Introduction
Ageing is defined as a sum of functional and structural changes accumulating in cells and tissues of an organism during life. These changes are associated with or responsible for the decreasing physiological performance of an organism and increasing susceptibility to disease and death (Harman. 1984). The cause of ageing may probably reside in the genetic material with which we are endowed from the moment of conception. The manifestation of ageing, however are unlikely to be present during the period of growth and development but slowly declare themselves during the period of maturity. Thus, ageing is the origin and cause of inevitable injury of cells and final death of the organism under the influence of possible external or internal deleterious factors (Medvedev, 1980).

Longevity, expressed in terms of life expectancy or life span is a continual process of change that progressively erodes tissue functions throughout the rest of life. Advancing age is thus accompanied by a number of degenerative cellular processes, including absence of self renewal, replacement and regeneration of cells that may lead to various pathological conditions such as immune dysfunction, brain dysfunction, cataract, cardiovascular disease, diabetes mellitus and cancer (Christen, 2000; Ames et al., 1993). Hence, geriatrics care for aged, has recently drawn attention of many researchers to unravel the underlying mystique.
THEORIES OF AGEING

Modern science has made tremendous attempts to explain the phenomenon of the ageing process. There are more than 300 theories of ageing put forward, many of which co-exist because they do not contradict each other or because they try to explain different and indifferent forms of senescence (Medvedevs 1980). An important group of ageing theories originates from the study of changes throughout life or changes which accumulate with time.

Most current theories of ageing maintain that age-related deterioration of physiological functions is primarily caused by qualitative and quantitative modifications of cellular constituents. The theories of ageing encompass genetic theory, defective protein synthesis theory, somatic mutation theory, immunological theory, free radical theory, macromolecular cross linking theory, endocrine theory. At present, the most popular and widely accepted ageing theory is the Free Radical Theory of Ageing, first proposed by Harman (1956) and forms the basis for the present work.

FREE RADICAL THEORY OF AGEING

Ageing though is likely to be a multifactorial process. There is now significant evidence implicating the generation of reactive oxygen species and the corresponding response to oxidative stress as key factors in determining longevity. Living in an oxygenated environment has required the evolution of effective cellular strategies to detect and detoxify metabolites of molecular
oxygen known as ‘Reactive Oxygen Species’ (ROS). Therefore, the appropriate and inappropriate production of oxidants, together with the ability of organisms to respond to oxidative stress is intricately connected to ageing and life span.

Free radicals are generated in all living beings and are highly reactive and short-lived. A free radical can be defined as any species with one or more unpaired electrons occupying an outer orbital and capable of independent existence. In general, they are very reactive due to the fact that they are not in a stable spin state. Once formed they are capable of propagating the free radical formation continuously. Free radical includes superoxide (O$_2^-$), hydroxyl radical (OH$^*$), peroxyl radical (OOH$^*$), thyl radical (RS$^*$), trichloromethyl (CCl$_3^*$) and nitric oxide (NO$^*$). The interaction of the free radicals with a number of cellular macromolecular targets initiates the eventual death of the cell. This theory thus, focuses on the senescence associated free radical damages at both the cellular and sub-cellular levels (Harman, 1981; Nohl, 1993; Sohal and Dubey, 1994).

**OXIDATIVE STRESS AND AGEING**

The imbalance between cellular production of ROS and the ability of cells to defend themselves against the free radicals is referred to as Oxidative stress. At the cellular level, the ROS inclination due to oxidative stress damages vital cell components like polyunsaturated fatty acids, protein and nucleic acids (Halliwell, 2001). This further enhances the development of one or better-recognized age related modifications, such as alteration of intrinsic
membrane properties (fluidity, ion transport, loss of enzyme activity, protein crosslinking), inhibition of protein synthesis and DNA damages (Bandyopadhyay et al., 1999) that ultimately results in cell death. Age associated oxidative damages may overwhelm the natural repair system in the organism (Kowald, 1999) leading to the onset of age-related diseases with a progressive increase in the chance of morbidity and mortality.

**ERYTHROCYTES**

Erythrocytes are well characterized with biconcave shape, which develops as immature multiglobular reticulocyte that mature first into a discoidal reticulocyte and then into a mature RBC over a span of two or three days initially in the bone marrow and then into the circulation (Goodman et al., 1988). Erythrocytes have a mean diameter of 7.2 μm and thickness of about 2.2 μm, near the circumference and about 1 μm at the centre. The average surface area of an erythrocyte is 120 μm² and the volume is 85 μm³ (Harris, 1970). They are equipped with a flexible and complex outer structure, which permits the cells to pass through the capillaries whose diameter is significantly smaller than the erythrocytes. They undergo a sequence of shape changes and the cell membrane moves around the deformed cell in a ‘tank-treading fashion’ (Sutera et al., 1983; Tran-Son-Tay et al., 1984). This deformability is due to the existence of a fine mesh work of cytoskeleton made up of several proteins and linked to the phospholipid membrane bilayer through integral proteins (Bennett, 1990 and Mohandas and Evans, 1994).
OXIDATIVE DAMAGES – A TOOL TO ACCELERATE ERYTHROCYTE AGEING

Erythrocyte successfully cope with a number of dangers, such as passage across narrow capillaries and spleenic slits, periodic high turbulence and hight shear stress and extremely hypertonic conditions (Arese et al., 2005). The erythrocyte is an ideal model for studies related to ageing (Bartosz, 1988) as it lack transcriptional and translational machinery, which cannot yield direct information on the DNA control of cellular ageing, and do allow the aged modifications of intracellular proteins and plasma membrane to continue without the interference of the repair processes and exchange of damaged macromolecules (Bartosz, 1988).

As carriers of oxygen, RBCs become susceptible to oxidative damage during their life span in circulation and senescent erythrocytes naturally reveal some of the signs due to constant insults by oxygen free radicals (Rice-Evans and Baysal, 1987). Internally, during oxidative stress, oxygen is released as superoxide radical anion upon hemoglobin oxidation to methemoglobin (Chiu and Lubin, 1989). The superoxide radical is not harmful as such but upon decomposition it forms H₂O₂ and the extremely reactive hydroxyl radical capable of damaging proteins and initiating lipid peroxidation (Chiu et al., 1989). Externally, granulocytes, macrophages and other metabolically active cells, generate free radicals that can diffuse into the extracellular environment and potentially interact with erythrocytes (Weiss, 1980). The cell membrane is a structural barrier that plays an essential role in protecting
cellular integrity. Changes in the macromolecules of membrane are one of the earliest signs of erythrocytes membrane alterations during ageing (Spiteller, 2002). ROS generated in the aqueous or lipid phase can attach to the erythrocyte membrane and can induce oxidation of lipids, proteins and carbohydrates, triggering disruption in membrane (Stadtman, 1992; Tappel, 1973).

Oxidative modifications including covalent aggregation, proteolytic degradation, and glycation and carboxyl methylation serves as a ‘molecular clock’ in terminating the erythrocyte life and enabling the selective recognition of damaged erythrocytes by macrophages (Bartosz, 1988). Enhanced protein degradation has shown to play a role in the events leading to the shortened life of the erythrocyte in old individuals (Glass and Gershon, 1984; Danon and Marikovsky, 1988). Modifications in senescent erythrocytes lead to an appearance of senescent neoantigen on the cell surface (Kay, 1993), which serves as a specific signal for the clearance of these cells by inducing binding of IgG-autoimmune antibody and subsequent phagocytosis (Glass et al., 1983). Studies indicated that erythrocytes from the old individuals have more membrane-bound IgG indicating an enhanced sensitivity to recognition by the reticuloendothelial system that would result in premature removal of the erythrocytes from circulation (Shperling and Danon, 1990). Further, in elderly, erythrocytes released from the bone marrow into the circulation produced immature erythrocytes that had diminishing functions and shortened life span in circulation (Gershon and Gershon, 1988).
Cell density measurements have shown that aged animals have more circulating erythrocytes in the lighter fractions and less in the denser fraction when compared with young animals (Glass and Gershon, 1984). The shift of erythrocytes to lighter fractions is consistent with shortened lifespan with advancement of age. A comparative study of erythrocytes of old and young animals was performed by measuring the ratio of loss of labeled cells from the circulation (Vomel, 1981). The loss of radioactivity following the injection of $^{59}$Fe was found to be significantly faster in aged rat (nearly 45 days) than in young rats (nearly 60 days) indicating an early senescence of RBC (Glass and Gershon, 1984). On the whole these investigations lead to the fact that animal age influenced the endurance of erythrocytes in passage.

**OXIDATIVE DAMAGE TO ERYTHROCYTE MEMBRANE LIPIDS**

Erythrocytes present many special properties, which are attributable to proteins and lipids associated with the flexible membrane surrounding the cell (Bennett, 1990) (Figure A). The structural organization of the membrane is thus, a primary determinant of the remarkable mechanical resiliency of the red blood cell membrane. The major lipid components are free cholesterol (~16%) and phospholipids (~20%) present in nearly equimolar quantities, while free fatty acid and glycolipids (~4%) present in lesser amounts. The phospholipid composition of the membrane consist of ~28% phosphatidylcholine (PC), ~25% sphingomyelin (SM), ~26% phosphatidyl ethanolamine (PE), 13% phosphatidylserine (PS) and phosphatidyl inositol 4,5 bisphosphate constituting about 1% (Zwall and Schroit, 1997). The phospholipids are asymmetrically distributed in the membrane, with choline containing neutral
phospholipids PC and SM are found on the outer monolayer of lipid bilayer while PC and PS (aminophospholipids) the negatively charged phospholipids are localized in the inner layer. The fatty acyl chain of PC and PS are more unsaturated than that of PC and SM making the inner monolayer of plasma membrane more fluid than the outer monolayer (Zachowski 1986)

**LIPID PEROXIDATION (LPO)**

Oxidation of lipids is characteristically a free radical chain reaction initiated by the abstraction of a hydrogen atom within the cells due to high concentration of PUFA in the RBC membrane. The oxidative destruction of PUFA known as Lipid Peroxidation is particularly damaging because it proceeds as a self perpetuating chain reaction (Lappel 1973 Pryor 1978) Oxidation of PUFA generates a fatty acyl radical (I·) that rapidly adds oxygen to form a lipid peroxyl radical (I OO·) These radicals are the carriers of the chain reactions and can react with further molecules of PUFA producing lipid hydroperoxides (I OOH) that contain at least three methylene interrupted double bonds and can lead to the formation of an aldehyde (Malondialdehyde) as breakdown product (F-sterbauer et al 1990) The presence of transition metals capable of serving as redox agents, hemoglobin and other heme containing proteins can also augment lipid peroxidation (Pryor, 1978)

High concentrations of lipid peroxidation products including malonaldehyde (MDA) is highly reactive bifunctional molecule has been shown to cross link erythrocyte phospholipids and proteins to impair a variety of the membrane related functions and ultimately to diminished RBC survival
Metabolism of MDA to lesser reactive molecule is therefore a prerequisite to ensure the survival and normal functioning of erythrocytes (Bekyarova et al., 1996). Consequently, such alterations in lipids have been suggested to be the cause of ageing and age-associated degenerations (Stringer et al., 1989).

MEMBRANE FLUIDITY

The dynamic asymmetric nature of biological membrane is characterized by thermodynamically stable fluid superstructure (Vereb et al., 2000). Membrane fluidity is a physiochemical factor of biomembranes and has been shown to interfere with various membrane functions (Choi and Yu, 1995). Studies have shown membrane lipid alterations as an important factor in decreasing lipid fluidity during ageing in erythrocytes resulting in erythrocytes membrane rigidity (Stocks and Dormandy, 1971; Yu et al., 1992). The rigidity of membrane seems to be an important factor in reducing cell deformability, a phenomenon due to enhanced intracellular viscosity (Borst et al., 2000). Alterations in membrane fluidity during ageing thereby lead to disturbance in blood rheological behaviour contributing to various pathophysiological implications including cardiovascular diseases, hypertension and stroke (Szapary et al., 2004). Peroxidative damage of the erythrocyte membrane could lead to progressive echinocyte transformation and membrane rigidity with age (Lubin and Chiu, 1982).
Figure B. Cytoskeletal protein of erythrocyte membrane
maintain the membrane structure and membrane junction (Rybicki et al., 1988).

*Spectrin* is the major protein constituent of the membrane skeleton accounting for about 25% of the total erythrocyte membrane protein and about 75% of the cytoskeletal mass. It is composed of alpha and beta intertwined chains side-to-side to form a heterodimer (Ji et al., 1980). Spectrin modification during ageing is a signal for premature clearance of RBC from circulation (Giuliani et al., 2000). Age-associated alterations in ATP levels in erythrocytes have direct profound effect on the network of the spectrin-actin junction that may lead to membrane destability (Marikovsky, 1996).

*Ankyrin*, a 200-kDa protein, is largely responsible for maintaining the close association of membrane skeletal proteins, the spectrin and band 3 with the lipid bilayer (Bennett, 1990). This serves as an anchor for the attachment of the skeletal matrix to the membrane. Ankyrin is extremely sensitive to proteolysis and causes modifications that affect the deformability of erythrocytes (Suzuki et al., 1988). Oxidation of the spectrin ankyrin-binding site would be expected to lead to abnormally shaped erythrocytes with the unstable membrane (Zail and Coetzee, 1984).

*Actin*, a 42 kDa protein together with protein 4.1 plays a key role in the junctional sites of the spectrin network, in the erythrocyte membrane skeleton (Gardner, 1989). Several reports have documented that upon oxidation RBC membrane stability is decreased by damaging band 4.1 protein and forming a
defective spectrin - band 4.1 - actin tertiary complex (Advani et al., 1992). Oxidative damage to band 4.1 proteins would also lead to an abnormal shape of erythrocytes and destability of membrane phospholipid asymmetry (Franck et al., 1988).

*Band 3 protein* also called anion exchanger, carries out passive exchange of HCO₃⁻ for chlorine ions and is thought to facilitate CO₂ removal and to increase the total CO₂ capacity of blood. Moreover, it carries a antigenic determinant important for blood groups (Victoria et al., 1981). In addition it may also play a role in RBC shape since it has been shown to bind to spectrin via ankyrin, the internal scaffold and thereby helps to maintain erythrocyte shape and stability (Nakao, 1990). Oxidative modifications also lead to ‘senescent cell antigen’, which was proposed as a major apoptotic signal for the elimination of erythrocytes from circulation by macrophages (Kay, 1993).

Interspaced within the bilayer are numerous *membrane proteins* with various functions as transporters, receptors, and enzymes. Substantial evidence indicates that the active center of the ATPase is localized inside the red cell membrane. The membrane ATPase activity in intact erythrocytes decreases following peroxidative damage (Marchesi and Palade, 1967). The loss of ATPase activity might well be related to the loss of PS following peroxidant injury. Furthermore, the carbonyl-containing substances derived from peroxidised phospholipids are potent inhibitors of erythrocyte membrane ATPase and it is also reported that the activity of acetylcholine esterase,
which is located exclusively on the exterior of the red cell membrane, decreases after peroxidant injury (Kesner et al., 1979).

**OXIDATIVE DAMAGE TO ERYTHROCYTE MEMBRANE CARBOHYDRATES**

Carbohydrates in erythrocytes have many important functions that are necessary for the proper functioning of the cell to survival. Carbohydrate is thought to be highly necessary for cell surface charges and have a crucial role in the clearance of senescent cells (Jovtchev et al., 2000). Glycoproteins are carbohydrate-containing proteins anchored in the lipid bilayer constituting approximately 2% of total erythrocyte membrane proteins (Eylar et al., 1962). Sialic acid is an important glycoprotein by virtue of both its location at the terminal position on glycans due to its net negative charge (Aminoff et al., 1988). Further numerous studies have demonstrated that cell-surface carbohydrates serve directly as molecular determinants responsible for mediating molecular and cellular interactions (Varki, 1992). Oxidative changes in the glycomoieties of erythrocyte glycoprotein affect the surface charge including aggregation and binding of erythrocytes to macrophages (Ando et al., 1995).

Age-related reduction of sialic acid on the cell surface associated with oxidative stress has been shown to be crucial for erythrocyte survival (Aminoff et al., 1988; Sangeetha et al., 2005). Alterations in carbohydrates with increase in erythrocyte aggregation were also found to be associated with cardiovascular risk factors such as hypertension, hyperlipoproteinemia (Vaya
et al., 2004) and myocardial ischemia (Chien et al., 1979). Moreover, it has been proposed that reduction in sialic acid level can expose cryptic α-galactosyl, which is recognized by lectin-like receptors on macrophages for phagocytes (Schlepper-Schafer et al., 1983).

**ERYTHROCYTES AND APOPTOSIS - ERYPTOSIS**

Apoptosis or cell death is a process in which a cell participates in its own termination by way of a cascade of molecular interactions. Erythrocytes may not undergo true apoptosis as they lack a nucleus, but it is attractive to suppose that evolution has seized on the same mechanisms for signalling removal of erythrocytes from the circulation. Suicidal death of erythrocytes (eryptosis) is triggered by erythrocyte injury after several stressors, including oxidative stress (Fabisiak, 1998). Eryptosis is characterized by cell shrinkage, membrane blebbing, activation of proteases, and phosphatidylserine exposure (PS) at the outer membrane leaflet (Buttke and Sandstrom, 1994).

**PHOSPHATIDYLSERINE EXPOSURE**

Erythrocyte membrane displays a characteristic asymmetry in the transbilayer distribution of its phospholipids. The aminophospholipids are mainly confined to the inner leaflet of the bilayer and choline phospholipids accounts for most of the outer leaflet lipids (Op den Kamp, 1979; Van Deenen, 1981). This distribution seems to be preserved throughout the life of the cell and it has been recently suggested that phospholipid asymmetry may be stabilized by specific interactions between aminophospholipids and
membrane skeletal proteins and enzymes (Franck et al., 1988). The dynamic steady state and asymmetric bilayer is regulated by the activities of three enzymes of which two are ATP-dependent that seem to work in concert to transport both amino phospholipids and choline phospholipids from inner to outer leaflet and another 37 kDa, Ca\(^{2+}\)-dependent scramblase which rapidly moves the phospholipids back and forth between two membrane leaflets (Connor et al., 1992; Manodori et al., 2000). Cytoplasmic Ca\(^{2+}\) concentration modulates membrane phospholipid asymmetry by activating the scramblase and inhibiting the Mg\(^{2+}\) ATPase aminophospholipid translocase.

The Effect of increased intracellular calcium other than the activation of the phospholipids scramblase is promotion of microvesicle shedding, which is generally accompanied by loss of phospholipids asymmetry (Kiefer and Snyder, 2000). In addition, destabilization of cytoskeleton proteins can result in blebbing, opening of cation channels and increase in the intracellular calcium level and activation of intracellular protease, calpain that can externalize PS. Erythrocytes that express PS in their outer membrane surface are bound by macrophages (Fadok et al., 1992a; 1992b; Utsugi et al., 1991) and are cleared from the peripheral circulation.

**MOLECULAR CHANGES IN APOPTOSIS**

Apoptosis at molecular level is carried out or affected by the action of a family of Ca\(^{2+}\)-dependent proteases, the calpains (Kothakota et al., 1997; Perrin and Huttenlocher, 2002). Two major calpain isoenzymes, \(\mu\)-calpain and m-calpain require \(\mu\)M to mM Ca\(^{2+}\) for activations respectively. Calpain is
found mainly in the cell cytosol and is usually present in its inactive form (Melloni et al., 1992). Elevated calcium levels activate calpain by autoproteolysis that lead to a decrease in their levels translocating to erythrocyte membrane thereby decreasing the cytosolic calpain level. Translocated calpain degrades membrane proteins and enzymes triggering apoptosis. The versatility of intracellular calcium mediating the majority of cellular process is reflected on the calpain system. It activates the functional reserves of the cell at moderate calcium concentrations and causes limited protein degradation.

Calpains thus, at the expense of free radical mediated reactions function as destruction dependent modulators that remove limited portions of protein substrates, cleaving specific proteins and signaling cascade proteins thereby suggesting a potential role for this enzyme in the regulation of cell death (Kim et al., 2002). Further, its specific inhibitor the calpastatin regulates the activity of calpain in erythrocytes (Kelly and Malley, 1993). Age-associated oxidative stress can activate calpain that degrades membrane cytoskeletal proteins leading to clustering and modification of band 3 protein tertiary structure in the plane of membrane generating the senescent cell antigen and thereby premature erythrocyte death (Kay and Goodman, 2003).

**ERYTHROCYTE PATHOPHYSIOLOGY WITH AGE**

Oxidative stress mediated shortened life span of erythrocytes is incriminated to play a central role in the pathogenesis in elderly. The most
obvious disease that occurs with advancement of age and related to
erthrocytes are anemia. cardiovascular diseases and Alzheimer's disease
(Anes et al., 1993).

Even after simultaneously adjusting for demographic characteristics
and health status the decline of hemoglobin is the major reason for anemia.
Various signs and symptoms associated with the anemia are characterized by
hypochromic, microcytic red cells with variable numbers of nucleated
erythrocytes and reticulocytes (Michalska et al., 1997), which could lead to
other pathological conditions.

Cardiovascular diseases in elderly persons cause cellular and
molecular changes in erythrocytes as a consequence of oxidative and
proteolytic stress. Alterations in band 3 profiles, promote the linkage of IgG
antibodies to erythrocyte membrane leading to implications in cardiovascular
diseases during oxidative and proteolytic stress in aged persons (Santos-Silva
et al., 1995).

Studies lead to the evidence that oxidative stress lead to early changes
in erythrocytes of Alzheimer's disease patients in elderly. Such age-associated
changes play a role in propagating amyloidosis when erythrocytes adhere to
the endothelial lining with greater affinity for the microvascular cells and
arterial cells (Ravi et al., 2004).
ANTIOXIDANT DEFENSES AGAINST OXIDATIVE THREATS

Erythrocytes by virtue of their physiological role are exposed to continuous oxidative stress. Over the course of evolution, erythrocytes have developed a comprehensive array of antioxidants to adapt to the continuous challenge of free radicals formed in the biological systems (Cheeseman and Slater, 1993). The free-radical theory of aging thus serves and plays a prominent role in invoking antioxidants as powerful defense mechanisms protecting the cells and tissues from oxidative stress. However, erythrocytes are prone to cumulative oxidant-mediated damage due to endogenously and exogenously produced radicals with advancement of age. Further, ageing is associated with greater susceptibility to nutritional deficiencies that lead to progressive senescence of cellular function. It has also been suggested that exogenous nutritional supplementation with antioxidants reduces the onset of ageing (Kalaiselvi and Pancerselvam, 1998). Antioxidants are therefore used as important nutritional supplements to prevent oxidative damages. The intake of plant-derived natural antioxidants is recently more in practice, as the usage of synthetic antioxidants has long been questioned for its safety.

PLANT-DERIVED NATURAL ANTIOXIDANTS

Herbal remedies have been used for various treatments from the days of caveman. Traditional medicine has been widely used to heal diseases in about 75-80% of world population. Increased demand for herbal products is the ‘back-to-nature’ global trend and the belief of the general public is that
natural products are much safer without deleterious side effects. Various parts of medicinal plants including stems, barks, roots, fruits and leaves act as potent antioxidants due to the presence of biologically active phytochemicals in them. *Solanum trilobatum* is one such medicinal herb rich in antioxidant supplement with wide beneficial biological property.

**Solanum trilobatum**

*Solanum trilobatum*, a medicinal treasure (*rejuvenator*), belongs to the family Solanaceae commonly found in the Deccan peninsula in India. It is a branched climbing shrub with sharp, recurred, short compressed spines, irregular lobed leaves, sinuate or ovate purple blue flowers and scarlet red berries with seeds.

**BIOLOGICAL PROPERTIES OF FLAVONOIDS**

Bioflavonoids and polyphenols are being recognized as important nutritional supplements benefiting human health. There are approximately
5000 known plant phenolics that have biological functions (Scalbert et al., 2005). The recent explosion of interest in the bioactivity of Solanum trilobatum is due, at least in part to the potential health benefits of these polyphenolic compounds present in them. The polyhydroxy flavones, flavones, flavonols, isoflavonoids and other groups of these compounds proved to have a high degree of antioxidant activity and they are found to be widespread phenolics in plant materials (Hudson, 1991).

Flavonoids, the benzo-O-pyrone derivatives have been shown to possess free radical scavenging ability that may be related to the arrangement of functional groups about the nuclear structure and the relative orientation of various moieties on the molecules (Rice Evans, 1996; Chen et al., 1990). The antiradical property of flavonoids is directed mostly towards hydroxyl, superoxide, peroxyl and alkoxy radicals (Husain et al., 1987; Huguet et al., 1990; Sichel et al., 1991). Furthermore, flavonoids possess metal chelating property that contributes to their antiperoxidative effect (Morel et al., 1993).

Phenolic compounds are prototype chain-breaking antioxidants (Klara et al., 1999) against lipoperoxidative damage depending on the hydrogen-donating capacity of a hydroxyl group in each molecule. However, the effectiveness of antioxidant protection by phenolic antioxidants has been found to be related also to their incorporation rate into cells and to their orientation in biomembrane (Kaneko et al., 1994).
CHEMICAL COMPOSITION AND ANTIOXIDANT PROPERTY OF Solanum trilobatum EXTRACT

Solanum trilobatum extract consists of high amounts of polyphenolic and flavonoid compounds including solasodine, linoleic acid, palmitic acid, oleic acid and stearic acid (Saradha Vasanth et al., 1990). Partially purified fraction of Solanum trilobatum contained sobatum, β-sitosterol, disogenin and β-solamarine, which are responsible for many biological properties (Mohanan and Devi, 1998b). Recently and a strong antioxidant activity has been reported in plant Solanum trilobatum due to the presence of isoflavonoids, phenols, phenolic acids, xanthones and lignans in them (Amir and Kumar, 2004; Sini and Devi, 2004).

THERAPEUTIC BENEFITS OF Solanum trilobatum

Solanum trilobatum possessed a broad spectrum of antibacterial, antifungal and antitumoural activities (Purushothaman, 1980). Mohanan et al. (1997) has also proved that Solanum trilobatum possess free radical scavenging activity when exposed to the ultraviolet rays. Solanum trilobatum, L. are widely used to treat respiratory diseases in Indian traditional system of Medicine (Govindan et al., 1999). The berries and flowers are administered for treatment of cough. The decoction of various parts of the plant is used in chronic bronchitis (Kirtkar and Basu, 1993). All parts of this common shrub of southern India are useful in asthma, chronic fibrile infections and difficult parturition. Further Solanum trilobatum is used in the treatment of tuberculosis and all kinds of lung disease (Chopra et al., 1956).
Reports have shown that the active principle from *Solanum trilobatum* possessed an antitumor effect against chemically induced tumors (Mohanand and Devi, 1996; Mohanan and Devi, 1998a). The chemopreventive effect against cyclophosphamide and radiation induced toxicity (Mohanan and Devi, 1998b). Studies also revealed the chemopreventive effect of *Solanum trilobatum* against carbon tetra chloride and DEN (N-diethylnitrosamine) induced toxicity (Shahjahan et al., 2004; Shahjahan et al., 2005) due to its antioxidant activity and hepatoprotective property. Leukocytopenia, as well as body weight loss and decreased hemoglobin produced by radiation treatment were remarkably reduced by *Solanum trilobatum* administration in rats (Mohanan and Devi, 1997).