health care resources. Cardiovascular diseases are the leading cause of death in individuals with type 1 diabetes.

**Aim:** The purpose of this study was to evaluate the miRNA-133a expression in cardiac tissues of the diabetic rats and its relation to the cardiovascular complications.

**Methods:** 20 male albino rats divided into Group I (control) and group II (diabetic) 10 rats in each. Rats were made diabetic by intra-peritoneal injection of streptozotocin (35 mg/kg body weight). Physiological cardiovascular functions (heart rate, systolic blood pressure & ECG) were assessed. Blood and cardiac tissue samples were taken from all rats for biochemical and histological studies. Quantitative RT-PCR for miRNA-133 expression in cardiac tissues was performed. Results: Systolic blood pressure and QTc interval were increased in diabetic rats. miR-133 expression was significantly increased in cardiac tissues of diabetic rats compared to control rats. Moreover, its expression was positively correlated with fasting blood glucose levels.

**Conclusion:** The present study suggests that there is a complex relationship between miR-133 expression and the cardiac functions in diabetic rats which needs more exploration.

### Adipokines in obesity and metabolic disease

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The worldwide obesity pandemic is strongly believed to be the leading cause for the rising rates of metabolic diseases, including diabetes mellitus, cardiovascular disease, hypertension and non-alcoholic fatty liver disease. This is partly related to the accompanying state of chronic low-grade inflammation that contributes to systemic metabolic dysfunction, and that is associated with obesity-linked disorders. Collectively, this is termed metabolic inflammation.

Adipose tissue is considered a large endocrine organ that plays a central role in regulating dynamic crosstalk between tissues and organs. This role is mainly mediated through releasing multiple bioactive substances, known as adipokines. Some adipokines exert a pro-inflammatory while others have an anti-inflammatory activity. A detailed description of molecules that are differentially expressed upon changes in adipose tissue mass is expected to increase our understanding of the molecular mechanisms that underlie obesity and related metabolic co-morbidities.

In the current presentation, we will focus on the role of adipokines in regulating metabolic inflammation. We will present examples of adipokines, both classic and newly-characterized, that form the basis of the endocrine and immune-inflammatory functions of adipose tissue. In addition, researches of adipokines in clinical settings and their therapeutic potential will be discussed.

### Urinary exosomal microRNA panel unravels novel biomarkers for diagnosis of type 2 diabetic kidney disease

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**Background:** A potential approach adopted in the current study is to design a panel based on in silico retrieval of novel miRNAs related to diabetic kidney disease and to evaluate its usefulness in disease diagnosis.

**Patient and Methods:** In the current study, we measured the differential expression of a 6 miRNA panel in urine pellet and exosome in an initial screening group using syber green-based PCR array. Also, we performed pathway enrichment analysis of the key target genes of these miRNAs. Finally, we selected the most significantly up-regulated miRNAs in DKD, exosomal miR-15b, miR-34a and miR-636, that were measured by real-time PCR in a larger independent set of 180 participants to evaluate their usefulness as novel urine biomarkers for diagnosis diabetic kidney disease.

**Results:** PCR array analysis showed that miR-15b, miR-34a, and miR-636 were upregulated in both urine pellet and exosome of type 2DKD patients. qRT-PCR validation in the larger independent set of participants confirmed the significant up-regulation of these urinary exosomal miRs (P<0.001). Notably, a positive correlation was found between these miRs, serum creatinine and urinary protein creatinine ratio. The sensitivity of this miRs based panel in urine exosomes reached 100% in diagnosis of DKD.

**Conclusion:** We identified urinary exosomal miR-15b, miR-34a, and miR-636 as a novel diagnostic panel and a major contributor in the pathogenesis of diabetic kidney disease.

### Role of snake venom disintegrin like domain on the homing of mesenchymal stem cells and possible hepato-protective effect

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Louka.,(2001) purified a novel monomeric metalloproteinase / disintegrin like component from crude venom. EL-Asmar et al.,(2007), cloned the disintegrin domain from the metalloproteinase gene. The domain was sequenced. The sequenced domain has 70 percent identification prediction with many disintegrins domains from different snakes. Zaki et al., (2007) studied the effect of the purified metallocproteinase / disintegrin like fraction and demonstrated that liver expression of genes of TNF-α and HO-1 following injection (0.6mg/kg body wt.) of the fraction to mice treated with the hepatotoxic agent CCl₄. The fraction showed a degree of hepatoprotection. Results obtained from auto-protolysis were detected by SDS-PAGE giving three bands of molecular weights about 19 kDa , 15 kDa, 6 kDa. Products of proteolysis were re-fractionated by gel filtration chromatography on Sephadex G 50 column and giving fractions P₁, P₂, and P₃. The purified P₁ fraction had a platelet aggregation inhibitory activity. This purified P₁ fraction with a disintegrin activity had a molecular weight of 19 kDa. The purified P₁ fraction could be of value as hepatoprotective effect in white mice model treated with CCl₄. Abd EL-Wahab et al. (2014) showed that fraction P₁ was observed to enhance the recruitment & homing of intravenous injected bone marrow derived mesenchymal stem cells (BM-MSCs). Both could have a role in liver regeneration. It was noticed that BM-MSCs: labeled with the PKH26 showed a stronger red auto-fluorescence after trans-plantation into animals intoxicated with CCl₄ followed by combined Disintegrin/ like fraction (P₁) and BM- MSCs than animals intoxicated with CCl₄ followed by BM- derived stem cells alone. Darwish et al. (2017) confirmed the previous work and demonstrated the probable mechanism of stem cells homing by Disintegrin like domain.