Globally, CVD are the leading cause of death. According to the World Health Organization, in 2008, 7.3 million of deaths were due to coronary heart disease and it is projected that by 2030 almost 23.6 million people will die from CVD. Even though clinical care has been upgraded, public awareness has been increased and health innovations are extensively used, MI still remains the foremost cause of death worldwide. Many synthetic drugs are used for the treatment of MI. However, they cannot meet the demands due to multiple etiological factors of MI and their certain side effects. Thus, a lot of studies focused on identifying new therapeutic strategies to prevent or reverse cardiac failure. Alternative therapies using phyto-nutrients are becoming increasingly popular, as these nutrients have no or less side effects and are cost effective. Epidemiological evidences reveal that diets rich in fruits and vegetables promote health and reduce or delay the onset of MI.

Therapeutic intervention along with antioxidants may be beneficial in preventing MI. Presently, there is much interest in flavonoids as their intake appears to be linked with reduced risk of certain diseases such as cancer, diabetes mellitus and CVD because of their potent antioxidant action. Diosmin (diosmetin 7-O-rutinoside), a natural flavone glycoside readily obtained by dehydrogenation of the corresponding flavanone glycoside hesperidin, is abundant in the pericarp of various citrus. Literature survey has revealed that there is no scientific report available on the effects of diosmin on MI. Therefore, we made an attempt to evaluate the preventive effects of diosmin in ISO-induced MI in the male Wistar rats. In this study, the following biochemical, electrocardiographic, histological, Transmission Electron Microscopic, *in vitro*, and docking studies were carried out.
Scope and Objectives

i. Cardiac diagnostic markers such as serum creatine kinase-MB, serum creatine kinase, serum lactate dehydrogenase, serum cardiac troponins-T and I, serum myoglobin and inflammatory markers such as serum high sensitive C-reactive protein and serum homocysteine.

ii. Electrocardiographic pattern.

iii. Lipid peroxidation products such as thiobarbituric acid reactive substances, lipid hydroperoxides and antioxidant system such as superoxide dismutase, glutathione peroxidase, glutathione-S-transferase, catalase in the heart and reduced glutathione, in the plasma and heart.

iv. Histopathology of heart.

v. Total cholesterol, triglycerides and free fatty acids in the serum and heart and serum lipoproteins such as low density lipoprotein-cholesterol, very low density lipoprotein-cholesterol, high density lipoprotein-cholesterol and rate limiting enzyme of cholesterol synthesis, 3-hydroxy-3-methyl glutaryl coenzyme-A reductase in the liver.

vi. Membrane bound-adenosine triphosphatases and electrolytes in the heart.

vii. Lipid peroxidation products, antioxidant system, malate dehydrogenase, reduced nicotinamide adenine dinucleotide dehydrogenase, isocitrate dehydrogenase, α-ketoglutarate dehydrogenase, cytochrome-C-oxidase and Ca$^{2+}$ in the mitochondrial fraction of the heart.

viii. Transmission electron microscopic study on the heart mitochondrial structure
ix. The *in vitro* studies

1,1-diphenyl-2-picrylhydrazyl radical, superoxide radical, hydroxyl radical scavenging and reducing activity of diosmin *in vitro*.

x. Docking studies