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ACUTE CORONARY SYNDROMES
SESSION TITLE: NEW INSIGHTS INTO ACUTE CORONARY SYNDROMES AND PREDICTING RISKS

Abstract 13051: Nourin-dependent *Mirna-137*: A Novel Early Diagnostic Biomarker for Unstable Angina Patients

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Abstract

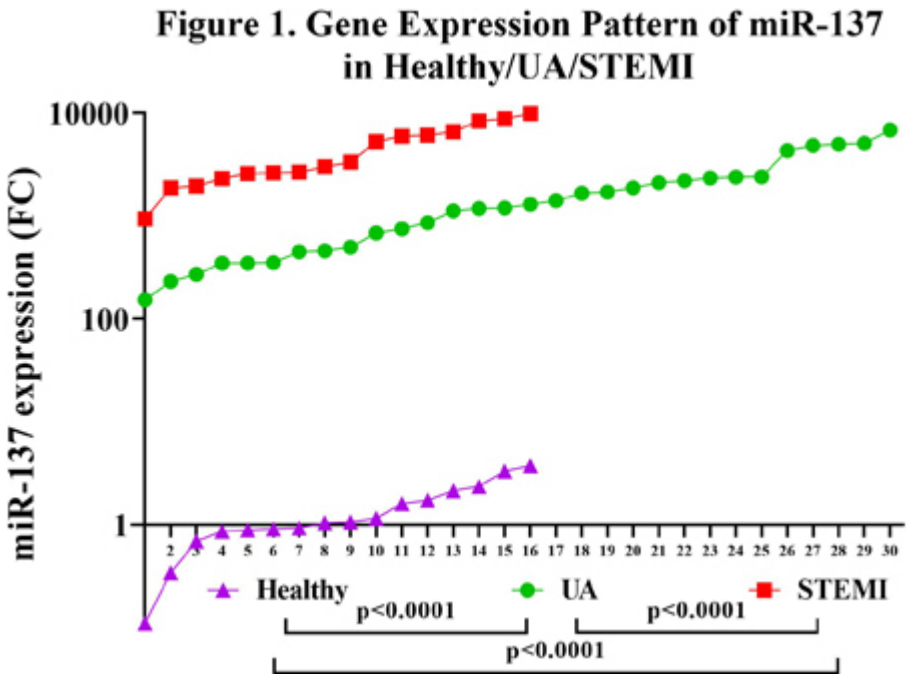
Introduction: No blood biomarker exits that can diagnose Unstable Angina (UA) patients. Nourin is ischemia-dependent inflammatory mediator rapidly released by reversible ischemic myocardium “before” necrosis, and by necrotic cells. Using Nourin amino acid sequence, Bioinformatics analysis indicated that Nourin is likely regulated by miR-137; a marker of cell damage and a hypoxiaresponsive autophagy-signaling pathway linked to myocardial ischemia and coronary artery disease.

Hypothesis: That the Nourin-dependent miR-137, is an early biomarker for UA patients when Troponin levels are below the decision limit. The underlying regulatory mechanism involves lncRCTB89H12.4 and mRNA-FTLH-17; also associated with ischemia.

Methods: We measured serum gene expression profile of lnc-CTB89H12.4/miR-137 and mRNAFTHL- 17 in 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-137 was up-regulated by 1,185-fold in UA (median=1,244.41) compared to healthy (1.05), and by 2.5-fold in STEMI (3,162.72) compared to UA (Fig. 1). Receiving Operator Characteristics (ROC) analysis revealed a statistically significant difference in miR-137 that discriminated UA from healthy controls with a test sensitivity & specificity of 96% & 93%, respectively. Diagnostic sensitivity was 75% & specificity was 83% for discriminating UA from STEMI. Additionally, Spearman’s correlation analysis revealed a significant association of miR-137 with lncRCTB89H12.4and mRNA-FTHL-17. The down-regulation of lncR-CTB89H12.4 after ischemia resulted in the up-regulation of miR-137 and activation of mRNA-FTLH-17.

Conclusions: As a marker of cell damage, the Nourin-dependent miR-137 is a promising early diagnostic biomarker indicating UA patients and discriminating between UA and STEMI. Regulations appears to be from lncR-CTB89H12.4 and mRNA-FTLH-17.



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Back to top

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