Hypothesis

The role of HSP60 in atherosclerosis has been limited to the initiation of auto-immune reactions that requires its expression on endothelial cell surface or secretion into the extracellular milieu. However, both surface expression and secretion of HSP60 are preceded by its intracellular upregulation in vascular cells. Hence, we hypothesized that HSP60 upregulation in vascular wall is a key event during atherogenic initiation irrespective of the initial physiological stress. We also believed that endogenous HSP60 has a regulatory function pertaining to the intracellular signaling pathways associated with atherogenic events in vascular cells.

Objective 1	*Assessing the status of HSP60 and HSP10 during stress induced atherogenic remodeling of thoracic aorta.*
Study 1	Status of HSP60 and HSP10 in diet induced atherogenic mice.
Study 2	Status of HSP60 and HSP10 in photoperiodic manipulation induced atherogenic changes in mice.

Objective 2	*Assessing the role of HSP60 in manifesting atherogenic transformation of endothelial cells.*
Study 1	OxLDL mediated atherogenic changes in endothelial cells and subsequent role of HSP60.
Study 2	Overexpression of HSP60 and assessment of endothelial dysfunction in absence of OxLDL.

Objective 3	*Investigating the role of HSP60 modulations in macrophage foam cell formation and polarization.*
Study 1	Impact of OxLDL on HSP60 of macrophages.
Study 2	OxLDL mediated atherogenic changes in HSP60 downregulated macrophages.