral fat.



Supplemental figure III: Expression of cyclooxygenases and synthases relevant to prostanoid production were upregulated in visceral arterioles compared to subcutaneous vessels (n=7). COX1=Cyclooxygenase-1, COX2=Cyclooxygenase-2, PGES=Prostaglandin E synthase, PGIS=Prostaglandin I synthase, TBXAS=Thromboxane synthase. **$p<0.01$ and *$p<0.05$.