INTRODUCTION
Diabetes Mellitus is a metabolic disorder characterized by varying or persistent hyperglycemia and either insufficient or ineffective insulin [1]. It is a chronic disease without a cure; however, with proper management and treatment, diabetics can live normal and healthy lives. Further, diabetes mellitus has been inveterated as a serious global problem and reached at epidemic level. It is more severe in India than other countries in the world. Moreover, diabetes is the seventh leading cause of death in India. The worse condition of diabetics in India in comparison to other countries might be because of negligence attitude of most of the Indian people in diet and health issues. However, the more deliberated countries in the health related matters are not absolutely free from the disease. For example, America, despite being highly cognizant in healthcare, has about 6% diabetics (2,3).

Diabetes Mellitus comprises a group of common metabolic disorders that shares the phenotype of hyperglycemia, dyslipidemia and hyperlipidemia, a consequence of disorder in carbohydrate and lipid metabolism. It usually leads to a series of late complications, include coronary heart disease, stroke, peripheral vascular disease, blindness, kidney disease, plasma lipid oxidation, amputation of limbs, impotence, vascular complications caused by endothelial cell dysfunction. It can also lead to pregnancy-related complications, both for the mother and the foetus or new-born baby. According to American Diabetic Association (ADA) in 1997 and on accepted classification by WHO, [4,5,6] Diabetes Mellitus are classified as: (1) TYPE I DM (2) TYPE II DM (3) Gestational diabetes mellitus (GDM) (4) Special diabetes types. In which Type 2 diabetes is the most common form of diabetes, accounting for up to 95% of all diabetes cases. Diabetes is the cause of about 3% of premature mortality most often because of ischaemic heart disease, stroke and renal disease. Type 1 diabetes (insulin-dependent/juvenile onset) comprises about 12% of all cases of diabetes and Type 2 diabetes (non-insulin-dependent/later onset) about 88% of cases. Gestational diabetes occurs during pregnancy in about 4-6% of women not previously known to have diabetes, while other forms of diabetes are less common.
Type 2 diabetes is the most common metabolic disease worldwide, with a prevalence estimated to rise from 171 million in 2000 to 366 million in 2030 [2]. Although the cause of type 2 diabetes appears to be multifactorial, it has been firmly established that the diet can play a major role in the incidence and progression of the disease [3]. Diabetes is projected to increase at a faster rate in men than in women. It is estimated that the disease burden might be doubled from 3% to 6% in men and it will probably increase from 3% to 4% in women (from 1996 to 2016).

Ischemic heart disease may also lead to "sudden" cardiac death—the cause of death for some 250,000 U.S. adults each year. An estimated 14 million people in the United States have ischemic heart disease. Of these, as many as 4 million have few or no symptoms and are unaware that they are at risk for angina (angina pectoris), heart attack (myocardial infarction), or sudden death. Several factors, such as high blood cholesterol levels, hypertension, cigarette smoking, and diabetes, are chief culprits in the promotion of heart.

The cause of Diabetes Mellitus and Ischemic heart disease is not fully understood. Recently, increasing evidences suggest that free radical formation is involved in the pathogenesis and the development of diabetic complications and Ischemic heart disease [7,8,9]. The American Heart Association has identified several risk factors. A person with a total cholesterol of 300 mg/dl has a greater risk than someone with a total cholesterol of 245 mg/dL, even though an individual with a total cholesterol greater than 240 is considered at high-risk.

Diabetes initiates atherosclerotic lesions without involving inflammatory cells. First, diabetes-associated hyper- and dyslipidemia are expected to accelerate LDL deposition in the arterial wall, while hyperglycemia promotes the formation of the highly reducing Amadori products in both LDL and collagen. Hyperglycemia also leads to the conversion of methylglyoxal to carboxyethyl-lysine (CEL) [10, 11]. All these processes occur non-oxidatively. Oxidation of polyunsaturated fatty acids in LDL is mediated by high glucose-driven superoxide formation by mitochondria and NADH oxidase [12, 13], will yield glyoxal, a potent precursor of N-carboxymethyl-lysine (CML) [13]. Indeed, CML has been detected immunochemically in early atheromatous lesions [14]. Evidence
for the presence of CEL in such lesions is still pending, but it is expected to be based on findings of elevated CEL in diabetic tissues [14]. Macrophages of the arterial wall, potently oxidizes LDL to generate the same oxidative modifications found in LDL isolated from atheromatous plaques. Both Cu2+-mediated and myeloperoxidase-mediated oxidation lead to an increase in O-tyrosine and M-tyrosine, but only the latter selectively generates dityrosine from tyrosine radical which selectively increased in fatty streaks and intermediate atheromatous lesions [13 to 18].

Amadori products and ceruloplasmin are expected to be potent precursors of oxidative damage and can oxidize lipoproteins [14]. Hyperglycemia-catalyzed superoxide formation from mitochondrial and cytoplasmic sources are expected to initiate the lipoxidation cascade and release of glyoxal, a potent CML precursor polyunsaturated fatty acid (PUFA). The strong relationship observed between glycated hemoglobin and hydroxyl radical damage suggests a concomitant process in which CML originates from Amadori products through hydroxyl radical-mediated oxidation [17]. Proteins rich in CML (18) and methylglyoxal-treated proteins have been found to bind redox-active Cu2+, providing a possible mechanism for the protein damage [19].

According to the California Dried Plum Board, Sacramento, the phenolic compounds found in fruits, spices and vegetables not only influence sensory properties, such as color, taste (e.g., bitterness) and flavor (i.e., aroma qualities), but also comprise much of the antioxidant capacity associated with these foods.

Medicinal plants have a lot of types of antioxidants, mostly polyphenols, flavonoids which exhibit high antioxidant activity. Intake of antioxidants present in food is an important health-protecting factor. These various herbal compounds known by ancient medicine are of growing interest in the domain of prevention of diseases. Therefore, oxidative stress can be reduced with the provision of additional antioxidants of these nature. Antioxidants are closely related with the prevention of degenerative illness, such as cardiovascular, neurological diseases, cancer and oxidative stress dys-functions [20 to 23].
A number of spices and herbs have a long history of traditional use in treating elevated blood sugar levels and cholesterol levels as well.(24). One such compound that has recently been the subject of intense research is cinnamon, a compound granted GRAS (Generally Recognized As Safe) status by the United States Food and Drug Administration. The beneficial effects of cinnamon on glucose control appear to be in part due to doubly-linked polyphenol type-A compounds(25). Cinnamon bark has been used for several thousand years in traditional Eastern and Western systems of medicine, for such purposes as anorexia, bloating, dyspepsia with nausea, flatulent colic, and spastic conditions of the gastrointestinal (GI) tract. (26) Over the past two decades, in vitro and in vivo data have been accumulating which support the role of cinnamon on glycemic control. For example, Jarvill-Taylor et al.(27) reported that cinnamon stimulated glucose uptake, glycogen synthesis, and activated glycogen synthase in 3T3-L1 adipocytes.

Therefore, the present study is an attempt to assess the antioxidative potential of C. Zeylanicum on oxidative stress which may be responsible for alteration and complications in carbohydrate and lipid metabolism and possibly depressed antioxidant defense system in NIDDM and Ischaemic heart disease.