Background The development of atherosclerosis proceeds through multiple maladaptive pathways that begins with endothelial cell (EC) injury caused by oxidised low-density lipoprotein (ox-LDL) which plays a critical role. Proliferation and migration of vascular smooth muscle cells (VSMCs) also play an important role in atherosclerosis. Data from our lab and other studies show that Follistatin related protein (FRP), which is expressed in the vasculature, suggesting that FRP has cardio-protective effects on the development of atherosclerosis.

Objective In this study, we determined whether FRP protects against EC and VSMC apoptosis induced by ox-LDL.

Methods Human umbilical vein endothelial cells (HUVECs) and aortic VSMCs were isolated and treated with ox-LDL and recombinant FRP. We measured the activation of signaling molecules of Bcl-2, AKT and Bad by Western blots and detected the apoptosis cell by flow cytometer (FCM) in EC and VSMCs which were treated by ox-LDL or/and FRP in different concentration.

Results In vitro experiments showed that the effect of FRP on EC and VSMC apoptosis was different. The effect of FRP on EC apoptosis was mediated by upregulation of expression of the antiapoptosis protein Bcl-2. But the effect of FRP on VSMCs apoptosis was no prominent protection.

Conclusion FRP maintains EC viability by preventing apoptosis via Bcl-2 upregulation and it has no prominent effect in protecting VSMCs from apoptosis.