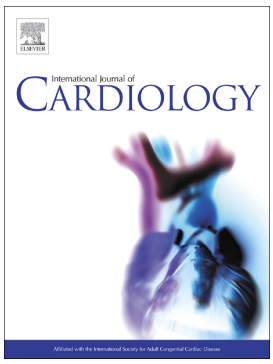

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Epigenetic regulation in diabetes-associated oxidative stress and myocardial dysfunction

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Diabetes is an important risk factor for ischemic heart disease and is associated with worse outcomes compared to non-diabetic patients following acute myocardial infarction (AMI). Specifically, the mortality following AMI was several times higher in diabetic patients compared to nondiabetics [1]. Emerging evidence indicates that hyperglycemia exerts long-lasting unfavorable effects on the cardiovascular system even after blood glucose normalization [2]. Short-term exposure to hyperglycemia may induce "Hyperglycemic memory" and cause long-lasting effects on myocardium [3]. Sarah Costantino et al. recently showed that oxidative stress and macrovascular complications persist even after 6 months of glycemic control and normalization of blood glucose levels in type 2 diabetic patients [4]. These data indicate that intensive glycemic control cannot rescue hyperglycemia-induced alterations and decrease the risk of heart failure in diabetic patients.

The pathogenetic mechanisms involved in diabetes-induced cardiovascular events are multifactorial and there is no clear scientific consensus of the factors underlying this deficit. One hypothesis proposes that the excessive production of reactive oxygen species (ROS) leads to inflammation, fibrosis and eventually cardiac dysfunction [5]. Alternatively, ROS might activate the cell survival pathway when the heart is exposed to preconditioning stimuli and exhibit beneficial effects [6]. Although the underlying mechanisms remain unclear, mitochondria-derived ROS, the primary source of ROS in cardiac myocytes, may act as a double-edged sword. The effects of ROS can be good or bad, which depends on multiple factors such as the quantity of ROS, the subcellular location of ROS, and the different types of oxidant [6].

Recent studies showed that epigenetic mechanisms may affect redox signaling through the actions of microRNAs (miRNAs), long noncoding RNAs, as well as DNA methylation and posttranslational modifications—perhaps contributing to cardiovascular