**MICRORNA REGULATION OF ENDOTHELIAL INFLAMMATION AND ATHEROSCLEROSIS**

**M. Feinberg**

Brigham and Women's Hospital, Boston, MA, and Harvard Medical School, Boston, MA, USA

Endothelial cell (EC) activation and vascular inflammation occur when the endothelium is exposed to various biochemical insults such as pro-inflammatory cytokines, oxidative stress, hypertension, hyperglycemia, aging, and biomechanical stimuli such as shear stress. These insults lead to the pathogenesis of a range of disease states, including atherosclerosis, insulin resistance, and obesity. Several signaling pathways, especially nuclear factor kappa-B mediated signaling, play crucial roles in these pathophysiological processes. Recently, microRNAs (miRNAs) have emerged as important regulators of EC function by fine-tuning gene expression. We show how a specific miRNA may regulate divergent targets in EC function and vascular inflammation in response to different pathophysiologic stimuli. Recent studies in mice and human subjects highlight an important role for miR-181b as a suppressor of endothelial inflammatory responses in both acute (e.g., sepsis) and chronic vascular disease states (e.g., atherosclerosis, insulin resistance, and obesity). These studies have uncovered emerging roles for novel miRNA targets in a cell-specific manner. An understanding of the role of miRNAs in EC activation and dysfunction may provide novel therapeutic opportunities for controlling a range inflammatory disease states.