

29 1030 AM

Summerlee Science Complex SSC 2315

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Emerging concepts in Noncoding RNA regulation of cardiovascular disease

MicroRNAs are small (~22 nucleotides) non-coding RNAs that function as "rheostats" to simultaneously tweak the expression of multiple genes within a genetic network, resulting in dramatic functional modulation of biological processes. The last decade has brought the identification of miRNAs, their targets and function(s) in



health and disease, including cardiometabolic diseases, there remains much to be deciphered from the human genome and its complexities in mechanistic regulation of entire genetic networks. Accumulating experimental evidence has revealed a key role for microRNAs in regulating cellular and molecular processes related to atherosclerosis development, ranging from risk factors, to plaque initiation and progression, up to atherosclerotic plaque rupture. We discovered that macrophages can promote proinflammatory and pro-atherogenic phenotypes in recipient cells through secretion of extracellular vesicles containing miRNAs and can inhibit macrophage migration *in vitro* and *in vivo*. Our studies suggest that EV-derived miRNAs secreted from atherogenic macrophages may accelerate the development of atherosclerosis lesions. We will discuss how microRNAs can influence atherosclerosis biology, as well as the potential clinical applications of microRNAs which are being developed as both targets and therapeutics for a growing industry hoping to harness the power of RNA-guided gene regulation to fight disease and infection.