**INTRALIPID PROTECTS THE HEART AGAINST ISCHEMIA/REPERFUSION INJURY BY REDUCING CARDIOMYOCYTE APOPTOSIS VIA MIR122 INDUCTION IN LATE PREGNANCY**

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The prevalence of coronary artery disease during late pregnancy (LP) has increased over the past decade due to increased maternal age and significant changes in women’s lifestyle patterns (stress, smoking, diabetes and chronic hypertension). Clinically, myocardial infarction during LP carries a markedly worse prognosis than in non-pregnant women and is associated with significant maternal mortality. We have recently demonstrated that myocardial infarct size ~4 fold greater in LP rodent compared to non-pregnant controls. We also discovered that administration of intralipid at reperfusion resulted in ~60% reduction in infarct size of the heart in LP rat subjected to I/R injury, but the mechanism is not well understood. Here we hypothesized that intralipid protects the heart in LP by regulating the levels of specific microRNAs. The left anterior descending coronary artery was occluded in LP rats (21-22 days of pregnancy) for 45 min followed by 3 hr of reperfusion. One single bolus of PBS (control group) or 20% intralipid (intralipid group) was applied through the femoral vein 5 min before the reperfusion. The hearts of control and intralipid groups were used for microRNA microarray analysis (Ocean Ridge Biosciences). MicroRNA-microarray analysis identified miR122 as a novel micro-RNA which its expression was strikingly upregulated more than 10 fold in the heart of LP rats in intralipid group compared to control group. In cardiomyocytes subjected to hypoxia/reoxygenation injury, overexpression of miR122 resulted in reduced apoptosis, whereas knockdown of miR122 enhanced apoptosis. Our data show that the expression of Pyruvate kinase isoform M2 (PKM2) and capase-3 in the heats subjected to I/R was significantly lower in intralipid group compared to control group in LP suggesting PKM2 and caspase 3 could be two targets of miR122. In conclusion intralipid protects the heart in LP against I/R injury by reducing cardiomyocyte apoptosis via inducing miR122.