Chapter 5

Conclusion and Recommendation

Activated oxLDL through LOX-1 receptor mediated intracellular ROS and RNS generation is dose-degree dependent fashion. It is the first report studied in vasculature that composes of endothelium, smooth muscle cell, adventitial fibroblast and extracellular matrix represents the physiological outcome of those vascular cells. Endothelium remains intact functions in mildly degree and a small dose of oxLDL suggesting the redox homeostasis at vasculature. Endothelial damage and proliferation of smooth muscle cell are documented by light microscope. Intracellular ROS generation after oxLDL activation via LOX-1 receptor may release from vascular enzymatic source rather than transition metal driven ROS pathway. Once ROS is overwhelmed, it induces RNS by two mechanisms; eNOS uncoupling and transformation rate of peroxynitrite formation. Remarkably inducible LOX-1 expression and induced endothelial dysfunction are result from both ROS and RNS and might be positive feedback loop effect. Increasing p38 MAPK activity is one result of redox-sensitive protein and should be reduced by iron chelator and peroxyl scavenger pretreatment. In this step, it might be the way look insight in transcription factors involved which let us correct endothelial dysfunction and make this strategies approach the treatment in atherosclerosis.