The pathways leading to cardiac dysfunction induced by diabetes mellitus (DM) and its amendments histopathology are not yet fully understood [1-3], in part due to the complex nature and multifactorial DM [4]. Possibly the paper more important has been attributed to persistent hyperglycemia [5], the condition two which increases the risk for developing heart failure two to five times [6].

People with DM can develop heart dysfunction called diabetic cardiomyopathy [5]. This is characterized by dilation and hypertrophy of the myocardium, accompanied by systolic dysfunction and / or left ventricular diastolic and their presence is independent of ischemic heart disease or hypertension coexistence [7]. Although diabetic cardiomyopathy is a chronic subclinical condition [8], eventually leads to reduced elasticity of the matrix extracellular and decreased contractile function of the heart [9]. The association between development of diabetic cardiomyopathy and high glucose levels have significant effects on the expression, organization and components for modifying the extracellular matrix in the heart [4].

Studies confirm that adiponectin has an important role in glucose metabolism and lipid [10] in addition to its significant impact on the pathogenesis of DM, insulin resistance and vascular injury [11]. Adiponectin is a plasma protein secreted primarily by adipocytes and has properties antidiabetic, anti-inflammatory, anti-apoptotic, anti-atherogenic [12], immunomodulatory and cardiac protective [13]. Among other cells, cardiomyocytes are capable of synthetize adiponectin [14], autocrine function increasing its cardio protective effect [15]. Signalling pathways of the cardio protective effects of adiponectin are mediated primarily by increased protein kinase activated by adenosine monophosphate (AMPK), activated receptor peroxisome proliferator range (PPAR gamma), sphingosine 1-phosphate (S1P) and sphingosine kinase 1 (SphK1) [15].

There are three major isoforms of adiponectin: low adiponectin molecular weight (LMW), middle molecular weight (MMW) and high molecular weight (HMW) [16]. It is believed that the HMW adiponectin is isoform more active in peripheral tissues [14]. There is evidence that adiponectin protects the heart of ischemic lesions, cardiomyopathy and systolic dysfunction [17]. Furthermore, adiponectin is capable of inhibiting pathological hypertrophy Cardiomyocytes and myocardial fibrosis [14], to reduce the oxidative and nitrative stress [17], inhibiting TNF-α expression and IL-6, and enhance the expression IL-10 in the heart [14, 17].

To date, few studies examined the relationship factors that influence adiponectin levels children and adolescents with T1DM, and the results of these studies are inconsistent [18]. Recent studies show reduced expression cardiac adiponectin in rats with streptozotocin-induced diabetes [19]. The first study investigating the levels of cardiac adiponectin in growing rats with streptozotocin-induced DM was conducted by Da Silva et al. [20]. Animals with DM presented a marked reduction of total adiponectin and HMW adiponectin levels in the left ventricle. Corroborating with other studies on diabetic adult animals the authors revealed that the total and the HMW adiponectin levels were inversely associated with DM. Decreased cardiac adiponectin levels and increased inflammatory cytokines TNF-α and IL-6 was found in diabetic rats. However, further studies are required in order to conclude a causal association between reduced cardiac adiponectin with diabetic cardiomyopathy.

**References**


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