**Additional file 1**

**Figure 1S. Effect of GLP-1 attenuated TNF-α-induced calcium deposition in HASMCs.**

HASMCs were cultured in osteogenic differentiation medium treatment with TNF-α for 4 days in the presence or absence of GLP-1 for 1 day. Calcium deposition was induced dose dependently by TNF-α for 4 days.

**Figure 2S. The schematic of sitagliptin suppressed the initiation and progression of artery calcification via anti-oxidation and inflammatory inhibition.**

Circulating TNF-α and S100A/AGE triggered ROS production by binding to TNF receptor and RAGE, respectively. S100A/AGE bound to RAGE resulted in Nox-1 activation and superoxide increase. Oxidative damage-induced NF-κB activation promoted up-regulation of calcification-related proteins including RAGE, Msx2, Runx2, and BMP2. NAC and APO reduced oxidative stress via neutralization of ROS production and inhibition of p47 translocation, respectively. Treatment of the dipeptidyl peptidase-4 inhibitor, sitagliptin, impeded arterial calcification by suppressing Nox-1 activation in HASMC.