in AD was mediated by Ca^{2+} dyshomeostasis (Berrocal et al. 2009). Ginkgolide B was observed to prevent Aβ-triggered Ca^{2+} influx through N-methyl-D-aspartic acid (NMDA) receptors. The hyperphosphorylation of tau protein is a major event in the AD pathogenesis, where EGb761 was found to dephosphorylate the hyperphosphorylated tau protein in the hippocampus and cortex of mice (Watanabe et al. 2001). The nerve growth factor (NGF) level was modulated in AD. Bilobalide, and a terpenoid constituent of EGb761 upregulated NGF, was reported along with the glial-derived neurotrophic factor and vascular endothelial growth factor in rat cortical astrocytes (Zheng et al. 2002).

9.3.8 *Glycyrrhiza glabra*

G. glabra roots and rhizomes exhibit antimicrobial, anti-inflammatory, antiulcer, anxiolytic and expectorant activities, hence they are used widely as medicine. A natural product from *G. glabra*, known as 2, 2', 4-trichydroxychalcone, prevents the generation of Aβ and its plaque formation, thus improving memory deficits (Dhingra et al. 2004) by inhibiting β-site amyloid precursor protein cleaving enzyme 1 (Zhu et al. 2010). Flavonoids, such as licorice, chalcone and licorice isoflavones from *G. glabra*, exhibited strong antioxidant activity scavenging free radicals, which may help in the prevention of AD. Glyderinine, glabridin and lichochalocone A from *G. glabra* exhibited *in vivo* anti-inflammatory effects (Azimov et al. 1998; Furuhashi et al. 2005). The improvement in learning and memory was evident in mice upon the administration of licorice extract (Parle et al. 2004). The AChE inhibitory activity of *G. glabra* might assist the improvement of memory through cholinergic pathways (Dhingra et al. 2006). The development of inflammatory lesions is part of the pathology of AD. *G. glabra* has anti-inflammatory effects that aid in the prevention of such lesions.

9.3.9 *Huperzia serrata*

H. serrata is a Chinese herb which contains an alkaloid Huperzine A, which is a natural cholinesterase inhibitor (Ashani et al. 1992). This alkaloid is commercially available as a food supplement and as a drug for memory loss and mental disabilities. Huperzine A demonstrates AChE inhibitor activity, antioxidant properties, memory improvement and neuroprotection (Kozikowski and Tuckmantel 1999). With the exhibition of these properties, Huperzine A significantly improved the symptoms of AD in a double-blind clinical trial of over 450 patients in China (Zhang et al. 2002). The neuroprotective effect of *H. serrata* was evidenced from protection against glutamate toxicity through antagonism at NMDA receptors (Ved et al. 1997; Wang et al. 1999). Huperzine A prevents the deleterious effect of Aβ and recuperates cholinergic and monoaminergic dysfunction in rats (Liang et al. 2008). The production of amyloidogenic fragments was suppressed by increased α-secretase activity with huperzine A treatment (Peng et al. 2006). This might diminish Aβ toxicity and accumulation of fibrillary amyloids in the brains of AD patients. *H. serrata* is a high potential AChE inhibitor with strong memory improvement capacity, which