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Cancer is a genetic disease resulting in the loss of normal cell-cycle control results unregulated growth and the lack of differentiation. Cancer cells share a number of characteristics including self dependence on positive regulatory signals; lack of response to growth inhibitory signals, limitless proliferation, resistance to apoptosis, capability of getting nutrients and oxygen by angiogenesis, and the ability to invade and establish distal metastasis (Hanahan & Weinberg, 2000). The genetic changes associated with cancer include gain or loss of entire chromosomes, chromosomal translocations, gene amplifications, mutations or deletions. These changes makes the cancer cell to acquire certain characteristics which include self sufficiency in growth signal, loss of sensitivity to antigrowth signals, uncontrolled proliferation through inhibition of telomerase action, evading apoptosis, capability to metastasize and stimulates production of new blood vessels by triggering angiogenesis.

The transformation process in which an apparently normal cell is converted or “transformed” into a malignant cell is called carcinogenesis. The three main steps of carcinogenesis are initiation, promotion and progression. A host of mutations in genes bring about these steps. Two distinct classes of genes, proto-oncogenes and tumor-suppressor genes are involved in the cancer process. The generation of excessive reactive oxygen species (ROS) as byproducts of aerobic metabolism and a concomitant fall in the intrinsic antioxidant capacity of cells leads to a state of oxidative stress, which contributes to carcinogenesis. High levels of ROS generated by external stimuli including chemical carcinogens, ultraviolet radiation, bacterial or viral infection, etc. elicit deleterious effects on human health (Surh et al. 2005). ROS, such as superoxide radical anion, hydroperoxyl radical, hydrogen peroxide and hydroxyl radical contribute to tumorigenesis either directly by damaging critical biomolecules or indirectly by modulating cellular signal transduction pathways (Kundu & Surh 2008). Moreover, accumulation of ROS in vivo leads to a state of persistent local inflammation. Inflammation plays a role in multistage carcinogenesis by several distinct mechanisms including damage of genomic DNA and alteration of intracellular signal transduction leading to abnormal cellular growth. Thus, both oxidative stress and inflammation not only initiate tumorigenesis but also promote the proliferation of damaged cells and create a tumor microenvironment favorable for the neoplastic transformation of premalignant cells (Kundu & Surh 2008; Surh et al. 2005).
Proliferation and differentiation involves many genes which are mainly involved in cell cycle control, apoptosis, DNA repair, aging, immortalization, angiogenesis and metastasis. The main oncogenes involved in the tumor progression and proliferation are ras, src, MAP kinase, raf, NF-kB, Bcl-2, c-myc, cyclin D etc. and tumor suppressor genes are P53, Rb, PTEN etc. (Sharma & Settleman, 2007).

Conventional strategies used in cancer treatment include surgery, chemotherapy, and radiation therapy hence each modalities having limitations in clinical use. Surgery is only possible for solid tumors. Ionizing radiations used in cancer therapy exert damaging effects in normal tissues and induce complex response at cellular and molecular level. When a chemotherapeutic drug enters the circulation, these agents not only kill the cancer cells but also the normal cells which result in side effects including organ toxicity and myelosuppression. The other modalities of cancer treatment with biological materials as monoclonal antibodies, cytokine therapies, gene therapy etc are still under primitive stage and some of them are not giving expected promising results. There is an urgent need to find out the cancer medicines which having anticancer properties and at the same time nontoxic to the normal cells. Moreover the toxicities associated with the present cancer modalities, radiation and chemotherapy and resistance to antineoplastic drugs had made the researchers for seeking new sources of natural chemopreventive agents with less toxicities. The search for potential anticancer agents from natural products dates back to centuries.

Carotenoids are a class of natural fat-soluble pigments that are found in many fruits and vegetables. Consumption of a diet rich in carotenoids has been epidemiologically correlated with a lower risk for several diseases (Di Mascio et al. 1991). The effects of nutraceuticals on apoptotic pathways, signaling pathways, and/or different targets in cancer mean that they could be helpful starting points in the design and development of novel cancer preventive agents. The carotenoid like β-carotene, lycopene and lutein present in fruits and vegetables exert antioxidant functions such as quenching of singlet oxygen and other electronically excited molecules and reduces the progression of many degenerative diseases (Di Mascio et al., 1989). Lutein (3,3’-dihydroxy-β,c-carotene) is a carotenoid, is abundantly present in nature, is commercially prepared from marigold flower (Tagetes erecta L)). The extended conjugated structure of lutein has been shown to have significant antioxidant potential and a protective effect against the oxidative damage to macula induced by singlet oxygen produced by ultraviolet light. Lutein and zeaxathin are important carotenoid components in the human diet and several investigators have
suggested that elevated intake of food rich in lutein is related to decreased macular
degeneration and the risk of cataracts (Stahl & Sies, 2005).

In the present study we have evaluated the anticancer and anitmutagenic activity
of lutein and its possible mechanism of action. This work also concentrated on the
antioxidant, anti-inflammatory, antitumor activity using *in vitro* and *in vivo* models,
and also analyzed the capability of lutein to protect mice from radiation and other
chemotherapeutic drugs induced damages. In addition, we have investigated the other
pharmacological actions of lutein such as gastro and hepatoprotective activities in rats.