Basic Research / Discovery - Identification of novel miRNAs in various specific skeletal & muscle tissues and understanding their mechanism of action and regulatory roles.

Molecular Diagnostics / Biomarkers – Identification of specific miRNAs or miRNA expression based signatures that can act as biomarkers for various diseases/pathologies.

1. Biomarkers in body fluids: serum, plasma, exosomes, & HDL particles
2. Make accurate and detailed clinical diagnosis
3. Potential to determine prognosis and predict treatment efficacy

Drug Discovery / Therapeutics – Identification of miRNAs that play essential roles in disease to act as drugs or possible therapeutic targets.

1. miRNAs as vascular regulating drugs
2. miRNAs as drug target
3. Study of miRNAs response to infections, stress, other stimuli

Why Study miRNA in the Cardiovascular System?

1. Several miRNA genes are specifically expressed or highly enriched in skeletal and/or cardiac muscle, the so-called muscle miRNAs.
2. miRNAs are essential for proper muscle development and exert post-transcriptional control during myogenesis.
4. Circulating miRNAs may be novel biomarkers for coronary artery disease (CAD) and acute myocardial infarction (AMI).
5. Dysregulation of miRNAs can contribute to cardiovascular related diseases and disorders: CAD, AMI, arrhythmia, hypertrophy and fibrosis.
6. miRNAs are likely associated with other muscle-related diseases and are potential therapeutic targets.

Characterization and discovery of novel miRNAs and miRNAs in JAK2V617F mutated SET2 cells

Identification of a miRNA signature of renal ischemia reperfusion injury

Mycardial infarction leads to cardiac remodeling and development of heart failure. Insufficient myocardial capillary density after myocardial infarction has been identified as a critical event in this process.

Using miRseq, they show that the small molecule micromodulator (24-26) is enriched in cardiac endothelial cells and considerably suppressed after cardiac ischemia. M24 induces endothelial cell apoptosis, inhibits endothelial cell migration, and reduces fibrosis of endocardial tissues.

Overexpression of M24 or silencing of its target significantly improved angiogenesis in ischemic embryos. Blocking of endothelial mi24 limited myocardial infarct size via prevention of endothelial apoptosis and enhancement of angiogenesis, which led to preserved cardiac function and survival.

These findings indicate that the miRNA-24 acts as a critical regulator of endothelial cell apoptosis and angiogenesis and is suitable for therapeutic intervention in the setting of ischemic heart disease.