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ACUTE CORONARY SYNDROMES
SESSION TITLE: UTILIZING BIOMARKERS TO PREDICT THE FUTURE

Abstract 13103: Nourin-dependent *Mirna-106b*: A Novel Early Inflammatory Diagnostic Biomarker for Cardiac Injury

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Abstract

Introduction: Nourin is an inflammatory mediator rapidly released *by ischemic myocardium "before" necrosis,and by necrotic cells*. It stimulates leukocyte "*chemotaxis*" and "*activates*" leukocytes and vascular endothelial cells to release several "*cytokine storm*" mediators. Using Nourin amino acid sequence, Bioinformatics analysis indicated that Nourin is likely regulated by *miRNA-106b*; an inflammatory-signaling pathway linked to myocardial ischemia.

Hypothesis: As an "initial" inflammatory marker, the Nourin-dependent *miR-106b* can early diagnose ischemia-induced injury in UA patients when Troponin levels are below the decision limit, and in STEMI patients. The underlying regulatory mechanism *involves lncR-CTB89H12.4 and mRNA-ANAPC11;* associated with ischemia.

Methods: Gene expression of *lncR-CTB89H12.4* / *miR-106b* and *mRNA-ANAPC11* were measured in serum samples from UA (n=30 - confirmed by invasive coronary angiography and negative Troponin) and STEMI (n=16) patients at presentation, and healthy volunteers (n=16).

Results: Gene expression of *miR-106b* was up-regulated by 150-fold in UA compared to healthy, and by 4.6-fold in STEMI compared to UA (Fig. 1). Receiving Operator Characteristics (ROC) analysis revealed a statistically significant difference in *miR-106b* that discriminated UA from healthy controls with a test sensitivity of 87% sensitivity & specificity of 88%. Diagnostic sensitivity was 86% and specificity was 90% for discriminating UA from STEMI. Additionally, Spearman's correlation analysis revealed a significant association of *miR-106b* with *lncR-CTB89H12.4* and *mRNA-ANAPC11.* The down regulation of *lncR-CTB89H12.4* after ischemia resulted in the up-regulation of *miR-106b* and activation of *mRNA-ANAPC11*.

Conclusions: The Nourin-dependent miR-106b is a promising early inflammatory biomarker indicating UA patients and discriminating between UA and STEMI. Regulations appears to be from *lncR-CTB89H12.4* and *mRNA-ANAPC11*.

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Footnotes

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