The process of maintaining life for an individual is a constant struggle to preserve his/her integrity. This comes with the price of responsiveness to systemic inflammation (Broad, 2000). Whenever there is a cell injury by noxious stimuli like trauma, infection, mechanical, thermal or chemical stimuli, the inflammatory response sets in, constituting a complex network of molecules and cellular interactions directed to facilitate a return to physiological homeostasis and repair (Ferrero-Miliani et al., 2007). This response is composed of both local events and a systemic activation mediated by cytokines. It occurs in three distinct phases, an acute phase characterized by local vasodilatation and increased capillary permeability, a sub-acute phase characterized by infiltration of leukocytes and phagocytic cells, and a chronic proliferative phase, in which tissue degeneration and fibrosis occurs. Acute inflammation is a short term process usually appearing in a few minutes or hours and ceasing once the injurious stimulus has been removed (Cotran et al., 1998), accompanied by tissue repair. If tissue repair is not restored, then inflammation becomes a chronic condition that continuously damages the surrounding tissues. The collateral damages caused by the chronic inflammation usually accumulates slowly, sometimes asymptomatically for years, but can eventually lead to severe tissue deterioration (Mitchell and Cotran, 2003).

Chronic inflammation, especially of low grade, is considered to be involved in the pathogenesis of many autoimmune and age-related diseases – Alzheimer's disease, asthma, atherosclerosis, cancer, diabetes and obesity, liver injuries, etc. (Vignola et al., 2000; Stalnikowitz and Weissbrod, 2003; Licastro et al., 2005; Wellen and Hotamisligil, 2005). The inflammatory response appears to be the prevalent triggering mechanism driving tissue damages associated with these diseases, and therefore, the term "Inflammaging" has been coined to explain the underlining inflammatory changes (Franceschi et al., 2000). A wide range of etiological factors like smoking, hyperlipidemia, hyperglycemia, decreased production of sex hormones, physical inactivity, etc. are likely to contribute to increased low-grade inflammatory activity (Krabbe et al., 2004) and have been suggested to produce pro-inflammatory cytokines (Rudin and
Barzillai, 2004). This low-grade chronic inflammation leads to increased levels of circulating inflammatory mediators (Ridker and Morrow, 2003). Thus, chronic inflammation and inflammatory mediators constitute a link between lifestyle factors, infections, and physiological changes i.e. diseases. The inflammatory response is a defense mechanism and not, *ipso facto*, a disease, and its role is to restore normal structure and function to the infected or damaged tissues; but the over-production of inflammatory mediators may aggravate immune-inflammatory-diseases and contribute to earlier death. Anti-inflammatory treatment might play a decisive role in prevention or significantly retarding the manifestation of these diseases.

Arachidonic acid metabolism plays central role in inflammatory process and associated disorders. Some of the anti-inflammatory drugs inhibit the lipoxygenase pathway and some inhibit cyclooxygenase pathway, and these two pathways can be used for potential intervention against inflammation. Unfortunately most of the anti-inflammatory drugs, particularly steroids and cyclooxygenase (COX) inhibitors are often associated with adverse/side effects including gastro-intestinal irritation, ulcers, hypertension, and cardiac abnormalities (William, 1989; Wolfe and David, 1999). There has also been some concern over the use of COX inhibitors for therapeutic intervention, especially since some of the products based on COX-2 were either withdrawn or made to carry warning by the US FDA (Naesdal and Brown, 2006; Salmon, 2006). Salicylates, phenylbutazone, and indomethacin frequently produce ulcers and dyscrasia (Ahmad et al., 1992). It is therefore inevitable to search for new, less toxic, and more effective anti-inflammatory agents. Moreover, synthetic drugs are very expensive to develop, and whose cost of development ranges from 0.5 to 5 million dollars. On the contrary many medicines of plant origin have been used since long time without any adverse effects. Exploring the healing power of plants is an ancient concept. For centuries people have been trying to alleviate and treat disease with different plant extracts and formulations (Cowan, 1999). Hence, the modern scientific researchers are diverted towards the herbal sources for newer anti-inflammatory agents. Numbers of references are available
in the field of ethnomedicinal plants used as anti-inflammatory drugs. The use of herbal remedies for inflammatory conditions has been getting momentum in recent years (Chrubasik et al., 2007). In the traditional Ayurvedic system of medicine, many polyherbal formulations are being prescribed for inflammatory conditions (Anonymous, 2003). The 5-lipoygenase inhibitors of plant origin are also reported to offer significant relief without any adverse effects, and are becoming choice of treatment for chronic inflammatory disorders (Oliver, 2007; Krishanu et al., 2008).

*Clitoria ternatea* Linn. is important medicinal plant of Ayurvedic system of medicine (Anonymous, 2003), and is known as 'Aparajita'. It is also popularly known as butterfly pea or conch flower. Traditionally, *Clitoria ternatea* (CT) is also employed in cases of ascetics, enlargement of the abdominal viscera, sore throat, skin diseases, chronic bronchitis, epilepsy, insanity, rheumatism, and swollen joints (Morris, 1999; Anonymous, 2003). It is one of important constituents of Ayurvedic preparations "Misraka sneha", and "Vata rakta antaka rasa". The plant is reported to have antimicrobial, antipyretic, anti-inflammatory, analgesic, diuretic, local anesthetic, anti-diabetic, insecticidal, blood platelet aggregation inhibiting, and vascular smooth muscle relaxant properties (Mukherjee et al., 2008). However, there are no enough evidences in literature to support the use of CT, especially seeds and roots in inflammation, and related conditions. Therefore in the light of above scenario, the present investigation was undertaken to study the potential of CT seed and root in inflammation, hepatic dysfunction, and related disorders using experimental animal models.