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VASCULAR DISEASE

MIR-155 AND MIR-221/222 ATTENUATE ANGIOTENSION II INDUCED ENDOTHELIAL INFLAMMATION BY TARGETING ETS-1

ACC Poster Contributions
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Backgroud: Inflammation participants in all stages of atherosclerosis, the initial stage is characterized by the recruitment of leukocytes to the activated endothelial cells (ECs). MicroRNAs are a class of 19-25 nucleotides, non-protein-coding RNAs which repress target genes expression by translational inhibition or mRNA degradation. The link between miRNA and endothelial inflammation is largely unknown. The aim of this study was to determine whether ECs highly expressed miRNAs could regulate endothelial inflammatory response.

Methods and Results: Northern blot showed that mir-155 and mir-221 were highly expressed in human umbilical vein endothelial cells (HUVECs) and vascular smooth muscle cells (VSMCs). Bioinformatics analysis proposed Ets-1, a key transcription factor of endothelial inflammation and tube formation, as a candidate target for mir-155 and miR-221/222 cluster. The effect was demonstrated by luciferase reporter assay and western blot. Quantitative PCR showed that Ets-1 and several of its downstream genes include VCAM1, MCP1 and FLT1 were upregulated in angiotensin II (Ang II) stimulated HUVECs and this effect was partially reversed by overexpression of mir-155 or mir-221/222. Furthermore, cell adhesion assay revealed that overexpression of mir-155 or mir-221/222 effectively decreased Jurkat T cells adherence to Ang II stimulated HUVECs.

Conclusion: In summary, we report that HUVECs highly expressed mir-155 and mir-221/222 may target Ets-1 and indirectly regulate several ECs inflammatory molecules expression and decrease Jurkat T cells adherence to activated HUVECs. These findings may present possible therapeutic targets in atherosclerosis.