Rationale

Various research groups have investigated onset and progression of atherosclerosis and owing to its complexity, an effective therapeutic intervention is not available till date. All the available treatment interventions including statins are designed to reduce the associated risk factors in order to decrease the risk and progression of atherosclerosis. Majority of research reports classically focus on foam cell formation; an event preceded by oxidative modification of LDL and subsequent immunologic events that help in progression of the disease. Recent reports have drawn the attention of scientific fraternity on Niemann–Pick C1-like protein 1 (NPC1L1) (Altmann et al., 2004), Proprotein convertase subtilisin/kexin type 9 (PCSK9) (Cohen et al., 2006), Myeloperoxidase (Nicholls & Hazel, 2009), cAMP-responsive element-binding protein H (CREBH) (Park et al., 2016), cannabinoid CB2 receptor (Zhao et al., 2010), etc. as the key genes regulating the sequence of events in atherogenesis. During the past three decades, a much of research has been carried out to understand the role of HSP60 in atherogenesis. Major proportion of the investigations explored the ectopic expression of HSP60 that correlates with expression of adhesion molecules in stressed endothelial cells and the relevant anti-HSP60 auto-immune reactions that play crucial role in atherogenic initiation. Based on the available data, strategies of using HSP60 as a biomarker for early detection of atherogenic lesions have been investigated. Also, HSP60 based vaccination protocols have been tested in animal models of atherosclerosis with promising results. The preliminary success in applicability of atherogenic insights in HSP60 and its importance in the early and reversible stage of atherogenic initiation calls for a profound investigation on the role of HSP60 in atherogenesis.