Chapter-2

Review of Literature & Plan of Work
2.1. REVIEW OF LITERATURE

Ellinger et al., (2011) reported that regular consumption of green tea in amounts of at least 0.6–1.5 l/day may increase plasma antioxidant capacity, reduce lipid/(protein) peroxidation (especially the oxidation of LDL) and may improve the protection against DNA damage in healthy subjects.

Rockenfeller and Madeo, (2010) demonstrated the anti-ageing activity of green tea.

Marco et al., (2009) proposed that chronic green tea consumption prevents age-related changes in rat hippocampal formation.

Kim et al., (2009) reported that L-theanine, an amino acid in green tea, attenuates beta-amyloid-induced cognitive dysfunction and neurotoxicity. In addition reduces oxidative damage and inactivation of ERK/p38 kinase and NF-kappa B pathways.

Liu et al., (2009) suggested that green polyphenols inhibit plasminogen activator inhibitor-1 expression and secretion in endothelial cells.

Larsen et al., (2009) and Yang et al., (2009) found anti-tumor activity of green tea in various type carcinomas.


Sharangi et al. (2009) published that tea flavonoids reduce inflammation, have antimicrobial effects and prevent tooth decay. Consumption of tea may have diuretic effects due to the caffeine.

Mejia et al., (2009) concluded that theanine, a unique amino acid in tea, enhances cognition in humans and has neuroprotective effects.

Li et al., (2009) published that long-term administration of green tea catechins prevents age-related spatial learning and memory decline in c57bl/6 j mice by regulating hippocampal cAMP-response element binding protein signaling cascade.

Kaur et al., (2008) reported green tea extract administration is effective in enhancing learning and memory in aged rats, and hence, may serve useful in reversing age-related deficits.
Ratnasooriya et al., (2008) concluded that black tea brew can function as a quick acting, safe, oral aphrodisiac which may also be useful in certain forms of sexual inadequacies such as premature ejaculation and impaired libido and other sexual functions.

Cao et al., (2007) observed that green tea increases anti-inflammatory tristetraprolin and decreases pro-inflammatory tumour necrosis factor mRNA levels in rats, thus have antiinflammatory action.

Guo et al., (2007) investigated the protective effects of green tea polyphenols in the 6-OHDA (6-hydroxyl dopamine) rat model of Parkinson’s disease through inhibition of ROS-NO Pathway. They found GTP treatment dose-dependently preserved the free radical scavenging capability of both the midbrain and the striatum.


Babu et al., (2006) reported that therapeutic effect of green tea extract on oxidative stress in aorta and heart of streptozotocin diabetic rats.

Kaul et al., (2004) suggested that green tea polyphenols (GTPs) down regulates genes coding for PPAR-gamma, CD36, LXR-alpha, C-myc coupled with up regulating of genes coding for LDL-R and PPAR-alpha at the transcriptional level. The genomics of atherosclerosis can result as a result of cross talk between the genes coding for the LDL receptor (LDL-R), LXR alpha, PPARs (alpha, gamma), CD36 and C-myc. Based upon these results, it is proposed that GTPs have the inherent capacity to inhibit the development of atherosclerosis lesions.

Liu et al., (2004) reported the inhibition of fish gill lipoxygenase and blood thining effects of green tea extract.

Sano et al., (2004) reported the effect of green tea intake on the development on coronary artery disease. The study was designed to determine whether the consumption of green tea is proportionally associated with a decreased incidence of coronary artery disease (CAD) and the cardiovascular and cerebrovascular
prognosis. Green tea consumption was associated with a lower incidence of CAD in the Japan population.

Vinson et al., (2004) found that green tea and black teas inhibit atherosclerosis by lipid, antioxidants, and fibrinolytic mechanism. Both teas were equally effective in inhibiting atherosclerosis with the lower dose decreasing it 26-46% and the high dose decreasing it 46-63%.

Son et al., (2004) investigated the antiplatelet effect of green tea catechins which is due to the effect on arachidonic acid metabolism.

Weinreb et al., (2004) reported that green tea polyphenols are now being considered as therapeutic agents in well controlled epidemiological studies, aimed to alter brain aging processes and to serve as possible neuroprotective agents in progressive neurodegenerative disorders such as Parkinson’s and Alzheimer’s diseases.

Unno et al., (2004) suggested that green tea-catechin intake partially improves the morphologic and functional alterations that occur naturally in the brains of aged SAMP10 mice.

Baolu, (2003) studied the antioxidant property of green tea. They reported that antioxidant activity of green tea polyphenols (GTP) is due to the apparent health benefits of tea drinking and the experimental results with the polyphenols. These results suggest that the galloylated catechins show stronger antioxidant effect than that of nongalloylated catechins and the double bond in C ring plays an important role in this effect.

Deana et al., (2003) reported the green tea epigallocatechin-3-gallate inhibit platelet signaling pathways triggered by both proteolytic and nonproteolytic agonists.

Maron et al., (2003) reported that theaflavin rich green tea extract is an effective adjuvant to a low saturated fat diet to reduce LDL-C in hypercholesterolemic adults and is well tolerated.

Dona et al., (2003) reported that green tea have anti inflammatory property.
Das et al., (2002) found the antitumour and anti-inflammatory property of green tea. The antitumour effect of tea was evaluated in the 3-methylcholangriene (3-MC) induced solid tumour model in mice. Both black and green tea inhibited tumour growth and prevented metastasis. Histopathological study showed that tea treatment was able to reduce malignancy.

Kang et al., (2001) reported antiplatelet activity of green tea catechins is mediated by inhibition of cytoplasmic calcium increase. These result shows that GTC significantly inhibited fibrinogen binding to human platelet surface GPIIb/IIIa complex. These results indicate that the antiplatelet activity of GTC may be due to inhibition of an intracellular pathway preceding GPIIb/IIIa complex exposure.

Kang et al., (1999) studied antithrombotic activity of green tea catechins and (-) - epigallocatechin gallate. GTC and EGCG prevented death caused by pulmonary thrombosis in mice in vivo in a dose dependent manner.

Yen et al., (1997) reported that green tea have antioxidant property.

Schreurs, (2010) demonstrated the effects of cholesterol on learning and memory.

Agrawal et al., (2009) studied effect of brain insulin receptors, AChE activity and oxidative stress in rat model of ICV STZ induced dementia.

Parle et al., (2007) abstracted age, stress, emotions are conditions that may lead to memory loss, amnesia, anxiety, dementia or to more ominous threats like schizophrenia and Alzheimer’s disease.

Sparks et al., (2002) reported that HMG-CoA reductase inhibitors (statins) are effective in the treatment of Alzheimer’s disease.

Staay et al., (2005), demonstrated that cholinesterase inhibitor antagonize scopolamine-induced spatial memory deficits. The cone field would be a useful component of a behavioral screening battery to test the effects of putative cognition enhancers.

DeVries et al., (2001) reviewed many stroke techniques to assess cognitive and behavioral studies in animal models.

Ester et al., (1997) reported effects of acute or daily administration of diazepam on spatial learning and working memory.
2.2. PLAN OF WORK

Poor memory, lower retention and slow recall are common problems in today’s stressful and competitive world. Age, stress, emotions are the conditions that may lead to memory loss, amnesia, anxiety, high blood pressure, dementia or to more ominous threat like schizophrenia and Alzheimer’s disease. Despite the severity and high prevalence of Alzheimer’s disease, allopathic system of medicine is yet to provide satisfactory antidote. Therefore we are motivated to explore the new approach in Indian traditional system. In this study we will try to explore the potential of green tea in reversing the memory deficits.

a. Experimental Animals

Swiss albino mice of either sex will be employed for study.

b. Extraction of Drug

The aqueous extract of green tea will be prepared in Soxhlet apparatus after authentication washing, drying and milling into coarse powder.

c. Evaluation of Memory

Memory of mice will be evaluated on Morris water Maze (for long term memory) and elevated plus maze (for short term memory). Piracetam, Indomethacin, Metrifonate and Atorvastatin will be used standard drugs.

d. Impairment of Memory

MK-801, Diazepam, High fat diet (HFD), Scopolamine, Steptozodosin (intracerebral injection), Age induced amnesia and Hypoxia induced amnesia (induced by temporary occlusion of both common carotid arteries) models will be used for impairment of memory. Assessment of mice memory impairment will be done by Morris water maze, elevated plus maze and on passive avoidance test.

e. Quantitative Estimation of Serum Cholesterol Concentration

Blood cholesterol will be estimated spectrophotometrically at 560 nm by one step method using a commercially available kit.

f. Quantitative Estimation of Serum Glucose

Serum glucose will be estimated spectrophotometrically at 505 nm by glucose oxidase/peroxidase method using a commercially available kit.
g. **Quantitative Estimation of Brain Nitric Oxide**
Brain nitric oxide will be estimated spectrophotometrically at 546 nm by Griess reagent method.

h. **Measurement of Brain Antioxidant Activity**
Antioxidant activity will be measured by estimation of thiobarbituric acid reactive substance (TBARS).

i. **Estimation of Brain Cholinesterase**
Cholinesterase activity will be measured by method of Ellman et. al. with a single modification.

j. **Collection of Brain Sample**
The animal will be sacrificed by cervical decapitation under light anesthesia. The whole brain will be carefully removed from the skull.